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ADVANCES AND PERSPECTIVES

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Chapter 7

Genetics of Resistance to Pests and Disease

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Potato is a host to many pathogens that affect all parts of the plant and cause reductions in the quantity and quality of yield. The development of new cultivars that are more resistant to economically important pests and diseases is therefore one of the top priorities for potato-breeding programs worldwide. Numerous resistance genes have been discovered in *Solanum* species and introgressed into the cultivated potato. Since the 1980s, the introduction of molecular marker techniques has facilitated gene mapping and shifted orientation from phenotype-based resistance genetics to genotype-based approaches. A number of loci conferring quantitative resistance [quantitative resistance loci (QRL)] and around forty single dominant genes (R-genes) conferring qualitative resistance have been positioned on the potato molecular map. As of 2006, eight of the mapped R-genes have been isolated and molecularly characterized. The analysis of mapped and cloned resistance genes shows that they often occur in clusters and that some of them can respond to more than one elicitor. Ongoing research on resistance gene evolution will help us in understanding the dynamic interaction between potato plants and pathogens and opens a way for the development of more resistant cultivars.

7.1 RESISTANCE SCREENING

To carry out breeding and genetic studies, one must correctly identify genotypes based on phenotypes. This may sound like a trivial matter. However, especially in disease and pest assays, the concept of resistance is necessarily linked to the screening method. Screening data must be used to assign resistance phenotypes, and rankings of clones may vary with the screening method. There is often no black and white difference between resistant and susceptible phenotypes, so judgments must be made to assign phenotypes and, consequently, genotypes. Finally, most host–pathogen interactions are strongly influenced by the environment, so it is important to ultimately evaluate plants under the range of field conditions in which the crop will be grown.

7.1.1 Field screening

Field assessments of resistance are especially valuable because they evaluate plants under the conditions in which they will eventually be grown. However, field screening can be

expensive in terms of both time and resources, and screening results may be variable because of environmental heterogeneity.

Disease pressure is an important variable in field trials, but it can be difficult to control for two reasons. First, the amount of inoculum that is naturally present in the field cannot be easily manipulated in most cases. Second, disease pressure is typically influenced by environmental factors such as temperature and humidity, which cannot be controlled. If disease pressure is too high, then clones with moderate, but possibly acceptable, levels of resistance will be eliminated. Stewart et al. (1983) found that if young seedlings were inoculated with *Phytophthora infestans* (the causal agent of late-blight disease), then even resistant plants died. Inoculations of older seedlings allowed for the successful identification of resistant plants. Similarly, Hilton et al. (2000) found that if tubers were exposed to *Helminthosporium solani* for too long under ideal conditions for the silver scurf fungus, then no differences were seen between resistant and susceptible cultivars. On the contrary, if disease pressure is too low, then selection for resistance will also be ineffective. For example, Hoyos et al. (1993) assayed vascular colonization by *Verticillium dahliae* in seedling transplants and identified putatively resistant clones. However, few of the putatively resistant clones selected as seedlings were resistant in the first clonal generation, probably because selection pressure was too low in the seedling generation.

Because breeders typically evaluate large populations of segregating plants, they must strive toward a balance between quantity and quality of disease-scoring data. On one hand, it is important to be able to quickly evaluate large numbers of clones in segregating populations. On the other hand, the data are meaningless if they do not effectively identify resistant phenotypes. It is important to identify screening methods that will consistently identify resistant plants in breeding programs with large numbers of genotypes. For example, a common Verticillium wilt-resistance-scoring technique involves the plating of stem tissue from infected plants, followed by the counting of *Verticillium* colonies that grew from that tissue. Because stem-to-stem variation for pathogen populations is very high, it is important to evaluate a large number of stems, which is time-consuming. A compromise offered by Treadwell (1991) substituted a rating scale for colony counts, allowing a larger number of stems to be evaluated. Similarly, Christ (1991) found that estimating leaf area covered by early-blight (*Alternaria solani*) lesions was faster than counting individual lesions.

Resistance assays can be based on disease symptoms or pathogen levels in the plant. Disease symptom expression is typically an easy, fast, and inexpensive assay. For example, Dale and Brown (1989) determined that foliage symptom expression correlated with yield loss due to potato cyst nematode (*Globodera pallida*) infection. Consequently, selection for foliage vigor allows for the identification of tolerant genotypes. Christ (1991) also effectively identified early-blight-resistant clones based on symptom development in the field.

Although symptom expression is commonly used for disease scoring, there are problems associated with this technique. It may be difficult to distinguish between symptoms caused by the pathogen of interest and those caused by other pathogens or by abiotic stresses. In addition, the interaction between the pathogen and both its biotic and abiotic environment may result in a wide range of symptoms, some of which are not typical. Consequently, methods have been developed to quantify pathogen populations in plant tissues. These methods are discussed in Section 7.1.3.

It is often desirable to partition resistance into components, each of which can be selected individually and may be controlled by its own genetic system. For example, resistance to potato leaf roll virus (PLRV) can result from resistance to infection, resistance to virus accumulation, and reduced translocation from leaves to tubers (Wilson and Jones, 1993). Each of these components appears to be controlled by a different genetic system. It is possible to select plants for each of these components, and combining the components may provide even more effective resistance (Solomon-Blackburn et al., 1994). Late-blight resistance can also be partitioned into components such as infection efficiency, latent period, lesion size, and sporulation. However, unlike PLRV resistance components, which are controlled by major genes, these components of late-blight resistance appear to be quantitatively inherited and they interact with each other to determine the resistance phenotype (Birhman and Singh, 1995). The heritability estimates for each component vary, as does the contribution of each to the resistance genotype. Similarly, scab (*Streptomyces scabies*) resistance can be partitioned into surface area infected, lesion type, and proportion of scabby tubers (Goth et al., 1993). Cluster analysis based on lesion type and surface area infected appears to most effectively identify resistant clones.

Host plant maturity is an important variable in most disease and pest resistance evaluations. Typically, plants are more resistant early in their life cycle, becoming more susceptible with age. Immature plant resistance is commonly observed for late blight (Dorrance and Inglis, 1997), early blight (Boiteux et al., 1995), and Verticillium wilt (Busch and Edgington, 1967). Consequently, it is important to evaluate disease resistance as plants senesce in the field. This can be a problem when scoring wild *Solanum* species, which typically do not senesce during the growing season in North temperate regions. In contrast to immature plant resistance, resistance to PLRV appears to increase in mature plants (DiFonzo et al., 1994).

7.1.2 Greenhouse screening

Greenhouse-based resistance assays can provide an attractive compromise between costly, time-consuming field trials and laboratory assays, which do not typically evaluate entire plants. They can identify putatively resistant plants that can then undergo more extensive field evaluations in subsequent studies.

A major advantage of greenhouse screening is that the environment can be controlled to optimize disease pressure while minimizing the effects of confounding biotic and abiotic factors. Greenhouse assays allow control over the timing, dose, and virulence of the pathogen. Stewart and Bradshaw (1993) optimized the greenhouse environment for early-blight resistance screening by controlling humidity, inoculum dose, and plant age at inoculation. Most plants identified as resistant or susceptible remained so in field tests. Similarly, Dorrance and Inglis (1997) found that resistance scores from greenhouse inoculations with *P. infestans* corresponded to those from field evaluations.

Another advantage of greenhouse screening is that it allows for the evaluation of resistance to pathogens that cannot feasibly be introduced to the outside environment. For example, greenhouse assays were used to evaluate resistance to ring rot (*Clavibacter michiganensis* ssp. *sepedonicus*) because field trials would release a serious pathogen to the environment (Kriel et al., 1995b). Similarly, in regions where breeders cannot risk

spreading *P. infestans* to growers' fields, they use greenhouse evaluations to screen for late-blight resistance.

Greenhouse screening in potato also allows plants to be grown under short day conditions in winter in order to induce tuberization in wild *Solanum* species. Breeders can then evaluate tuber traits, which cannot be studied in the field because wild species do not tuberize under the long days of summer in North temperate regions. Hosaka et al. (2000) transplanted wild species seedlings into pots containing *S. scabies*. As the plants tuberized in the greenhouse during the winter, they were evaluated for resistance to common scab. Because short day conditions stimulate wild species to senesce, winter greenhouse trials also allow screening for true resistance rather than immature plant resistance, as in Section 7.1.1.

One limitation of greenhouse screening is that the controlled environment may not adequately mimic the complexities of a field trial. Consequently, levels of resistance may not correlate strongly with those in the greenhouse. For example, DiFonzo et al. (1994) reported that the variety 'Cascade' was more resistant to PLRV in the field than in the greenhouse. Apparently, resistance was more complex than the greenhouse screen alone could assess. Similarly, 'Desirée' was more resistant to early blight in field than greenhouse assays (Stewart and Bradshaw, 1993).

7.1.3 Laboratory screening

Laboratory-based assays are based on samples ranging from whole plants to individual cells. A number of biochemical assays have been developed to quantify pathogen levels in host plant tissue. A main advantage of these assays is that they can confirm the presence of the pathogen in both symptomatic (susceptible) and asymptomatic (tolerant) plants. Consequently, they can distinguish between tolerant and resistant plants.

Pathogen populations in inoculated host plant tissues can be quantified in a number of ways. For example, fungal pathogens can be quantified using culture plating, an enzyme-linked immunosorbent assay (ELISA), or the polymerase chain reaction (PCR). Culture-plating assays are commonly used for Verticillium wilt resistance screening. Hoyos et al. (1991) plated sap, whereas Davis et al. (1983) plated dried stem tissue from plants grown in *V. dahliae*-infested fields. Both reported a strong correlation between the numbers of colony-forming units and wilt scores in the field. Davis et al. (2001) suggest that the plating of roots on *V. dahliae* on selective medium provides an even more effective measure of colonization by the pathogen. Culture plating is simple and inexpensive, but it can be time-consuming and does not provide information until the pathogen grows, which may take weeks. In addition, it is sometimes difficult to distinguish between the pathogen and other organisms that grow on the plate. ELISA provides rapid results and does not require specialized equipment other than a plate reader. It has been used to assay for resistance to late blight (Harrison et al., 1991; Beckman et al., 1994), Verticillium wilt (Plasencia et al., 1996), and potato viruses (Valkonen et al., 1992a; Singh et al., 2000). PCR is now commonly used to quantify pathogen levels in disease resistance assays. The limit of detection of this technique is typically lower than that of ELISA tests or culture plating. Quantitative PCR has been used to distinguish between Verticillium wilt-resistant and Verticillium wilt-tolerant clones (Dan et al., 2001). In addition, PCR detection can

be species specific. For example, PCR can specifically detect *V. dahliae*, whereas ELISA tests detect both pathogenic forms of *Verticillium* (*Verticillium albo-atrum* and *V. dahliae*) (Plasencia et al., 1996; Dan et al., 2001). However, a limitation of both PCR and ELISA methods is that they cannot distinguish between living and dead pathogens, whereas culture plating detects only living material. Moreover, the pathogen population levels do not always mirror disease symptoms. Jansky and Rouse (2000) noted that Verticillium wilt symptoms did not correlate with levels of stem colonization. Similarly, Harrison et al. (1991) noted that levels of *P. infestans* in leaf tissue based on ELISA assays did not always relate to visual estimates of disease.

As an alternative to pathogen exposure in the field, plants or plant parts may be inoculated in the laboratory. Laboratory inoculations have been used effectively in resistance screening for silver scurf (Rodriguez et al., 1995; Hilton et al., 2000), soft rot (*Erwinia carotovora*) (Łojkowska and Kelman, 1994), powdery scab (*Spongospora subterranea*) (Merz et al., 2004), early blight (Bussey and Stevenson, 1991), and late blight (Dorrance and Inglis, 1998). However, the latter study illustrates a limitation of laboratory-based assays – they cannot simulate a field environment. Tubers of some cultivars that were susceptible in the laboratory assay were resistant in the field, probably because placement of tubers in the hill allowed them to escape infection in the field.

In some cases, it may be possible to screen for resistance using toxins produced by the pathogen rather than the pathogen itself. Christinzio and Testa (1999) screened potato leaves for late-blight resistance by applying fungal culture filtrates and then measuring electrolyte leakage. Similarly, Lawrence et al. (1990) were able to induce scab lesions by inoculating minitubers with thaxtomin, the toxin produced by *S. scabies*. Lynch et al. (1991) found that in vitro assays for early-blight resistance using culture filtrates containing toxic metabolites produced results similar to those using fungal spores.

7.2 RESISTANCE GENETICS IN POTATO

Wild and cultivated relatives of potato are credited with contributing the majority of disease resistance genes to breeding programs. It is interesting that most emphasis has been placed on a few species, such as late-blight resistance genes from *Solanum demissum* and virus resistance genes from *Solanum stoloniferum*. Only 13 wild species have contributed germplasm to European cultivars (Hawkes, 1990). Consequently, wild *Solanum* germplasm represents a largely untapped reservoir of genetic diversity for disease resistance genes. Ruiz de Galarreta et al. (1998) found that over 70% of the 98 wild *Solanum* species accessions they screened expressed resistance to one or more diseases. Much work remains to be done to identify and characterize the resistance factors in these species.

7.2.1 Resistance breeding

7.2.1.1 Exotic sources of disease and pest resistance

Reports of disease resistance in wild and cultivated relatives of potato are abundant (Table 7.1). Some species are especially potent sources of resistance to a number of

Table 7.1 Sources of potato disease and pest resistance.

Pathogen/Pest	Source of resistance	Reference
Virus		
Alfalfa mosaic virus (AMV)	*Solanum palustre*	(Valkonen et al., 1992b)
Andean potato latent virus	*S. palustre*	(Valkonen et al., 1992b)
Cucumber mosaic virus (CMV)	*Solanum fernandezianum, Solanum stoloniferum*	(Horvath, 1994; Valkonen et al., 1995)
Henbane mosaic virus	*S. stoloniferum*	(Horvath and Wolf, 1991)
Potato leaf roll virus (PLRV)	*Solanum chacoense, S. fernandezianum, S. palustre, Solanum sparsipilum, Solanum spegazzinii*	(Helgeson et al., 1986; Valkonen et al., 1992a; Brown and Thomas, 1994; Ruiz de Galarreta et al., 1998)
Potato virus A (PVA)	*Solanum polyadenium*	(Valkonen, 1997)
Potato virus M (PVM)	*S. palustre, S. sparsipilum*	(Valkonen et al., 1992b; Ruiz de Galarreta et al., 1998)
Potato virus S (PVS)	*S. palustre*	(Valkonen et al., 1992b)
Potato virus V (PVV)	*Solanum maglia*	(Valkonen, 1997)
Potato virus X (PVX)	*Solanum acaule, Solanum commersonii, Solanum lesteri, Solanum marinasense, Solanum oplocense, S. palustre, S. sparsipilum*	(Horvath et al., 1988; Tozzini et al., 1991; Valkonen et al., 1992b)
Potato virus Y (PVY)	*S. acaule, Solanum achacachense, Solanum acroscopicum, Solanum ambosinum, Solanum arnezii, S. chacoense, Solanum doddsii, S. fernandezianum, Solanum megistacrolobum, S. palustre, S. polyadenium, Solanum polytrichon, S. sparsipilum, S. stoloniferum, Solanum sucrense, Solanum tarnii, Solanum trifidum*	(Horvath and Wolf, 1991; Horvath et al., 1988; Valkonen et al., 1992a; Singh et al., 1994; Bosze et al., 1996; Valkonen, 1997; Takacs et al., 1999a)
Potato-yellowing virus	*S. palustre*	(Valkonen et al., 1992b)
Tobacco etch virus	*S. commersonii*	(Valkonen, 1997)
Bacteria		
Clavibacter michiganensis	*S. acaule, Solanum phureja, Solanum sanctae-rosae, Solanum stenotomum, Solanum verrucosum*	(Kurowski and Manzer, 1992; Ishimaru et al., 1994; Kriel et al., 1995a)

Erwinia carotovora/ Erwinia chrysanthemi	*Solanum bukasovii, Solanum bulbocastanum, Solanum circaeifolium, Solanum demissum, S. marinasense, S. phureja, S. stoloniferum, S. tarijense*	(Łojkowska and Kelman, 1989; Carputo et al., 1996; Chen et al., 2003)
Streptomyces scabies	*Solanum boliviense, S. bukasovii, Solanum canasense, Solanum multidissectum*	(Hosaka et al., 2000)
Fungi		
Fusarium sambucinum	*S. boliviense, Solanum fendleri, Solanum gandarillasii, Solanum gourlayi, Solanum kurtzianum, Solanum microdontum, S. oplocense, S. sanctae-rosae, Solanum vidaurrei*	(Lynch et al., 2003)
Helminthosporium solani	*Solanum albicans, S. chacoense, S. demissum, S. oplocense, Solanum oxycarpum, S. stoloniferum*	(Rodriguez et al., 1995)
Verticillium albo-atrum	*S. chacoense*	(Concibido et al., 1994; Lynch et al., 1997)
Verticillium dahliae	*Solanum berthaultii, S. chacoense, S. commersonii, S. phureja, Solanum raphanifolium, S. sparsipilum, S. tarijense*	(Corsini et al., 1988; Concibido et al., 1994; Bastia et al., 2000)
Oomycete		
Phytophthora infestans	*S. ambosinum, S. berthaultii, Solanum brachycarpum, S. bulbocastanum, Solanum cardiophyllum, S. chacoense, S. circaeifolium, S. commersonii, S. demissum, S. fendleri, Solanum guerreroense, Solanum iopetalum, S. megistacrolobum, S. microdontum, S. pinnatisectum, S. raphanifolium, S. sparsipilum, S. stoloniferum, S. sucrense, S. verrucosum*	(Holley et al., 1987; Tooley, 1990; Ruiz de Galarreta et al., 1998; Micheletto et al., 1999; Douches et al., 2001; Perez et al., 2001; Chen et al., 2003)

(*Continued*)

Table 7.1 *(Continued)*

Pathogen/Pest	Source of resistance	Reference
Nematodes		
Globodera pallida	*S. acaule, Solanum brevicaule, S. sparsipilum*	(Jackson et al., 1988)
Meloidogyne arenaria	*S. chacoense*	(Di Vito et al., 2003)
Meloidogyne chitwoodi	*S. acaule, Solanum andreanum, S. boliviense, S. bulbocastanum, S. fendleri, Solanum hougasii*	(Brown et al., 1989, 1991, 1999, 2004; Mojtahedi et al., 1995)
Meloidogyne hapla	*S. bulbocastanum, S. chacoense*	(Brown et al., 1995; Di Vito et al., 2003)
Meloidogyne incognita	*S. chacoense*	(Di Vito et al., 2003)
Meloidogyne javanica	*S. chacoense, S. commersonii, S. tarijense*	(Di Vito et al., 2003)
Insects		
Empoasca fabae	*S. berthaultii*	(De Medeiros et al., 2004)
Leptinotarsa decemlineata	*S. berthaultii, S. chacoense, S. circaeifolium, Solanum jamesii, Solanum neocardenasii, Solanum okadae, S. oplocense, S. pinnatisectum, S. polyadenium, S. tarijense, S. trifidum*	(Dimock et al., 1986; Groden and Casagrande, 1986; Sinden et al., 1986; Cantelo et al., 1987; Neal et al., 1989; Pelletier and Smilowitz, 1990, 1991; Neal et al., 1991; Franca and Tingey, 1994; Franca et al., 1994; Bamberg et al., 1996; Sikinyi et al., 1997; Rangarajan et al., 2000; Pelletier and Tai, 2001; Pelletier et al., 2001; Chen et al., 2003)
Myzus persicae	*S. berthaultii, S. bukasovii, S. bulbocastanum, Solanum chiquidenum, Solanum chomatophilum, S. circaeifolium, Solanum etuberosum, S. trifidum*	(Lapointe and Tingey, 1986; Radcliffe et al., 1988)
Phthorimaea operculella	*S. berthaultii*	(Malakar and Tingey, 1999)
Thrips palmi	*S. chacoense*	(Fernandez and Bernardo, 1999)

Potato accessions rated as resistant/highly resistant (or hypersensitive/immune for virus resistance) are included in the table. More information about accessions can be found on the United States Potato Genebank website (http://www.ars-grin.gov/ars/MidWest/NR6/).

diseases and pests. Resistance to ring rot, potato cyst nematode, root knot nematode (*Meloidogyne chitwoodi*), potato virus X (PVX), and potato virus Y (PVY) has been reported in *Solanum acaule*; resistance to Colorado potato beetle (CPB) (*Leptinotarsa decemlineata*), green peach aphid (*Myzus persicae*), potato tuberworm (*Phthorimaea operculella*), late blight, and Verticillium wilt has been reported in *Solanum berthaultii*; resistance to silver scurf, CPB, four species of root knot nematode, late blight, PLRV, PVY, thrips (*Thrips palmi*), and both Verticillium wilt species has been reported in *Solanum chacoense*; resistance to root knot nematode, late blight, PVX, tobacco etch virus, and Verticillium wilt has been reported in *Solanum commersonii*; resistance to potato cyst nematode, late blight, PLRV, Verticillium wilt, and potato viruses M, X, and Y has been reported in *Solanum sparsipilum*; resistance to soft rot, silver scurf, late blight, cucumber mosaic virus (CMV), henbane mosaic virus, and PVY has been reported in *S. stoloniferum*; and resistance to soft rot, CPB, root knot nematode, and Verticillium wilt has been reported in *Solanum tarijense*. The outgroup *Solanum palustre* seems to be an especially rich source of virus resistance genes. It is important to note that there is tremendous diversity within wild species and even within accessions, so fine screening is necessary to identify individual clones with resistance genes. From a breeding standpoint, it is encouraging to note that several of the wild species that are rich in disease resistance genes (e.g. *S. berthaultii, S. chacoense, S. sparsipilum*, and *S. tarijense*) are also easily accessible through the simple ploidy manipulations outlined below.

7.2.1.2 Sexual hybridization

Resistance genes are often derived from wild *Solanum* relatives, most of which are diploid. Consequently, the ploidy of selected wild species clones can be doubled somatically or through sexual polyploidization to make them crossable with cultivars. Alternatively, diploid species clones can be crossed to parthenogenetically derived haploids ($2n = 2x = 24$) of cultivars, followed by polyploidization. The track record for transferring disease resistance to tetraploids through sexual polyploidization is impressive. It has successfully created hybrids with resistance to bacterial wilt (*Pseudomonas solanacearum*) (Watanabe et al., 1992), early blight (Herriott et al., 1990), common scab (Murphy et al., 1995), potato cyst nematode (De Maine et al., 1986; Ortiz et al., 1997), Verticillium wilt (K.E. Frost and S.H. Jansky, unpublished), and soft rot (Carputo et al., 2000; Capo et al., 2002). Iwanaga et al. (1989) crossed root knot nematode-resistant diploids to 'Atzimba' (4x) and to a haploid (2x) of 'Atzimba', both of which are susceptible to the nematode. A significantly higher proportion of resistant offspring (25%) was obtained in the $4x \times 2x$ crosses than in the $2x \times 2x$ crosses (11%). Presumably, alleles at loci between the centromere and the first crossover on each chromosome were transferred to offspring intact in $2n$ gametes, whereas those alleles were randomly reassorted in n gametes. This may explain why $2n$ gametes transmitted resistance to bacterial wilt, root knot nematodes, late blight, and glandular trichomes to a high proportion of $4x \times 2x$ offspring (Watanabe et al., 1999).

Diploid, 1 endosperm balance number (1EBN) species are often good sources of disease resistance genes. Several strategies have been developed to tap into this germplasm resource. Carputo et al. (1997) doubled the chromosome number of *S. commersonii* and

crossed the resulting 4x, 2EBN clone with a 2x, 2EBN Tuberosum Group–Phureja Group hybrid, which was introgressed into tetraploid clones, some of which exhibited resistance to bacterial soft rot (Carputo et al., 2002; Iovene et al., 2004). Ramon and Hanneman (2002) used embryo rescue to incorporate late-blight resistance from the 2x, 1EBN species *Solanum pinnatisectum* into *Solanum tuberosum*.

Extremely wide hybridizations may introduce novel resistance genes into the potato gene pool. Colon et al. (1993) used embryo rescue to introduce late-blight resistance genes into the potato from the Solanaceous weed species *Solanum nigrum* and *Solanum villosum*. Valkonen et al. (1995) used embryo rescue to transfer the extreme resistance to PVY found in the non-tuber-bearing 2x, 1EBN species *Solanum brevidens* to the cultivated potato. Chavez et al. (1988) used bridging crosses, ploidy manipulations, and embryo rescue to transfer PLRV resistance from the non-tuber-bearing 2x, 1EBN species *Solanum etuberosum* to tuber-bearing species.

Breeding strategies may be designed to incorporate multiple sources of disease resistance genes. Solomon-Blackburn and Barker (1993) created clones with strong PLRV resistance by combining genes that limit virus multiplication with those for resistance to infection. Spitters and Ward (1988) found that resistance to potato cyst nematodes was more durable in clones with two resistance genes instead of one. Colon et al. (1995a) combined minor genes for late-blight resistance from four wild *Solanum* species with diploid Tuberosum Group clones. Murphy et al. (1999) used conventional hybridization between two tetraploid breeding clones, each with different disease resistance traits, to create a clone with resistance to several diseases. Hybrids containing a large proportion of wild germplasm may express multiple resistances because wild *Solanum* relatives are rich in disease resistance genes. Jansky and Rouse (2003) identified resistance to several diseases in populations of diploid interspecific hybrids. Chen et al. (2003) identified wild species genotypes with multiple resistances to late blight, CPB, and blackleg (*E. carotovora*). Similarly, De Maine et al. (1993) argue that Phureja Group is a valuable source of multiple disease resistance genes. The authors noted that resistance levels decrease as the exotic germplasm is diluted following recurrent backcrosses to *S. tuberosum*.

7.2.1.3 Somatic hybridization

High levels of disease resistance are sometimes found in wild *Solanum* species that are sexually incompatible (or nearly so) with the cultivated potato. Somatic fusion offers an effective strategy to access this germplasm. The 2x non-tuber-bearing species (*Solanum fernandezianum, S. brevidens*, and *S. etuberosum*) seem to be especially rich in disease resistance genes. For example, somatic hybrids between cultivated potatoes and *S. brevidens* express resistance to PLRV (Austin et al., 1985), PVX, PVY, and PLRV (Gibson et al., 1988; Valkonen and Rokka, 1998), and soft rot (Austin et al., 1988; Zimnoch-Guzowska and Łojkowska, 1993; Allefs et al., 1995; McGrath et al., 2002). Somatic fusion hybrids containing the 2x, 1EBN species *S. commersonii* are resistant to bacterial wilt (Leferriere et al., 1999; Kim-Lee et al., 2005), soft rot (Carputo et al., 1997), and Verticillium wilt (Bastia et al., 2000), whereas those with *Solanum bulbocastanum* are resistant to late blight (Helgeson et al., 1998; Song et al., 2003).

Somatic hybrids involving non-tuber-bearing species generally tuberize poorly, and as backcrosses are made to Tuberosum Group to improve tuberization, levels of resistance

may decrease (Austin et al., 1988; Allefs et al., 1995). Rokka et al. (1995) suggest that repeated cycles of somatic fusion and anther culture may provide an effective strategy to improve tuberization while retaining virus resistance. In other cases, though, resistance genes are retained and expressed following multiple backcrosses to Tuberosum Group (Novy et al., 2002; Tek et al., 2004).

Another application of somatic fusion is to combine resistance genes from two sexually compatible parents. Thach et al. (1993) fused diploids carrying major genes for resistance to PVX (*Rx*) or PVY (*Ry*). When *Rx*-carrying clones were fused with *Ry*-carrying clones, most of the hybrids were resistant to both PVX and PVY. Rasmussen et al. (1996) fused a Tuberosum Group haploid carrying the gene for resistance to potato cyst nematode pathotype *Pa2* with another haploid carrying the *Pa3* resistance gene. Some of the hybrids exhibited a high level of resistance to both pathotypes of the nematode. Valkonen and Rokka (1998) fused a *S. brevidens* clone with a Tuberosum Group clone, both of which are resistant to PVA, PVX, PVY, and CMV, but through different mechanisms. Surprisingly, the simultaneous expression of two resistance mechanisms in the hybrids resulted in variable responses to the pathogens but not typically complementation for resistance. Carrasco et al. (2000) fused *Solanum verrucosum* with Tuberosum Group to combine PLRV resistance from *S. verrucosum* with adaptation and tuber yield from Tuberosum Group.

It is interesting to note that a dilution effect is sometimes reported for resistance traits in somatic fusion hybrids. Cooper-Bland et al. (1994) fused a potato cyst nematode-susceptible Tuberosum Group haploid with a resistant one and produced hybrids that were more similar to the susceptible parent. Somatic hybrids created by Rasmussen et al. (1998) had lower levels of quantitative resistance to both tuber and foliar late blight than the resistant donors. Similarly, soft rot resistance was reduced in backcross generations compared with *S. brevidens* + Tuberosum Group fusions (McGrath et al., 2002) and *S. commersonii* + Tuberosum Group fusions (Carputo et al., 2002). Gavrilenko et al. (2003) reported that 6x somatic fusion hybrids with four doses of the resistant parent (*S. etuberosum*) genome expressed extreme resistance to PVY, whereas 6x hybrids with four doses of the susceptible parent (Tuberosum Group) genome were susceptible. On the contrary, Zimnoch-Guzowska et al. (2003) reported that somatic hybrids produced from fusions of Tuberosum Group with the wild species *S. nigrum* were often more resistant than the resistant wild parent, perhaps due to complementation of resistance genes.

7.2.2 Resistance genetics based on disease phenotype

7.2.2.1 Vertical resistance

Disease resistance genetic studies are often based on tetraploid families. However, even major genes are difficult to identify at the tetraploid level due to the complexities of tetrasomic segregation. For example, Brown et al. (1997) intercrossed tetraploid Tuberosum Group clones and obtained high heritability estimates for PLRV resistance. The authors suggest that a few major genes are mainly responsible for resistance, but individual genes and their effects could not be elucidated. Based on crosses among tetraploid Tuberosum Group clones, Barker et al. (1994) proposed a two-gene model for PLRV resistance.

Barker and Solomon (1990) observed an approximately 1:1 segregation ratio in a cross between a susceptible tetraploid clone and a clone with resistance to PLRV multiplication. They suggest that a single dominant gene may confer resistance, but it was not possible to determine the genotypes of the parents. In contrast, when Brown and Thomas (1994) carried out inheritance studies at the diploid level, with the wild species *S. chacoense*, a single dominant resistance gene was identified and parental genotypes were determined based on offspring ratios.

Major genes for resistance to other potato viruses have also been identified. Solomon-Blackburn and Barker (2001) listed 28 major genes/alleles responsible for virus resistance in potato. Most originate from wild *Solanum* species, although Tuberosum and Stenotomum Groups have also contributed resistance genes. Some virus resistance genes may be found in closely linked clusters or they may be single genes that confer broad-spectrum resistance. For example, Barker (1997) suggested that a single dominant gene confers resistance to PVA, PVX, and PVY.

Dominant R-genes[1] for late-blight resistance have been used by breeders for decades. Eleven R-gene alleles have been identified in the Mexican wild species *S. demissum* (Black et al., 1953). An additional resistance gene, *Rpi1*, has been identified in the Mexican wild species *S. pinnatisectum* (Kuhl et al., 2001). Characterization of this gene indicates that it might correspond to R9 from *S. demissum*. These resistance genes are presumed to have evolved as a result of long-term interactions between the host wild species and the late-blight pathogen in Mexico. It is interesting that R-genes have been identified in Argentinian wild species even though these species have not evolved in the presence of *P. infestans* (Micheletto et al., 1999). In breeding studies using clones with major gene resistance, an excess of susceptible progeny is sometimes detected in segregating populations. A dominant suppressor of R-genes has been proposed to explain these data (El-Kharbotly et al., 1996b; Ordoñez et al., 1997). A long history of R-gene-resistant cultivar releases has demonstrated that vertical resistance to late blight is not durable because the pathogen is capable of evolving quickly to overcome R-genes.

Surprisingly, a single late-blight resistance gene derived from *S. bulbocastanum, RB*, appears to differ from other R-genes by conferring durable resistance. Clones containing the *RB* gene are even resistant to 'super-races' of the fungus that can overcome all 11 R-genes (Song et al., 2003; van der Vossen et al., 2003).

It is interesting that there are several examples of two-gene disease resistance systems in potato. Vallejo et al. (1995) suggested that PVY resistance in a diploid Phureja–Stenotomum Group population is controlled by complementary action of two dominant genes. Both genes must be present to confer resistance. Similarly, Kriel et al. (1995b)

[1] Classification of the genes involved in plant resistance can sometimes be confusing with different authors using somewhat different terminology. In this chapter, the term 'R-gene' refers to a phenotypically defined single gene that specifically recognizes an avirulence gene product of the pathogen. The R-gene-based type of resistance is often described in literature as monogenic, qualitative, vertical, narrow, specific, or complete. On the contrary, if the resistance phenotype is not controlled by individually recognizable R-genes and the resistance is measured quantitatively rather than qualitatively, the loci associated with the resistance are referred to as 'quantitative resistance loci' (QRL). The QRL-based type of resistance is frequently described as polygenic, quantitative, horizontal, broad, general, partial, or field resistance. However, the distinction between the two types of resistance is often equivocal at the phenotypic level.

found that complementary gene action is responsible for resistance to ring rot in *S. acaule*. In addition, Singh et al. (2000) determined that resistance to PVA in potato cultivars due to two independent genes with complementary gene action. Vallejo et al. (1995) also found that two dominant genes control resistance to PVX in Phureja–Stenotomum Group hybrids. However, this system exhibits duplicate dominant epistasis, as only one of the two genes is necessary for resistance. Jansky et al. (2004) also reported that a similar genetic mechanism exists for resistance to Verticillium wilt in diploid interspecific hybrids. Conversely, Lynch et al. (1997) identified a single dominant gene resistance is sufficient for Verticillium wilt resistance in *S. chacoense*. Perhaps a second resistance gene could not be detected because it was not segregating in that population.

7.2.2.2 *Horizontal resistance*

There is considerable interest in the development of potato cultivars with durable late-blight resistance due to polygenes. The so-called field resistance is complexly inherited and may involve the production of phytoalexins, phenolics, and glycoalkaloids (Andreu et al., 2001). Breeding progress has been slow because the genetic basis of resistance is not yet understood. Horizontal resistance to late blight, presumably due to minor genes, has been reported in wild *Solanum* species (Rivera-Peña, 1990; Colon et al., 1995b), in cultivated relatives of potato (Cañizares and Forbes, 1995; Haynes and Christ, 1999; Trognitz et al., 2001; Zlesak and Thill, 2004), and in Tuberosum Group cultivars (Colon et al., 1995b; Grünwald et al., 2002; Porter et al., 2004). Late-blight resistance is present in some heirloom cultivars and appears to be durable, because the resistance levels are similar to those when the cultivars were released more than 40 years ago. However, the most resistant cultivars are also late-maturing, and it is not known whether durable resistance can be combined with early maturity.

Resistance to early blight (*A. solani*) appears to be quantitatively inherited. Good sources of resistance are rare, and the combination of resistance and early maturity is even more difficult to find (Boiteux et al., 1995). Christ and Haynes (2001) reported a relatively high heritability estimate (0.61) for early-blight resistance in a Phureja–Stenotomum Group population, indicating that additive genetic variance is important. Similarly, Herriott et al. (1990) and Ortiz et al. (1993) found that additive genetic variance is important for early-blight resistance. According to Brandolini et al. (1992) and Gopal (1988), non-additive gene action contributes to resistance as well. In most populations developed for early-blight resistance, the most resistant clones are late to mature. However, Boiteaux et al. (1995) surveyed a large number of clones (934) and found some with both early maturity and early-blight resistance.

A genetic study using diploid interspecific hybrids determined that broad- and narrow-sense heritability values for soft rot resistance are high (0.92 and 0.89, respectively) (Lebecka and Zimnoch-Guzowska, 2004). Consequently, additive genetic variance is more important than non-additive variance. Although individual resistance genes were not identified in this study, the high heritability estimates may indicate that only a few genes control resistance.

Several studies have identified quantitative resistance for Verticillium wilt. Tsror and Nachmias (1995) suggest that minor genes are responsible for resistance in some cultivars.

Pavek and Corsini (1994) also favor a horizontal resistance model, with additive genetic variance contributing significantly to resistance. Recently, Simko et al. (2004b) identified four quantitative trait loci that contribute to Verticillium wilt resistance in diploid populations. It is interesting to note that different types of Verticillium wilt resistance may be controlled by different genetic systems. Lynch et al. (1997) believe that, in diploid *S. chacoense*, tolerance is a polygenic trait, whereas resistance to infection and colonization is due to a major gene.

7.3 MOLECULAR ANALYSIS OF POTATO RESISTANCE

7.3.1 Experimental strategies for gene mapping and cloning

7.3.1.1 Mapping plant resistance genes

The mapping of plant resistance genes is typically carried out on segregating populations derived from parents with contrasting phenotypes. To localize genes associated with particular resistance on a molecular linkage map, the resistance phenotype has to be assessed for the individuals in the mapping population. Then, linkage between marker loci and the resistance trait is calculated. Unfortunately, the cultivated potato (*S. tuberosum* ssp. *tuberosum*) is a highly heterozygous autotetraploid ($2n = 4x = 48$) species with complex genetic inheritance that complicates gene mapping. To limit the complexity of potato genetics, diploid ($2n = 2x = 24$) individuals are frequently used as parents for molecular map construction and linkage analysis. Diploids can be derived from tetraploid genotypes through anther or pollen culture, or through interspecific hybridization with certain genotypes of *Solanum phureja* ($2n = 2x = 24$). However, the diploid potato genotypes are self-incompatible (or are having large inbreeding depression), the feature that precludes development of pure lines. Therefore, a number of common mapping approaches based on homozygous lines, and often used in plant genetics, cannot be applied in potato. On the contrary, because alleles of heterozygous parents segregate in meiosis, already an F1-hybrid population can be used for potato gene mapping.

Typically, the initial screening is performed on a population consisting of 100–200 individuals and a series of markers ideally spaced at even intervals of about 10 cM on each chromosome. The necessary requirement for gene detection is a phenotype that segregates within the population and can be clearly scored in each individual. Following detection of linkage between the resistance phenotype and a molecular marker, saturation of the genomic region with more markers can be carried out on an expanded population of several hundred or even thousands of individuals. The large population size allows detection of flanking markers that are more closely linked to the resistance gene. Closely linked markers then may be used for either marker-assisted selection (MAS) or the map-based cloning of the resistance gene.

It should be noted, however, that populations originating from two diploid parental genotypes sample only a small proportion of all possible alleles. Moreover, if wild species are used for the development of mapping populations, the observed gene effects are not often representative of those encountered in elite cultivars. Detecting variation

in economically important traits within genetic backgrounds that are relevant to plant breeders can be improved by complementary mapping techniques, such as association mapping. The association mapping method is a linkage disequilibrium (LD)-based technique that exploits biodiversity observed in existing cultivars and breeding lines without developing new mapping populations. This method has previously been used in diploid species and was recently successfully applied to map resistance genes in tetraploid potato (Gebhardt et al., 2004; Simko, 2004; Simko et al., 2004a,b). The association mapping approach effectively incorporates the effect of many past generations of recombinants into a single analysis. Because no mapping population needs to be created for the study, the linkage test can be performed relatively quickly and inexpensively. The association mapping technique that provides the means for detecting genes underlying the variation of a trait among existing genotypes is thus complementary to linkage-mapping methods that effectively locate genes segregating in a population originating from two individuals. A significant difference between association mapping in a general population and genetic linkage mapping in a defined segregating population is that association mapping generally identifies the association of common alleles (rare alleles do not reach statistical significance), whereas a population originating from a biparental cross enables the identification of alleles rare in the population at large (Simko, 2004). The resolution of the association-mapping approach depends on the structure of LD within the test population. The extent of LD in potato is not yet known; however, preliminary analysis of 66 loci indicates a relatively fast decay of LD within 1kb, but slow decay afterwards (Simko et al., 2006b).

7.3.1.2 Map-based cloning of resistance genes

Thus far, eight potato resistance genes have been cloned, and all of them were isolated using variants of the map-based strategy often combined with the candidate gene approach. Therefore, we will describe briefly this cloning technique, although other approaches can also be used for gene isolation.

After detecting molecular markers that are flanking a resistance gene locus, the screening of a large-insert genomic library (usually a bacterial artificial chromosome – BAC) with the identified flanking markers is performed. If the flanking markers are separated by a large distance, then the 'chromosome walking' method (Bender et al., 1983) is used to identify cloned DNA between the two markers through a stepwise analysis of successive overlapping clones. Alternatively, the 'chromosome landing' (Tanksley et al., 1995) approach may be used to pinpoint a resistance gene location with more accuracy. The idea behind the chromosome-landing method is that several thousand markers can be screened on the population to search for markers at a genetic distance corresponding to the physical size of large-insert clones. Identified markers are then used for screening genomic libraries. Ideally, the markers will land directly in the clones that contain, among other genes, the target resistance gene.

Once a large-insert clone(s) that encompasses the resistance gene is detected, the resistance gene identity needs to be established. This is usually done through a combination of sequencing, bioinformatic analyses (to reveal relevant candidates), and functional testing of the candidate alleles in transgenic plants.

7.3.2 Resistance factors mapped in potato

Numerous genes conferring resistance to viruses, nematodes, bacteria, fungi, oomycetes, and insects have been mapped in potato, and their location is summarized in Tables 7.2 and 7.3.

Two common types of single gene resistance to viruses in potato are hypersensitive resistance and extreme resistance. The genes for hypersensitive resistance are often virus strain group specific. When plants carrying these genes are inoculated with viruses, they usually develop either local necrotic lesions in the infected tissue or systemic necrosis. Several genes coding hypersensitive resistance to potato viruses A, S, X, and Y have been mapped in potato. On the contrary, very limited (or no) necrosis is observed on plants having genes for extreme resistance. The extreme resistance genes confer comprehensive resistance to several virus strains, and only an extremely low level of virus can be detected in some of the inoculated plants. Genes for extreme resistance to PVX and PVY originating from at least four different potato species have been placed on the potato molecular map. QRL for resistance to PLRV have also been reported in some mapping progenies.

Resistance genes to three economically important species of nematodes have been mapped in potato. Two of the species (*Globodera rostochiensis* and *G. pallida*) are root cyst nematodes, whereas *M. chitwoodi* is a root knot nematode. The first nematode resistance gene (*H1*) was discovered in the 1950s (Toxopeus and Huijsman, 1953), and since then, it has been introgressed into many commercially available cultivars to control *G. rostochiensis* pathotypes. The gene is located on potato chromosome 5. Additional dominant genes for qualitative resistance to *G. rostochiensis* and *G. pallida* have been mapped, together with several major QRL. However, only a single resistance locus against *M. chitwoodi* species has been identified so far in the potato genome. The R_{Mcl} gene from *S. bulbocastanum* was introgressed into cultivated potato by somatic hybridization (Brown et al., 1996). It was demonstrated later that the resistance spectrum of R_{Mcl} includes not only *M. chitwoodi* and the related species *Meloidogyne fallax* but also a genetically distinct population of *Meloidogyne hapla* (Rouppe van der Voort et al., 1999).

Quantitative resistance loci against bacteria *E. carotovora* ssp. *atroseptica*, a causal agent (together with other *Erwinia* species) of potato black leg and tuber soft rot, were detected in a diploid population with complex pedigree that included three *Solanum* species: *Solanum yungasense, S. tuberosum*, and *S. chacoense*. Genetic factors affecting resistance to *E. carotovora* ssp. *atroseptica* were found on all 12 potato chromosomes (Zimnoch-Guzowska et al., 2000).

Potato is affected by a number of fungal pathogens; however, only a limited number generate major loss of a crop. As of 2006, monogenic resistance to *Synchytrium endobioticum* (potato wart), and QRL for resistance to *A. solani* (early blight), *V. dahliae*, and *V. albo-atrum* (Verticillium wilt) have been identified.

The most economically important disease of potato is late blight caused by oomycete *P. infestans*. Twenty R-genes, conferring potato foliage resistance against late blight, have been placed on a molecular map so far. Eleven of the genes (*R1, R2, R3a, R3b, R5–R11*) come from *S. demissum*, four genes (*RB/Rpi-blb1, Rpi-blb2, Rpi-blb3, Rpi-abpt*) from *S. bulbocastanum*, and one each from *S. berthaultii* (R_{ber}/R_{Pi-ber}), *S. pinnatisectum*

Table 7.2 R-genes mapped in potato.

Chromosome	R-gene	Anchor marker	Pathogen resistance	Source of resistance	Reference	Class of resistance proteins	Reference
4	*Ny_{tbr}*	TG316	PVY	*Solanum tuberosum* ssp. *tuberosum*	(Celebi-Toprak et al., 2002)		
4	*Rpi-abpt*	TG370	*Phytophthora infestans*	*Solanum bulbocastanum* ssp. *bulbocastanum*	(Park et al., 2005c)		
4	*Rpi-blb3*	TG370	*P. infestans*	*S. bulbocastanum* ssp. *dolichophyllum*	(Park et al., 2005a)		
4	*R2*	TG370	*P. infestans*	*Solanum demissum*	(Li et al., 1998)		
4	*R2*-like	TG370	*P. infestans*	–	(Park et al., 2005b)		
5	*R1*	GP21	*P. infestans*	*S. demissum*	(Leonards-Schippers et al., 1992)	CC–NBS–LRR	(Ballvora et al., 2002)
5	*Nb*	GP21	PVX	–	(De Jong et al., 1997)		
5	*Rx2*	GP21	PVX	*Solanum acaule*	(Ritter et al., 1991)	CC–NBS–LRR	(Bendahmane et al., 2000)
5	*Grp1*	GP21	*Globodera rostochiensis, Globodera pallida*	–	(Rouppe van der Voort et al., 1998)		
5	*H1*	CP113	*G. rostochiensis*	*S. tuberosum* ssp. *andigena*	(Gebhardt et al., 1993; Pineda et al., 1993)		
5	*GroV1*	TG69	*G. rostochiensis*	*Solanum vernei*	(Jacobs et al., 1996)		
6	*Rpi-blb2*	CT119	*P. infestans*	*S. bulbocastanum*	(van der Vossen et al., 2005)	CC–NBS–LRR	(van der Vossen et al., 2005)

(*Continued*)

Table 7.2 (*Continued*)

Chromosome	R-gene	Anchor marker	Pathogen resistance	Source of resistance	Reference	Class of resistance proteins	Reference
7	*Rpi1*	TG20a	*P. infestans*	*Solanum pinnatisectum*	(Kuhl et al., 2001)		
7	*Gro1*	CP56	*G. rostochiensis*	*Solanum spegazzinii*	(Barone et al., 1990)	TIR–NBS–LRR	(Paal et al., 2004)
8	*RB/Rpi-blb1*	CP53	*P. infestans*	*S. bulbocastanum*	(Naess et al., 2000)	CC–NBS–LRR	(Song et al., 2003; van der Vossen et al., 2003)
8	*Ns*	CP16	PVS	*S. tuberosum* ssp. *andigena*	(Marczewski et al., 1998, 2002)		
9	Nx_{phu}	TG424	PVX	*Solanum phureja*	(Tommiska et al., 1998)		
9	*Rpi-moc1*	TG328	*P. infestans*	*Solanum mochiquense*	(Smilde et al., 2005)		
9	Ry_{chc}	CT220	PVY	*Solanum chacoense*	(Hosaka et al., 2001; K. Hosaka, personal communication)		
10	R_{ber}/R_{Pi-ber}	TG63	*P. infestans*	*Solanum berthaultii*	(Ewing et al., 2000; Rauscher et al., 2006)		
11	Ry_{adg}	CP58	PVY	*S. tuberosum* ssp. *andigena*	(Hämäläinen et al., 1997)		
11	Ry_{sto}[a]	CP58	PVY	*Solanum stoloniferum*	(Brigneti et al., 1997)		
11	*Sen1*	CP58	*Solanum endobioticum*	–	(Hehl et al., 1999)		
11	R_{Mc1}	TG523	*M. chitwodii*	*S. bulbocastanum*	(Brown et al., 1996)		

11	Na_{adg}	TG523	PVA	*S. tuberosum* ssp. *andigena*	(Hämäläinen et al., 2000)		
11	*R3a*	GP185	*P. infestans*	*S. demissum*	(El-Kharbotly et al., 1994; Huang et al., 2004)	CC–NBS–LRR	(Huang et al., 2005)
11	*R3b*	GP185	*P. infestans*	*S. demissum*	(El-Kharbotly et al., 1994; Huang et al., 2004)		
11	*R5*	GP185	*P. infestans*	*S. demissum*	(Huang, 2005)		
11	*R6*	GP185	*P. infestans*	*S. demissum*	(El-Kharbotly et al., 1996a)		
11	*R7*	GP185	*P. infestans*	*S. demissum*	(El-Kharbotly et al., 1996a)		
11	*R8*	GP185	*P. infestans*	*S. demissum*	(Huang, 2005)		
11	*R9*	GP185	*P. infestans*	*S. demissum*	(Huang, 2005)		
11	*R10*	GP185	*P. infestans*	*S. demissum*	(Huang, 2005; Bradshaw et al., 2006)		
11	*R11*	GP185	*P. infestans*	*S. demissum*	(Huang, 2005; Bradshaw et al., 2006)		
12	$Ry-f_{sto}$[b]	GP122	PVY	*S. stoloniferum*	(Flis et al., 2005)		
12	Ry_{sto}[b]	STM0003d	PVY	*S. stoloniferum*	(Song et al., 2005)		
12	*Gpa2*	GP34	*G. pallida*	*S. tuberosum* ssp. *andigena*	(Rouppe van der Voort et al., 1997)	CC–NBS–LRR	(van der Vossen et al., 2000)
12	*Rx*[c]	GP34	PVX	*S. tuberosum* ssp. *andigena*	(Bendahmane et al., 1997)	CC–NBS–LRR	(Bendahmane et al., 1999)
12	*Rx1*[c]	GP34	PVX	*S. tuberosum* ssp. *andigena*	(Ritter et al., 1991)		

[a] Song et al. (2005) dispute position of this gene.
[b] $Ry-f_{sto}$ and Ry_{sto} genes might be identical.
[c] *Rx* and *Rx1* genes might be identical.

Table 7.3 Quantitative resistance loci (QRL) mapped in potato.

Chromosome	Pathogen resistance	Tested tissue	Reference
1	*Phytophthora infestans*	Foliage	(Collins et al., 1999; Oberhagemann et al., 1999; Simko, 2002)
1	*Erwinic carotovora* ssp. *atroseptica*	Foliage	(Zimnoch-Guzowska et al., 2000)
1	*E. carotovora* ssp. *atroseptica*	Tubers	(Zimnoch-Guzowska et al., 2000)
1	*Leptinofarsa decemlineata*	Foliage	(Yencho et al., 1996)
2	*P. infestans*	Foliage	(Leonards-Schippers et al., 1994; Collins et al., 1999)
2	*P. infestans*	Tubers	(Collins et al., 1999; Simko et al., 2006a)
2	*E. carotovora* ssp. *atroseptica*	Foliage	(Zimnoch-Guzowska et al., 2000)
2	*E. carotovora* ssp. *atroseptica*	Tubers	(Zimnoch-Guzowska et al., 2000)
2	*Verticillium albo-atrum*	Roots[a]	(Simko et al., 2004b)
3	*P. infestans*	Foliage	(Leonards-Schippers et al., 1994; Collins et al., 1999; Oberhagemann et al., 1999; Ewing et al., 2000; Ghislain et al., 2001; Bink et al., 2002; Visker et al., 2003; Bormann et al., 2004; Costanzo et al., 2005)
3	*E. carotovora* ssp. *atroseptica*	Foliage	(Zimnoch-Guzowska et al., 2000)
3	*E. carotovora* ssp. *atroseptica*	Tubers	(Zimnoch-Guzowska et al., 2000)
3	*Globodera rostochiensis*	Roots	(Kreike et al., 1996)
4	*P. infestans*	Foliage	(Leonards-Schippers et al., 1994; Meyer et al., 1998; Collins et al., 1999, Oberhagemann et al., 1999; Sandbrink et al., 2000; Bormann et al., 2004; Bradshaw et al., 2004a,b)[(b)]
4	*E. carotovora* ssp. *atroseptica*	Foliage	(Zimnoch-Guzowska et al., 2000)
4	*E. carotovora* ssp. *atroseptica*	Tuber	(Zimnoch-Guzowska et al., 2000)
4	*G. pallida*	Roots	(Bradshaw et al., 1998; Bryan et al., 2004)
4	*Alternaria solani*	Foliage	(Zhang, 2004)
5	*P. infestans*	Foliage	(Leonards-Schippers et al., 1994; van Eck and Jacobsen, 1996; Collins et al., 1999; Oberhagemann et al., 1999; Sandbrink et al., 2000; Ghislain et al., 2001; Simko, 2002; Visker et al., 2003; Bormann et al., 2004; Bradshaw et al., 2004a,b; Costanzo et al., 2005; Mayton et al., 2005)

5	*P. infestans*	Tuber	(Collins et al., 1999; Oberhagemann et al., 1999; Bradshaw et al., 2004a,b; Mayton et al., 2005)
5	*E. carotovora* ssp. *atroseptica*	Foliage	(Zimnoch-Guzowska et al., 2000)
5	*E. carotovora* ssp. *atroseptica*	Tuber	(Zimnoch-Guzowska et al., 2000)
5	*G. pallida*	Roots	(Kreike et al., 1994; Rouppe van der Voort et al., 2000; Bryan et al., 2002; Caromel et al., 2003, 2005)
5	*A. solani*	Foliage	(Zhang, 2004)
5	PLRV	Foliage	(Marczewski et al., 2001)
5	*L. decemlineata*	Foliage[c]	(Yencho et al., 1996)
6	*P. infestans*	Foliage	(Leonards-Schippers et al., 1994; Collins et al., 1999; Oberhagemann et al., 1999)
6	*P. infestans*	Tubers	(Simko et al., 2006a)
6	*E. carotovora* ssp. *atroseptica*	Foliage	(Zimnoch-Guzowska et al., 2000)
6	*E. carotovora* ssp. *atroseptica*	Tubers	(Zimnoch-Guzowska et al., 2000)
6	*G. pallida*	Roots	(Caromel et al., 2003)
6	*V. albo-atrum*	Roots[a]	(Simko et al., 2004b)
6	PLRV	Foliage	(Marczewski et al., 2001)
7	*P. infestans*	Foliage	(Leonards-Schippers et al., 1994; Collins et al., 1999; Ghislain et al., 2001; Costanzo et al., 2005)
7	*E. carotovora* ssp. *atroseptica*	Foliage	(Zimnoch-Guzowska et al., 2000)
8	*P. infestans*	Foliage	(Collins et al., 1999; Oberhagemann et al., 1999; Ghislain et al., 2001; Simko, 2002; Bormann et al., 2004)
8	*P. infestans*	Tubers	(Simko et al., 2006a)
8	*E. carotovora* ssp. *atroseptica*	Foliage	(Zimnoch-Guzowska et al., 2000)
8	*L. decemlineata*	Foliage[c]	(Yencho et al., 1996)
9	*P. infestans*	Foliage	(Leonards-Schippers et al., 1994; Collins et al., 1999; Oberhagemann et al., 1999; Simko, 2002; Bormann et al., 2004)

(*Continued*)

Table 7.3 (*Continued*)

Chromosome	Pathogen resistance	Tested tissue	Reference
9	*P. infestans*	Tuber	(Collins et al., 1999)
9	*E. carotovora* ssp. *atroseptica*	Foliage	(Zimnoch-Guzowska et al., 2000)
9	*E. carotovora* ssp. *atroseptica*	Tuber	(Zimnoch-Guzowska et al., 2000)
9	*V. albo-atrum*	Roots[a]	(Simko et al., 2004b)
9	*V. dahliae*	Roots[a]	(Simko et al., 2004a)
9	*G. pallida*	Roots	(Rouppe van der Voort et al., 2000; Bryan et al., 2002)
9	*A. solani*	Foliage	(Zhang, 2004)
10	*P. infestans*	Foliage	(Sandbrink et al., 2000; Mayton et al., 2005)
10	*P. infestans*	Tubers	(Mayton et al., 2005; Simko et al., 2006a)
10	*E. carotovora* ssp. *atroseptica*	Foliage	(Zimnoch-Guzowska et al., 2000)
10	*E. carotovora* ssp. *atroseptica*	Tubers	(Zimnoch-Guzowska et al., 2000)
10	*G. rostochiensis*	Roots	(Kreike et al., 1993)
10	*L. decemlineata*	Foliage[c]	(Yencho et al., 1996)
11	*P. infestans*	Foliage	(Leonards-Schippers et al., 1994; Collins et al., 1999; Oberhagemann et al., 1999; Ghislain et al., 2001; Bormann et al., 2004; Costanzo et al., 2005)
11	*E. carotovora* ssp. *atroseptica*	Foliage	(Zimnoch-Guzowska et al., 2000)
11	*E. carotovora* ssp. *atroseptica*	Tubers	(Zimnoch-Guzowska et al., 2000)
11	*G. rostochiensis*	Roots	(Kreike et al., 1993)
11	*G. pallida*	Roots	(Bryan et al., 2004; Caromel et al., 2005)
11	PLRV	Foliage	(Marczewski et al., 2001; Marczewski et al., 2004)
11	*A. solani*	Foliage	(Zhang, 2004)
12	*P. infestans*	Foliage	(Leonards-Schippers et al., 1994; Collins et al., 1999; Ghislain et al., 2001; Bormann et al., 2004)
12	*E. carotovora* ssp. *atroseptica*	Tubers	(Zimnoch-Guzowska et al., 2000)
12	*G. pallida*	Roots	(Caromel et al., 2003)
12	*V. albo-atrum*	Roots[a]	(Simko et al., 2004b)
12	*A. solani*	Foliage	(Zhang, 2004)

[a] Fungus infects plant through roots, but the disease symptoms were observed on foliage.
[b] Meyer et al. (1998) originally detected the resistance locus on chromosome 8. The locus was later placed on chromosome 4.
[c] Possibly, a trichome-related resistance.

(*Rpi1*), and *Solanum mochiquense* (*Rpi-moc1*). Nine R-genes (*R3a, R3b, R5–R11*) are clustered in the late-blight resistance hotspot on the distal part of chromosome 11. In addition to phenotypically characterized classical R-genes originating from *S. demissum* (Black et al., 1953), several of the fully functional allelic versions or duplications of the resistance genes were detected on chromosomes 4 (*R2-like*) (Park et al., 2005b) and 11 (*SH R3, Ma R3, Sc R3*, and *FS R3*) (Huang et al., 2005).

Polygenic factors affecting foliage resistance to late blight have been identified on all 12 chromosomes. Most of the QRL have a relatively small or moderate effect on the resistance, although about one-tenth of the mapped QRL explain 30–50% of the trait variation. Rarely, the detected QRL explains more than half of the total phenotypic variation. Because QRL are positioned on the molecular map with less precision than single genes and the confidence interval of the QRL position may exceed 40 cM (Simko, 2002), it is problematic to compare the location of resistance QRL from different studies. Nevertheless, when the likelihood of QRL being detected by different studies in the same general genomic region was estimated from a binomial distribution, regions on chromosomes 3, 4, and 5 were identified with a high probability as late-blight resistance hotspots (Simko, 2002; Jones and Simko, 2005). The most obvious and consistent resistance hotspot is located on chromosome 5, near the marker locus GP179. Unfortunately, plants having alleles at this locus, which increases foliage resistance, usually exhibit later maturity. Linkage between resistance and maturity at this chromosomal location was confirmed by association mapping performed on almost 600 cultivars. In this study, the markers tightly linked to the *R1* gene were significantly associated with quantitative resistance to late blight and late maturity (Gebhardt et al., 2004). There is a possibility that a single gene near the GP179 marker locus has a pleiotropic effect on both plant resistance and maturity (Bradshaw et al., 2004b). However, the presence of two tightly linked loci cannot be ruled out with one locus having a pleiotropic effect on both late-blight resistance and foliage maturity, and another having merely an effect on resistance (Visker et al., 2003).

While several genes have been mapped for foliage resistance to late blight, there is relatively little information about the genes affecting resistance in tubers. In many cases, the relationship between late-blight resistance in foliage and tuber can be ambiguous. Oberhagemann et al. (1999) hypothesized that a differential expression in leaves and tubers of multiple alleles and allele combinations results in a differential effect on late-blight resistance in the two tissues.

Very little information has been published concerning natural insect resistance loci in potato. In one study, two reciprocal backcross *S. tuberosum* × *S. berthaultii* potato progenies were screened for resistance to CPB consumption, oviposition, and defoliation (Yencho et al., 1996). Most of the QRL for resistance to CPB were linked to the loci for glandular trichome traits (Bonierbale et al., 1994). However, a relatively strong and consistent QRL for trichome-independent insect resistance was observed in both backcross populations on chromosome 1 (Yencho et al., 1996).

Depicting the location of known R-genes and QRL on the potato linkage map reveals clustering of resistance genes (Fig. 7.1). These hotspots contain multiple gene families conferring resistance to a range of different pathogens. The most prominent R-gene clusters are located on chromosomes 4, 5, 9, 11, and 12. Such clustered resistance gene families

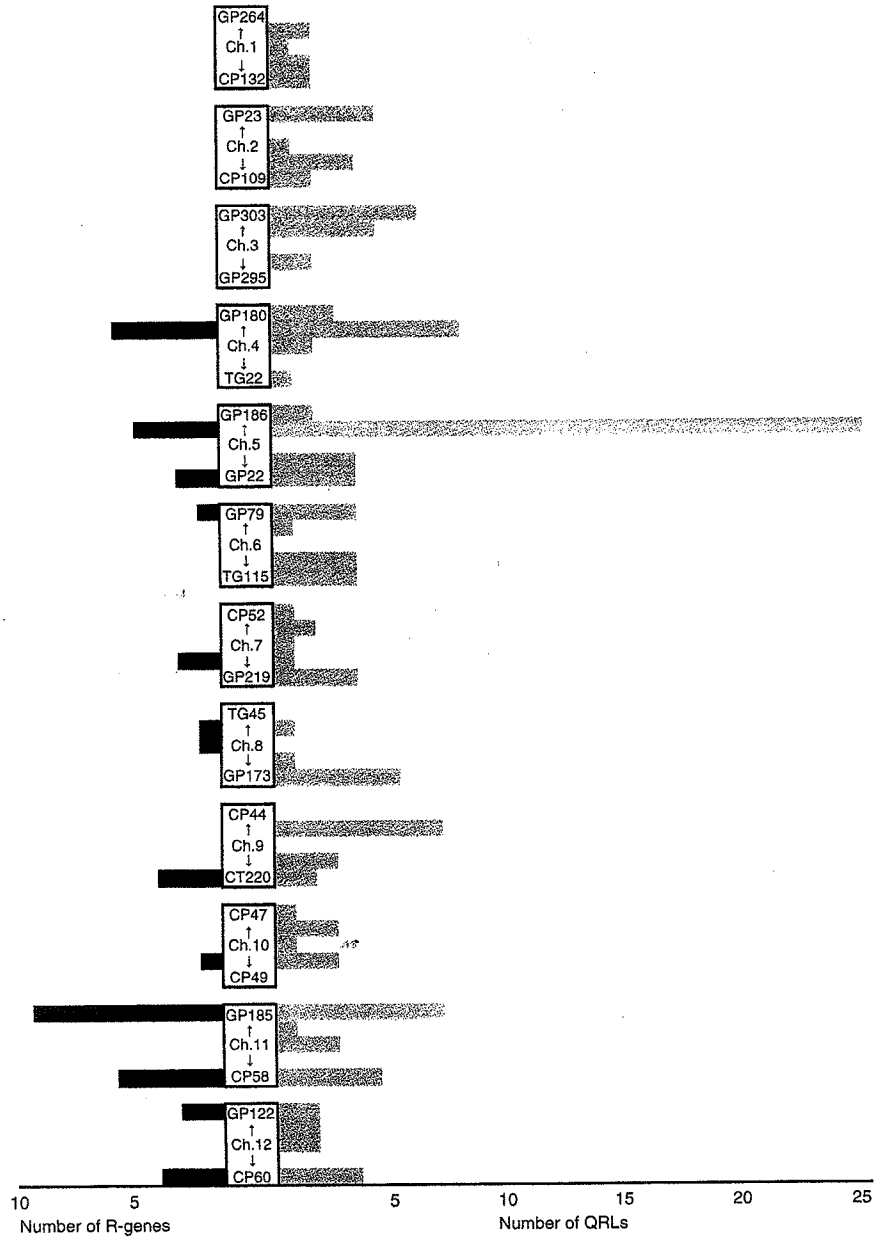

Fig. 7.1. Genomic position of R-genes (black bars) and quantitative resistance loci (QRL) (gray bars) conferring resistance to potato pathogens. Relative location of the loci associated with resistance to viruses, bacteria, fungi, oomycetes, and nematodes (Tables 7.2 and 7.3) was estimated from anchor markers. Loci coding mechanically different (trichome-related) type of resistance to insects are not included. Two restriction fragment length polymorphism (RFLP) markers per chromosome indicate orientation of the molecular linkage map according to Dong et al. (2000). More information about resistance loci is available in Tables 7.2 and 7.3. Ch., chromosome.

likely evolved from common ancestors by means of gene duplication with subsequent structural and functional diversification. Integrating the position of genes for quantitative resistance into the functional map shows a positional linkage between clusters of R-genes and QRL. A major QRL cluster on chromosome 5 comprises loci conferring resistance to virus, fungi, oomycete, bacteria, and nematodes. As new resistance genes are continuously mapped, the reader is referred to the potato functional map of pathogen resistance in the PoMaMo (Meyer et al., 2005) database (https://gabi.rzpd.de/PoMaMo.html).

7.3.3 Resistance genes cloned and characterized

Plant R-genes are presumed to enable the detection of avirulence (*Avr*) gene-specified pathogen molecules, initiate signal transduction to activate defenses, and have the capacity to evolve new specificities rapidly (Hammond-Kosack and Jones, 1997). Despite interactions with a wide range of pathogens, plant R-genes encode only five classes of resistance proteins. The majority of the cloned R-genes encode proteins containing a predicted nucleotide-binding site (NBS) followed by a series of leucine-rich repeats (LRR) at their C termini. NBS–LRR resistance proteins generally contain one of two types of N-terminal domains that have homology with the Toll and interleukin-1 receptor (TIR) proteins or a predicted coiled-coil domain (CC) (Ellis et al., 2000; Pan et al., 2000b; Dangl and Jones, 2001). The other four classes encode LRR, CC, kinase, and LRR plus kinase-conserved domains (Dangl and Jones, 2001) (Fig. 7.2). As of 2006, eight different resistance genes have been cloned from potato, and all of them are members of the NBS–LRR superfamily. The cloned genes are involved in the recognition of avirulence factors of viruses, nematodes, and oomycetes. It has been estimated that the potato genome contains at least 100–200 genes of this class (Gebhardt and Valkonen, 2001).

7.3.3.1 Virus resistance genes Rx *and* Rx2

The first resistance gene that was cloned and sequenced from potato was the *Rx* gene for extreme resistance against PVX (Bendahmane et al., 1999). The *Rx*-mediated extreme resistance in potato does not involve a necrotic hypersensitive response at the site of initial infection; however, the *Rx* protein is structurally similar to products of disease resistance genes conferring the hypersensitive response (Bendahmane et al., 1999). The highest degree of similarity is between *Rx* and a subclass of CC–NBS–LRR resistance proteins. Sequence analysis has revealed that *Rx* encodes a protein of 937 amino acid residues with molecular weight of 107.5 kDa (Bendahmane et al., 1999). The *Rx* locus has been introgressed into a cultivated *S. tuberosum* potato from *Solanum andigena* and maps to chromosome 12 (Bendahmane et al., 1997). In contrast, *Rx2*, the second PVX resistance gene cloned from potato (Bendahmane et al., 2000), was introgressed from *S. acaule* and is located on chromosome 5 (Ritter et al., 1991). The two genes share 95% sequence identity. Based on sequence conservation in *Rx* and *Rx2*, it is clear that there is a direct evolutionary relationship between these proteins, though the proteins are encoded on different chromosomes of different species (Bendahmane et al., 2000).

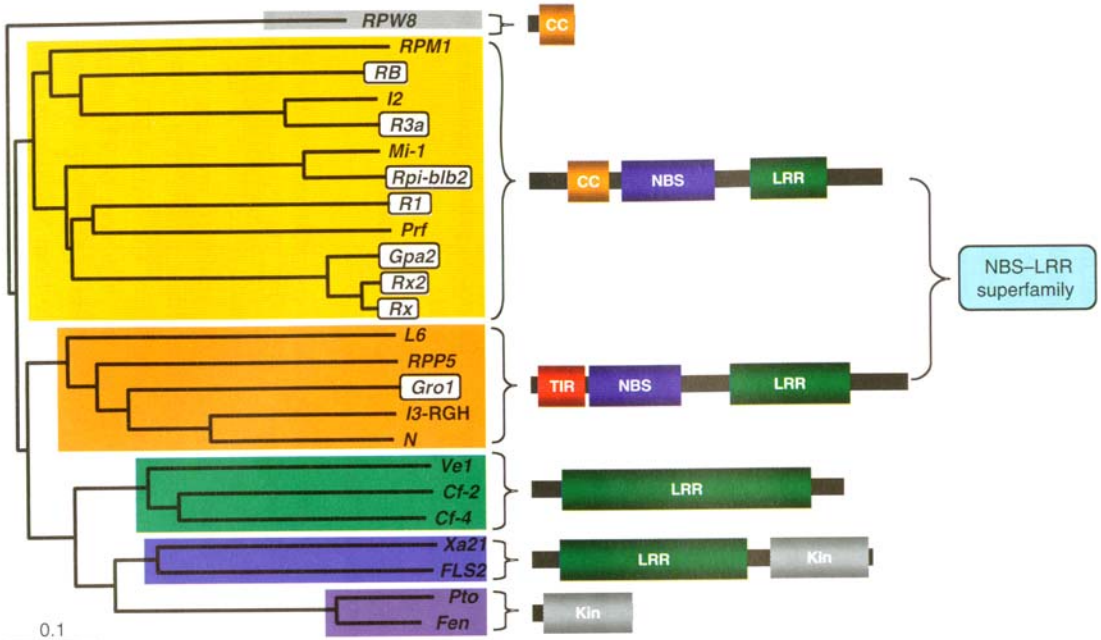

Fig. 7.2. Dendogram and representation of the structure of the five classes of plant disease resistance proteins. Amino acid alignment was carried out on the proteins that are (1) typical for the five main resistance classes (*Cf-2, Cf-4, Fen, FLS2, L6, N, Pto, RPP5, RPM1, RPW8, Ve1, Xa21*) (Hammond-Kosack and Jones, 1997; Dangl and Jones, 2001; Kawchuk et al., 2001) or (2) similar to the potato resistance proteins (*I2, I3*-linked resistance gene homolog, *Mi-1, Prf*) (Ballvora et al., 2002; Paal et al., 2004; Huang, 2005; van der Vossen et al., 2005) or (3) known potato resistance proteins (*Gpa2, Gro1, R1, R3a, RB, Rpi-blb2, Rx, Rx2*) (Bendahmane et al., 1999; Bendahmane et al., 2000; van der Vossen et al., 2000; Ballvora et al., 2002; Song et al., 2003; Paal et al., 2004; Huang et al., 2005; van der Vossen et al., 2003; van der Vossen et al., 2005). Conserved domains in the resistance proteins are CC, coiled-coil; Kin, kinase; LRR, leucine-rich repeats; NBS, nucleotide-binding site; and TIR, Toll and interleukin-1 receptor (Dangl and Jones, 2001). Resistance proteins isolated from potato are boxed.

7.3.3.2 *Nematode resistance genes* Gpa2 *and* Gro1

The isolation of the nematode resistance gene *Gpa2* (van der Vossen et al., 2000) on chromosome 12 was achieved when the genomic region containing the *Rx* resistance gene was further analyzed. Molecular analysis of this chromosomal region revealed that the *Gpa2* locus is one of four highly homologous genes in a region of approximately 115 kb. At least two of these genes are active. One homolog corresponds to the previously isolated *Rx* gene that confers resistance to PVX, whereas the other corresponds to the *Gpa2* gene that confers resistance to potato cyst nematode *G. pallida*. The deduced open reading frame of the *Gpa2* gene encodes a predicted polypeptide of 912 amino acids with a molecular weight of 104.5 kDa (van der Vossen et al., 2000). The *Gpa2* protein belongs to the CC–NBS–LRR class of plant resistance genes and shares over 88% amino acid identity with *Rx* and *Rx2*. Sequence conservation points to a direct evolutionary relationship of these proteins and to the fact that genes for resistance to distinct pathogens might be structurally similar. Isolation of *Gpa2* from the *Rx*-gene cluster demonstrates that within a single haplotype, members of the same R-gene cluster can evolve to confer resistance to distinct pathogen species (van der Vossen et al., 2000).

Gro1-4, a major, dominant locus conferring resistance to *G. rostochiensis* that is localized on potato chromosome 7 (Barone et al., 1990; Ballvora et al., 1995), has been recently cloned (Paal et al., 2004). *Gro1-4* is a member of the *Gro1* candidate gene family consisting of 15 closely related candidate resistance genes. The gene encodes a protein of 1136 amino acids that contains a Toll–interleukin 1 receptor (TIR) domain (Paal et al., 2004) and belongs to the TIR–NBS–LRR group of resistance genes (Dangl and Jones, 2001). *Gro1-4* is the first potato resistance gene of this type. At the nucleotide level, *Gro1-4* is more than 90% identical with two NBS–LRR-type R-gene homologs of tomato, which map to syntenic positions and are tightly linked to the tomato *I3* gene for resistance to *Fusarium oxysporum* (Pan et al., 2000a). Analysis of the susceptible cultivar Desirée complemented with *Gro1-4* showed that the gene is able to confer resistance to *G. rostochiensis* pathotype Ro1. Reverse transcriptase (RT)–PCR products of the gene family were generated from stem, leaf, flower, tuber, and stolon cDNA, demonstrating that the members of the *Gro1* gene family are expressed in all tested potato tissues. The possibility exists that other members of the *Gro1* gene family are also functional nematode R-genes conferring resistance to pathotypes other than Ro1 (Paal et al., 2004).

7.3.3.3 *Oomycete resistance genes* R1, R3a, RB/Rpi-blb1, *and* Rpi-blb2

Ballvora et al. (2002) used a combination of positional cloning and a candidate gene approach to clone *R1*, the race-specific gene for resistance to late blight. The *R1* gene encodes a protein of 1293 amino acids with a molecular mass of 149.4 kDa. Based on the deduced protein sequence, *R1* is a member of the CC–NBS–LRR class of plant resistance genes (Hammond-Kosack and Jones, 1997). The most closely related plant resistance gene (36% identity) is the *Prf* gene for resistance to *Pseudomonas syringae* of tomato (Ballvora et al., 2002). Two potato genes for resistance to PVX, *Rx2* and *Nb*, map to similar positions as *R1* (Ritter et al., 1991; De Jong et al., 1997). The *Rx2* gene has been cloned and is, like *R1*, a member of the CC–NBS–LRR class of resistance genes (Bendahmane et al., 2000). The two resistance genes share 32% sequence identity and

are therefore different members of the same superfamily of genes. Analysis of the *R1* genomic region indicates that further paralogous members may be located in the region. There is the possibility that the *R1* locus hosts more than one R-gene with the same race specificity, or, in addition to *R1*, other R-genes with so far unknown specificities. The interaction between *R1* and the late-blight pathogen *P. infestans* is in accordance with the gene-for-gene concept (Flor, 1971). Transfer of a single gene to susceptible cultivar Desirée was sufficient to elicit the hypersensitive resistance response upon infection with a *P. infestans* race incompatible with *R1* (Ballvora et al., 2002). The *R1* gene is located within a resistance hotspot on chromosome 5 that includes major QRL for resistance to *P. infestans* (Gebhardt and Valkonen, 2001; Simko, 2002; Jones and Simko, 2005).

Huang et al. (2005) used the comparative genomics approach to isolate the *R3a* resistance gene. Analysis of *R3a* revealed that the gene encodes a predicted polypeptide of 1282 amino acids with a relative molecular mass of 145.9 kDa. The *R3a* gene encodes a putative CC–NBS–LRR protein and shares 88% DNA identity and 83% amino acid similarity to *I2* of tomato that confers resistance to fungus *F. oxysporum*. The *R3a* and *I2* proteins are more related to each other than to other known R-proteins. Thus, the *I2* and *R3a* genes belong to the same R-gene family. The *R3a* protein bears only limited similarity (15 and 30% amino acid identity) to the other two late-blight R-proteins, *R1* and *RB/Rpi-blb1* respectively (Huang et al., 2005). The *R3a*-gene family contains at least four full-length and many truncated paralogues. Constitutive expression was observed for the *R3a* gene, as well as some of its paralogues whose functions remain unknown (Huang et al., 2005). Comparative analyses of the *R3* complex locus with the corresponding *I2* complex locus in tomato suggest that this is an ancient locus involved in plant innate immunity against oomycete and fungal pathogens. However, the *R3* complex locus has evolved after its divergence from tomato. This expansion has resulted in an increase in the number of R-genes and in functional diversification (Huang et al., 2005). The *R3* locus is composed of two genes with distinct specificity. The two genes – *R3a* and *R3b* – have been mapped 0.4 cM apart and both have been introgressed from *S. demissum* (Huang et al., 2004). At least 10 additional *I2* gene analogs have been found in the *R3b* region (Huang et al., 2005).

Potato germplasm derived from *S. bulbocastanum* has shown durable and effective resistance against *P. infestans* in field tests. Two groups independently isolated the resistance gene from *S. bulbocastanum*-derived material. The coding sequence of the *RB* gene (Song et al., 2003) is identical to that of the *Rpi-blb1* gene (van der Vossen et al., 2003), which suggests that the two isolated genes are identical. However, when flanking sequences and the intron of these two genes were compared, single nucleotide polymorphisms (SNPs) were found at a frequency ranging from 0.4 to 0.8%. Van der Vossen et al. (2003) therefore concluded that *RB* and *Rpi-blb1* are allelic, although functionally equivalent. Molecular analysis of the resistance locus identified a cluster of resistance genes belonging to the CC–NBS–LRR class of resistance genes. The *RB/Rpi-blb1* gene family includes one truncated and four complete genes within a 40-kb region. The four complete genes are similar in length (2895–2979 bp) and have conserved intron–exon structures (Song et al., 2003; van der Vossen et al., 2003). The *RB/Rpi-blb1* gene encodes a polypeptide of 970 amino acids and is more closely related to the *I2* protein of tomato (30% identity, 47% similarity over 1070 amino acids) than it is to any other

known R protein. *RB/Rpi-blb1* has limited similarity with the protein of *R1* (22% identity, 49% similarity over 902 amino acids), a gene derived from *S. demissum* that confers hypersensitive resistance to potato late blight (Song et al., 2003; van der Vossen et al., 2003). The *S. bulbocastanum* resistance gene is able to complement the susceptible phenotype in a *S. tuberosum* and tomato background, demonstrating the potential of interspecific transfer of broad-spectrum late-blight resistance to cultivated *Solanaceae* from sexually incompatible host species (van der Vossen et al., 2003). Similarly, as with the original *S. bulbocastanum* material, the transgenic plants with the *RB/Rpi-blb1* gene developed limited lesions on the lower leaves when inoculated with *P. infestans* (Song et al., 2003). The resistance gene transcript was detected in unchallenged plants, indicating the gene is constitutively expressed (Song et al., 2003).

Recently, Van der Vossen et al. (2005) cloned another resistance gene originating from *S. bulbocastanum* – *Rpi-blb2*. Similarly to *RB/Rpi-blb1*, the *Rpi-blb2* gene confers resistance to complex isolates of *P. infestans*. The *Rpi-blb2* locus is located on chromosome 6 and encodes a protein that shares 82% sequence identity with the *Mi-1* protein. However, whereas *Rpi-blb2* confers resistance to late blight in potato, the *Mi-1* gene confers resistance to root knot nematodes (*Meloidogyne* spp.), aphids (*Macrosiphum euphorbiae*), and whiteflies (*Bemisia tabaci*) in tomato. Molecular analysis of *S. bulbocastanum*-derived BAC clones spanning the *Rpi-blb2* locus has identified at least 14 additional *Mi-1* homologs (van der Vossen et al., 2005).

7.3.3.4 Resistance genes in potato

Eight resistance genes have been cloned so far from potato. All of these molecularly characterized genes have been found in clusters of various tightly linked R-genes and/or R-gene homologs of unknown function. Most of the homologs appear to encode proteins similar to that of the functional R-genes. Further complementation studies are required to elucidate the function of these homologs in the plant–pathogen interaction. An intriguing question concerning resistance genes is whether the rate of their evolution is directly related to gene specificity; hence, broad-spectrum resistance genes (e.g. *Rpi-blb1, Rpi-blb2*, and *Rpi-abpt*) evolve at a slower rate than race-specific genes (e.g. *R1–R11*). The isolation and molecular analysis of additional resistance genes may help with the identification of molecular motifs that determine resistance protein specificity and the rate of gene evolution. Given the fast progress in genetics and genomics, and the rapidly expanding knowledge about functionality of resistance genes, the number of mapped, isolated, and sequenced R-genes and QRL in potato will increase rapidly in the near future. Conserved domains from known resistance loci (Leister et al., 1996) and plant defense gene families (Trognitz et al., 2002) can facilitate more efficient detection and cloning of resistance genes through the 'candidate gene' approach.

7.3.4 Synteny of resistance loci in *Solanaceae*

In pepper, two major QRL for resistance to *Phytophthora capsici* (Lefebvre and Palloix, 1996) are located in the genomic regions corresponding to the potato late-blight R-gene clusters on chromosomes 4 and 11, respectively. Three R-genes (*Ph-1, Ph-2*, and *Ph-3*)

confer resistance to *P. infestans* in tomato. All three loci map to genomic positions corresponding to potato monogenic resistance. The *Ph-1* gene from tomato chromosome 7 (Peirce, 1971) appears to be positioned similarly to the *Rpi1* gene from *S. pinnatisectum* (Kuhl et al., 2001), whereas *Ph-3* (Chunwongse et al., 2002) is located on chromosome 9 in the same genomic region as *Rpi-moc1* from *S. mochiquense* (Smilde et al., 2005). *Ph-2* (Moreau et al., 1998b) from tomato and R_{ber}/R_{Pi-ber} from *S. berthaultii* (Ewing et al., 2000; Rauscher et al., 2006) are both located on chromosome 10. However, the *Ph-2* and R_{ber}/R_{Pi-ber} genes do not appear to be orthologous because they show different race specificity when tested with the US-7 race of *P. infestans* (Ewing et al., 2000). Nonetheless, the similarity in the location of genes for resistance to *Phytophthora* species in potato, tomato, and pepper suggests possible evolutionary conservation of resistance genes. Grube et al. (2000) hypothesized that the general function of resistance alleles (e.g. initiation of the resistance response) may be conserved at homologous loci in related plant genera, although the taxonomic specificity of host resistance genes may be evolving rapidly. In case of potato, tomato, and pepper, it is possible that the resistance gene specificity across genera remains relatively conserved and those homologous genes are still conferring resistance to *Phytophthora* species.

In addition to resistance against *Phytophthora*, the *Solanaceae* family shows a conserved position of genes conferring resistance to some other pathogens. Three potato genes encoding resistance to PVY (Ry_{adg} and Ry_{sto}) and PVA (Na_{adg}) reside in the resistance-gene hotspot on the long arm of chromosome 11 (Brigneti et al., 1997; Hämäläinen et al., 1998; Hämäläinen et al., 2000). The corresponding region in tobacco (*Nicotiana tabacum*) carries the *N* gene conferring resistance to tobacco mosaic virus (TMV) (Leister et al., 1996). The *Ve* locus from tomato confers resistance to *Verticillium* species. The locus is located on the short arm of chromosome 9 (Diwan et al., 1999). Positional cloning of the gene identified not one but two closely linked inverted genes (*Ve1* and *Ve2*) that independently confer resistance to the same pathogen. When the tomato *Ve1* and *Ve2* genes were expressed functionally in potato, resistance was observed in otherwise susceptible plants (Kawchuk et al., 2001). This observation indicates that all necessary components of the resistance response are present and functional in the related host genus. A probe derived from the tomato *Ve1* gene identified homologous sequences on potato chromosome 9 that are significantly associated with resistance to both *V. dahliae* (Simko et al., 2004a) and *V. albo-atrum* (Simko et al., 2004b). It appears that the *Verticillium* resistance genes in potato and tomato retain positional and functional synteny.

However, even within a single species, the positional and structural synteny of loci does not necessarily imply functional synteny. It is known that tightly linked and highly similar genes can confer resistance to different pathogens, for example *Rx* to virus and *Gpa2* to nematodes. Therefore, it is plausible to assume that structurally similar loci from corresponding chromosomal regions of different *Solanaceae* species might confer resistance to distinct pathogens, a fact that can be used for gene isolation. The *I2* locus encodes resistance to the fungi *F. oxysporum* in tomato (Ori et al., 1997; Simons et al., 1998). The resistance gene is located at the distal end of the short arm of chromosome 11, in the same genomic region as the *R3a* gene from potato conferring resistance to the oomycete *P. infestans* (Huang et al., 2004). Information from the *I2* locus was used to develop resistance gene analog (RGA)-specific primers to amplify all candidate RGAs at

the target region. This elegant approach eventually led to the isolation of the *R3a* gene with 88% DNA identity to *I2* (Huang et al., 2005). Similarly, the *Rpi-blb2* resistance gene from *S. bulbocastanum* (van der Vossen et al., 2005) is positioned in the same genomic region of chromosome 6 as the *Mi-1* gene from tomato (Milligan et al., 1998). The two genes share 82% protein identity, although they encode resistance to very different pathogens: *Rpi-blb2* to *P. infestans* (van der Vossen et al., 2005) whereas *Mi-1* to nematodes, aphids, and whiteflies (Milligan et al., 1998; Nombela et al., 2003).

These examples demonstrate how synteny within *Solanaceae* can be effectively used for comparative mapping and gene isolation. The completion of the tomato genome-sequencing project (http://www.sgn.cornell.edu/help/about/tomato_sequencing.html) will provide additional extremely valuable information for the efficient cloning of other potato resistance genes.

7.3.5 Marker-assisted resistance breeding

Selection of individuals with desirable traits from a breeding population can be based on phenotype, genotype (assessed with molecular markers), or a combination of the two. Phenotypic selection is more efficient for a trait with high heritability because it uses the sources of variation of all the loci, while markers can use only those loci to which they are linked (Charcosset and Gallais, 2003). MAS will be more effective than phenotypic selection when the proportion of additive variance accounted for by the marker loci is greater than the heritability of the trait (Dudley, 1993). Computer simulation shows that MAS can be more efficient than selection based only on a phenotype if the heritability of the trait is between 0.05 and 0.5 and the markers are close to the loci of interest (Moreau et al., 1998a).

If the evaluation of a target trait is time-consuming, difficult, or costly, MAS has an added advantage over phenotypic selection. For example, in breeding for resistance, MAS allows the breeder to conduct selection without depending on the natural occurrence of the pathogen or pest, or to perform selection during the off-season. Moreover, molecular markers in genomic regions of interest enable the selection of individuals with resistance to several pathogens simultaneously, unlike a phenotype-based selection that requires a number of individual trials. Obviously, when the percentage of genetic variation explained by the marker(s) is high, the efficacy of molecular marker(s) for MAS is also high. Hence, MAS is the simplest and most effective for R-gene-based resistance traits with markers tightly linked with, or residing within, the resistance gene itself. However, as the recombination frequency between the marker and the resistance locus increases, the value of the marker for MAS decreases.

In potato, molecular markers have been developed and successfully tested for a gene conferring extreme resistance to PVY. Kasai et al. (2000) developed a sequence-characterized amplified region (SCAR) marker to the PVY resistance gene Ry_{adg} (Hämäläinen et al., 1998). The marker was generated only in genotypes carrying Ry_{adg}, when tested on 103 breeding lines and cultivars with diverse genetic backgrounds (Kasai et al., 2000). Other known R-genes that are tagged with molecular markers can be conveniently used in MAS as well. For example, markers linked to the *Ns* gene conferring resistance to PVS are currently being used for indirect selection in diploid

breeding programs (Marczewski et al., 2002). With allele-specific primers, the presence of the R-gene can be followed even if other R-genes exist in the same plant material. Thus, resistance genes from diverse sources can be incorporated into a single genotype. Whether R-gene polyculture (multilane development) or pyramiding of several resistance genes or the opposite approach of eliminating the race-specific R-genes is an objective, MAS is a valuable tool to achieve the goal more efficiently. Recently, Gebhardt et al. (2006) elegantly demonstrated how MAS could be efficiently used in resistance breeding programs. The authors applied screening with PCR-based molecular markers to develop breeding material that carries combination of four resistance genes: Ry_{adg} for extreme resistance to PVY, *Gro1* for resistance to *G. rostochiensis, Rx1* for extreme resistance to PVX, or *Sen1* for resistance to potato wart. When tested in the presence of pathogen, all selected plants showed expected resistant phenotype. However, an important requirement for molecular markers used in MAS is their universality in a wide gene pool, not just in a specific cross. This problem was well documented when markers linked to the *Gro1* and *H1* resistance genes in the diploid population were tested on 136 unrelated tetraploid cultivars. The *Gro1*-specific marker was not correlated with the resistance phenotype, whereas the *H1*-specific marker was indicative of resistance in only four cultivars (Niewöhner et al., 1995).

Lately, markers tagging QRL conferring resistance to *P. infestans, V. dahliae*, and *V. albo-atrum* have been developed and effectively tested on sizable tetraploid populations. A highly significant association was detected between PCR markers specific for the *R1* gene (or markers flanking the *R1* locus) and the QRL for resistance to foliar and tuber blight. The marker–trait association was tested on 600 cultivars originating from different countries. The marker alleles associated with increased resistance were traced to an introgression from the wild species *S. demissum* (Gebhardt et al., 2004). Simko et al. (2004a) observed a highly significant association between a simple sequence repeat (SSR) marker and quantitative resistance to *V. dahliae* in a collection of 137 North American potato cultivars. When the pedigree of these cultivars was analyzed, the marker traced the origin of the resistance and susceptibility to two breeding lines widely used in the North American breeding program (Simko et al., 2004c). Similarly, a highly significant association was detected between the resistance to *V. albo-atrum* and an SNP marker developed from the *StVe1* locus. The marker–trait association was confirmed on 150 tetraploid cultivars and breeding lines as well as on a population developed from crosses between highly susceptible and resistant cultivars (Simko et al., 2004b). However, as each individual QRL explains only a relatively small portion of the variation in resistance, for a practical application, additional disease resistance loci need to be identified and tagged with molecular markers. After detecting and tagging enough resistance loci, the MAS will facilitate more efficient development of new potato cultivars carrying a desirable resistance gene combination.

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