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Status and way-forward in breeding potato (*Solanum tuberosum*) for resistance to late blight

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ABSTRACT

Improving host resistance to late blight (*Phytophthora infestans*) has been a major concern of potato breeders since the Irish famine of 1840s. Since then, a number of late blight resistant potato (*Solanum tuberosum* L.) cultivars have been developed using both conventional and molecular techniques. However, resistance to late blight did not last long and broke down due to the emergence of more virulent races of *Phytophthora infestans*. This perspective paper presents the status of sources of resistance to late blight, defence mechanism of host against the pathogen and late blight resistance genes mapped, cloned, and transferred to cultivated potato. Considerable progress has been made in all aspects of breeding potato for resistance to late blight. However, a critical appraisal of the status of potato breeding for resistance to late blight suggests the continued need to identify new sources of resistance, transfer of resistance genes from diverse sources and stacking them in maximum number into individual plants. Other breeding strategies to combat this dreaded disease are also discussed.

Keywords: Late blight, *Phytophthora infestans*, Potato Breeding, R/Rpi genes

Late blight of potato (*Solanum tuberosum* L.) is one of the oldest known plant diseases, which came into limelight in 1840s when the great Iris famine led to the death of over 1 million people due to complete destruction of potato crop. The oomycete *Phytophthora infestans* (Mont) de Bary, the causal organism of this dreaded disease is an important and interesting pathogen threatening food security (Yuen 2021). Potato varieties with durable resistance to late blight are essentially required for organic farming (Keizer *et al.* 2022). Both conventional and molecular breeding techniques are being used to develop late blight resistant varieties. This paper presents an overview of the status of potato breeding for resistance to foliage late blight and outlines the way-forward in managing this malady.

Search for late blight resistance

The Irish famine was the result of cultivation of potato varieties highly susceptible to late blight over a large area in Ireland and neighbouring European countries in 1840s. Actually, at that time no late blight resistant potato variety was available. The search thus started for late blight resistance in related *Solanum* species. There are more than 240 tubers bearing *Solanum* species which could be searched and used in breeding potato for late blight resistance. This resulted in the identification of high resistance to late blight in a hexaploid species *S. demissum*. It is the source of first 11 *R* genes governing resistance to late blight. New potato varieties were developed through interspecific hybridization of tetraploid cultivated potato with this species, but the resistance from this source broke down soon due to the emergence of new races of *P. infestans*. Until the later part of the 20th century, the genetic variation in *P. infestans* was limited to only one mating type A1, which reproduced only asexually (Fry 2008). However, in the 1950s, the A2 mating type was discovered in central Mexico, where the pathogen originated. In 1970s–1980s, the A2 mating type spread through the USA, Europe, Asia and North Africa (Fry *et al.* 1992). The presence of A1 and A2 mating types together allows sexual reproduction between them resulting in pathotypes adapted to certain fungicides and breaking host resistance (Smart and Fry 2001). Mating also results in the formation of more resistant oospores that contribute to early infections (Keizer *et al.* 2022). *P. infestans* has three-four fold bigger genome (240 Mbp) as compared to the known genome size of other *Phytophthora* species. The *P. infestans* genome also has many transposons and repetitive sequences. This leads to dynamic evolution of new strains in *P. infestans* by non-allelic homologous recombination and tandem gene duplications (Hajianfar *et al.* 2014).

The potato genetic resources have been extensively screened for late blight resistance and a number of species have been found resistant and are source of novel resistant

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(*R*) genes, later called as *Rpi* (resistant to *P. infestans*) genes. The Wageningen University hosts a database (SolRgenes) of resistance sources (Vleeshouwerrs *et al.* 2011). Rodewald and Trognitz (2013), Nachtigall *et al.* (2018), Sliwka *et al.* (2019) and Blossei *et al.* (2022) have extensively listed the sources of *R/Rpi* genes of late blight resistance. Maximum of 15 late blight resistant genes in a *Solanum* species have been reported in *S. demissum* followed by 5 each in *S. bulbocastanum* and *S. stoloniferum*. *Solanum* species each with 4 *R/Rpi* genes are *S. berthaultii, S. edinense, S. pimpinellifolium* and *S. venturi*. Species *S. hjertinglii,* has 3 and *S. dulcamara, S. mochiquense* and *S. pinnatisectum* have 2 *R/Rpi* genes each. *S. americanum, S. capsicibaccatum, S. chacoence, S. michoacanum, S. microdontum, S. papita, S. polytrichum, S. phureja, S. sparsipilum* and many more have been reported with single *Rpi* gene.

Late blight incidence is highly influenced by genotype × environment interaction (Nachtigall *et al.* 2018). Hence, screening must extend over years and locations to ascertain stable sources of resistance. Further, there are population to population, accession to accession and plant to plant variations in levels of resistance to late blight within a species. Hence, the evaluation for resistance needs to be carried out at individual plant level to identify the confirmed sources of resistance within a species. Ideally, individual clones should be screened for a least 3 years under epiphytotic field conditions and simultaneously should also screened in lab tests against individual and complex races (Gopal and Singh 2002). Clones found to be resistant may be evaluated again after a gap of 3–4 years to ascertain the durability of resistance.

Defence system in potato against late blight

Both race-specific (vertical, qualitative, major gene) and non-race specific (horizontal, quantitative, polygenic) resistance to late blight have been reported in potato (Nowicki *et al.* 2011, Juyo Rojas *et al.* 2019). However, vertical resistance breaks down easily due to the development of new races of *P. infestans*. Horizontal resistance though durable, is highly dependent on environmental conditions and is partial in nature.

Defence of potato plant gets activated upon its first contact with the pathogen (*P. infestans*). According to PAMP–triggered immunity mechanism, pathogen-associated molecular patterns (PAMPs) are perceived by the receptors on plasma membrane of the plant. This leads to intracellular signal transduction events culminating in defence responses. A known example of PAMP-triggered immunity (PTI) is the receptor-like protein ELR (elicitin response) that mediates the broad-spectrum recognition of PAMPs from several *Phytophthora* species (Du *et al.* 2015).

Another defence mechanism is elicitor-triggered immunity (ETI). According to this, the pathogen has elictor or effector molecules which help the pathogen to invade the host tissue. The plant defence system detects these effectors, and this interaction triggers immunity response. Detected effectors are called "*Avr*' genes. *P. infestans* is known to have hundreds of *Avr* genes many of which are redundant (Birch *et al.* 2008, Ghislain *et al.* 2019). Potato plant resists effector triggered infection through host *Rpi* genes. The PTI and ETI have many common signalling components. However, immune response in ETI is more robust and occurs more quickly than those in PTI (Tao *et al.* 2003, Tsuda and Katagiri 2010, Thomma *et al.* 2011).

More than 20 *Rpi* genes have been identified in *Solanum* species (Rodewald and Trognitz 2013, Jo *et al.* 2016, Ghislain *et al.* 2019) for which the cognate effectors have been reported. However, unrelated *Rpi* genes from different species may also recognize the same *Avr* gene out of 10 cognate *Avr* genes (Jo *et al.* 2016, Elanhal *et al.* 2020).

The *R* genes code for the nucleotide-binding domain and the leucine-rich repeat containing proteins (NB-LRR). Elicitor proteins are perceived by NB-LRR by diverse mechanistic and structural features thereby making the host immune to the infection by the pathogen (Cesari 2017). The durability of such resistance, however, depends on the host ability to recognize and suppress the effectors for pathogenicity (Rodewald and Trognitz 2013). This is expansion of the gene-for-gene model originally proposed by Flor (1971). As per this model, the incompatible interaction of the *R* gene in plant and the corresponding *Avr* gene of the pathogen results in resistance. On the other hand, absence of either the *R* gene or the *Avr* gene results in disease susceptibility (compatible interaction).

Pavan *et al.* (2011) reported resistance to late blight in potato due to loss of function of susceptible '*S'* genes. Normally *S* genes help the pathogen to infect, grow and colonize the host. However, if these genes are disabled, the plants become resistant as non-functional *S* genes do not assist the colonization of the pathogen on the host. These disabled *S* genes are recessively inherited and may provide race-nonspecific resistance. Sun *et al.* (2016) reported that by silencing 5 *S* genes using RNAi, high level of resistance to late blight was obtained in transgenics of otherwise highly susceptible potato variety Desiree.

Potato plant also defends *P. infestans* through genes that code for defence related hormones such as ethylene, jasmonic acid, salicylic acid and anionic peroxidases or in over-expression of pathogenesis-related proteins (Jo *et al.* 2016, Yang *et al.* 2020). A number of anti-fungal compounds including phytoalexine, phenols, reactive oxygen species, protease inhibitors, oxidases *etc.* are produced by the potato plant as defence against *P. infestans* infection and growth. Their role and involvement in signalling pathways of defence have been reviewed by Hajianfar *et al.* (2014). The resistance through these biochemical pathways is many a times non race-specific.

The defence mechanisms reported from time to time by different workers may actually be operating simultaneously through cascades of interacting factors. This explains the complexity of managing and breeding for durable resistance to late blight as defence for resistance may break down at different levels.

Mapping R genes for late blight resistance

Several genes imparting late blight resistance derived from different *Solanum* species have been mapped (Geberhardt and Valkonen 2001, Nachtigall *et al.* 2018, Paluchowska *et al.* 2022). *R* genes from *S. demissum* were the first to be mapped. Out of 11 known *R* genes from *S. demissum*, 5 have been mapped and cloned (Nachtigall *et al.* 2018). According to Paluchowska *et al.* (2022) 70 *Rpi* genes have been identified and mapped in 32 *Solanum* species. Hajianfar *et al.* (2014) and Nachtigall *et al.* (2018) reported that 27 *Rpi* genes have been cloned. Further, they reported that there are a large numbers of homologs/orthologs of *R* genes in potato having high sequence similarity (Hajianfar *et al.* 2014). Many workers (Rodewald and Trognitz 2013, Tiwari *et al.* 2013, Nachtigate *et al.* 2018, Elanhal *et al.* 2020) have listed the *R/Rpi* genes cloned and their chromosomal locations. Earlier maps were primarily based on Restriction Fragment Length Polymorphism (RFLP) and Amplified Fragment Length Polymorphism (AFLP) markers (van Os *et al.* 2006). Later, Diversity Array Technology (DArT) marker mapping was used which yielded a genome-wide linkage map in resistant wild species *S. bulbocastanum*. Syverson and Braden (2011) used Single Nucleotide Polymorphism (SNP) against *P. infestans*. Recently, Nachtigale *et al.* (2018) used DArT/SSR (Simple Sequence Repeat) markers and assigned late blight resistance locus from *S. pinnatisectum* to chromosome VII.

The above referred reviews show that most of late blight resistance *Rpi* genes are located on chromosome IV and XI, forming two major clusters. However, use of different markers systems and populations led to mapping of same genes on different locations on same or different chromosomes in some studies. For example, effector-based studies have shown that *Rpi-sto1* and *Rpi-pta1* are indeed functionally equivalent to *Rpi-blb1* (Vleeshouwers *et al.* 2008). Similarly, *R2, R2-like* and *Rpi-abpt* were found to be functionally equivalent to *Rpi-blb3* (Hein *et al.* 2009, Lokossou *et al.* 2009). The sequence of *Rpi-phu1* was shown to be identical to *Rpi-vnt1.1* (Foster *et al.* 2009, Pel *et al.* 2009). These reports show that the number of distinct functional *Rpi* genes may be somewhat smaller than actually reported. So, further refinement and confirmations are needed in mapping of *Rpi* genes. The mapping populations should not only be diverse but also large enough and screened over years to assure reliable and repeatable results.

Mapping QTLs for late blight resistance

Mapping of Quantitative Trait Loci (QTLs) for late blight resistance is important to breed for durable horizontal resistance also referred as 'field resistance'. This resistance, however, is of partial nature as infection does not stop, but spread is slow so that crop does not have high intensity of the disease. Several association studies have been done in potatoes to map QTLs for disease resistance (Gebhardt *et al.* 2004, Simko 2004, Pajerowska-Mukhtar *et al.* 2009, Alvarez *et al.* 2017, Juyo Rojas *et al.* 2019). Tiwari *et al.* (2013) and Hajianfar *et al.* (2014) reviewed the QTLs for late blight found in various *Solanum* species. Hajianfar *et al.* (2014) concluded that these are distributed throughout the potato genome/chromosomes. But many of these QTLs were reported to have pleiotropic effect for late blight resistance and foliage maturity. Further, these studies have been influenced by biology, genetics and diversity of pathogen as well as the environmental conditions (Corwin and Kliebenstein 2017, Juyo Rojas *et al.* 2019). Juyo Rojas *et al.* (2019) used multi environmental data of two locations for organ-specific phenotypic response to late blight of 150 accessions of *S. tuberosum* Gp. Phureja. These data were merged with a genotyping matrix and using Genome Wide Association (GWA) they mapped 16 organ-specific QTLs for resistance to late blight. Lindqvist-Kreuze *et al.* (2020) identified stable late blight resistant tetraploid genotypes from 380 CIP clones bred for resistance to late blight and viruses, and evaluated in Peru, China and Ethiopia. Using GWA with SNP markers, they mapped the stable QTL for late blight resistance on chromosome 9 and environment specific late blight QTLs on chromosomes 3, 5, 6 and 10. Meade *et al.* (2020) mapped QTLs in F_1 populations from crosses between blight susceptible parents and lines with strong partial resistance to late blight derived from *S. microdontum* and *S. pampasense*. Using SNP markers, they found the QTLs for late blight resistance that were consistently expressed over multiple years in both populations. In *S. microdontum* derived population QTLs were on chromosomes 5 and 6, and in the *S. pampasense* source, QTLs for resistance were found on chromosomes 11 and 12. Further studies are needed to identify QTLs associated with known *Rpi* genes. Early maturing clones/varieties are in general susceptible to late blight. Combining late blight resistance and early foliage maturity is a challenge for the breeders. Mapping studies thus should aim to identify QTLs useful in combining early maturity and late blight resistance.

Inheritance of resistance to late blight

Several workers have studied the inheritance pattern of field resistance to late blight in potato. Killick and Malcomson (1973) and Kaushik *et al.* (2000) found that mainly non-additive gene action is important in the inheritance of horizontal quantitative resistance to late blight in potato. On the other hand, Malcomson and Killick (1980) and Tung *et al.* (2018) reported only additive gene action for this trait. Kumar *et al.* (2007) and Namugga *et al.* (2020) found both additive and non-additive gene actions to be equally important. The variations in the results of various studies in the control of this polygenic trait may be due to several factors including the types of material and mating design used, and the environmental conditions. Further, results may also vary according to methods and scales used for recording disease intensity (Gopal and Singh 2003/2004) and generation to generation variations observed in the combining ability of such characters (Gopal 1998, Kumar and Gopal 2006) in potato. Vertical qualitative resistance controlled by major genes (*R/Rpi*) is inherited in simple Mendelian pattern, but yet the presence of genotype

× environment interaction for this trait is not ruled out (Nachtigall *et al.* 2018). Studies are needed to identify good general combiners for resistance to late blight particularly in early maturing germplasm (Kumar *et al.* 2005).

Introgression of late blight resistance

The use of conventional methods to breed for late blight resistance (Gebhardt and Valkonen 2001, Juyo Rojas 2019) was successful in breeding only partially resistant varieties (Juyo Rojas *et al.* 2019). Genes *R1, R2, R3, R4* and *R10* from *S. demissum* have been widely used in potato breeding (Vleeshouwers *et al.* 2008). Commercial varieties with one to four *R* genes have been produced. It is estimated that more than 50% of the common potato cultivars have genes from *S. demissum* (Bradeen and Kole 2011). But this resistance did not last long due to emergence of A2 mating type and sexual recombination between A1 and A2 mating types leading to more aggressive virulent strains of *P. infestans* (Fry 2008*)*. For example, Kufri Jyoti bred in 1968 in India and carrying *R3, R4* and *R7* genes from *S. demissum* had become susceptible to late blight in 1980s. Pentland Dell released in 1963 in Great Britain and having genes *R1, R2* and *R3a* became susceptible to late blight within 4 years (Van Weymers *et al.* 2016). Similarly, previously resistant potato cultivars Lady Belfour and Stirling became susceptible to late blight after the emergence of 13-A2 or Blue 13 race of *P. infestans* in Europe (Van Weymers *et al.* 2016). Thus breeders have been looking for durable resistance to late blight which can last for long periods even under favourable conditions of disease development, and which does not break easily even when new races of *P. infestans* appear and infect the potato crop. To achieve this, late blight resistant genes from diverse germplasm sources are being transferred to commercially acceptable cultivars.

Stacking (pyramiding) of resistant genes has been advocated by many workers to achieve durable resistance. There are many reports on pyramiding of late blight resistant genes in potato using conventional as well as recent molecular techniques (Rakosy-Tican *et al.* 2020). However, Bradshaw (2009) had estimated that less than 7 out of 220 tuber bearing *Solanum* species have been used in potato breeding because of sexual incompatibility of wild species with cultivated *S. tuberosum.* Many approaches like bridge species, double pollination, embryo rescue, protoplast fusion etc. have been used to overcome these barriers.

S. bulbocastanum proved to be a good source of broad-spectrum *Rpi* genes. The *RBRpi-blb1* gene from this diploid *Solanum* species provided resistance to all known races of late blight when transferred to susceptible cultivar Katahdin (Song *et al.* 2003). Transfer of such resistance through conventional crosses and also via somatic hybridization had been achieved with hard labour spending long time due to genetic drag and elimination of undesirable genes of the wild species through back crossing. For example, it took 45 years to develop two late blight resistant varieties Bioncia and Toluca with resistance genes derived originally from *S. bulbocastanum* (Haverkort *et al.* 2009). Marker-assisted breeding (Barone 2004, Tiwari *et al.* 2013) and genomic selection (Meuwissen *et al.* 2001) help in accelerating the process of selection for resistance to late blight. Mainly phenotype-single marker associations have been used for selecting late blight resistant genotypes in breeding programmes (Gebhardt *et al.* 2004, Malosetti *et al.* 2007, Muktar *et al.* 2015, Mosquera *et al.* 2016, Braun *et al.* 2017). Tiwari *et al.* (2013) reviewed the markers reported to be associated with *R/Rpi/QTLs* for resistance to late blight. Genomic selection for late blight has been attempted by Enciso-Rodriguez *et al.* (2008). However, the genomic heritability estimates reported by them were of moderate magnitude (0.46) only for late blight.

Transgenic approach also is being adopted as a short cut to conventional breeding. Transgenic potatoes with multiple *R* genes for late blight have been produced by stable transformation incorporating high resistance to late blight without affecting the yield and other agronomic characters. For example, Ghislain *et al.* (2019) reported the transfer of 3 *Rpi* genes from wild relatives (*RB, Rpi-blb2* from *S. bulbocastanum* and *Rpi-vnt1.1* from *S. venturii*) into LB susceptible potato varieties Desiree and Victoria. The 13 transgenic events so produced had complete resistance to late blight in the field over several seasons in Sub-Saharan Africa where only A1 mating type is present. In transgenics, the *RB* mediated resistance is known to be positively correlated with the number of *RB* transcripts (Bradeln *et al.* 2009, Kramer *et al.* 2009). Expression of resistance owning to the same gene also varies with the host's genetic background (Sundresha *et al.* 2018). Further, due to environmental and health risks, transgenic cultivation is not allowed in many countries and there are complicated legislations in this regard. One major reason is the origin of genes transferred to transgenic. Transfer of genes from unrelated species, animals, bacteria, viruses etc. is viewed with scepticism. However, transgenic with genes from naturally crossable species (cisgenes) has better acceptance provided such transgenic is free of transformation process selectable markers from bacteria etc. This intragenic approach of producing cis-transgenic is now being advocated (Jo *et al.* 2016) where in coding sequences as well as promoters are also of potato origin. Approaches to boost the potato plant's immune system against late blight using intragenesis have been reviewed by Jo *et al.* (2016). Target for such modifications can be genes involved in recognition of pathogen on initial attack and/or downstream signal transductions genes involved in defence mechanism at different levels after attack. These modifications many a times can provide non-race specific resistance.

Gene editing using CRISPR-cas9 would allow production of late blight resistant genotypes by replacing the detrimental susceptible sequence with the resistance governing sequence (Bethke *et al.* 2019). Such methods can reduce the time required to produce the resistant cultivars, but phenotyping assays will still be required to confirm that introduced or edited genes provide the required level of broad-spectrum resistance to late blight. Recently Kieu

et al. (2021) demonstrated that mutated S genes (StDMR 6-1 and StCHL 1) generated by CRISPR/Cas9 increased late blight resistance in potato.

Several cultivars with high level of resistance to late blight have been registered (Śliwka *et al.* 2019). However, the resistance of many newly developed potato varieties with *Rpi* genes introgressed by different approaches has been lost or being lost. This further stress the need of combining in one plant many resistant genes which could recognise not only several existing races and but also the new ones which are likely to emerge due to high genome plasticity of *P. infestans*.

Way forward

- Long existence of host (mainly wild species) with *P. infestans* leads to co-evolution (Bethke *et al.* 2019). This unending process of co-evolution ensures the availability new resistance genes corresponding to the avirulence genes. So, the search for and introgression of new resistant genes should be a continuous process in breeding potato for resistance to late blight.
- There is need for pre-emptive or anticipatory breeding where in hybrids with more and more new resistant genes from diverse sources are produced and kept ready in advance to counter the likely emergence of new virulent races of *P. infestans* genome. A recent report (Rogozina *et al.* 2021) claims that N.I. Vavilov Institute of Plant Genetic Resources (VIR), Russia has a collection of potato hybrids with late blight resistance derived from 22–26 *Solanum* species with 8–9 species in the pedigree of individual hybrids. These hybrids were confirmed to have 4–6 *Rpi* genes per plant and had long lasting resistance to late blight which may also be due to some still uncharacterized resistant genes (Rogozina *et al.* 2021).
- High plasticity of *P. infestans* genome leads us to think of identifying late blight resistance genes which too should be dynamic enough to evolve to counter the attack of virulence genes of emerging strains of *P. infestans*. One may call this a Utopian Idea. But this phenomenon does exist in nature otherwise wild species of potato known to be resistant to late blight had not remained resistant to it for long periods. The evolution of host *Rpi* genes, however, is much slower than the *P. infestans Avr* genes. So highly dynamic *Rpi* genes governing resistance needs to be identified in wild potato species and transferred to cultivated potato so that cultivars may co-evolve directly when pathogen mutates and the cultivar remains resistant. The existing *Rpi* genes may be made transposon rich so that these mutate at their own to resist to new *Avr* effectors of *P. infestans*.
- As discussed above, resistance mechanism in potato against late blight involves both no-recognition and *R* gene- recognition approaches. So, combined application of all approaches is required for incremental additions leading to durable and long-lasting resistance.
- Another approach can be to grow a number of cultivars with mosaic of *Rpi* genes in different cultivars (mixed cropping) to slow/resist the spread of late blight in a potato stand by the variable response of different plants to various virulent races of *P. infestans* attacking the crop. The cultivars selected for this purpose, however, should have similar agronomic characters mainly tuber colour, size and shape etc. or cultivars with contrasting tuber characters should be grown together so that these are separated out easily after harvest. The crop from True Potato Seed may meet this requirement. Hybrid TPS lines should be possible at the diploid level as homozygous diploid potato inbred lines are now available (Lindhout *et al.* 2011, Blossei *et al.* 2022). The *Rpi* genes from diploid *Solanum* species can be easily transferred to these lines. After marker-assisted two backcrossing followed by a selfing one can get lines with *Rpi* genes, but similar to the original homozygous diploid parent (Su *et al.* 2020). Such lines with different *Rpi* genes can be inter-crossed to produce lines with more than one *Rpi* gene. The hybrids TPS populations from such material can be commercialized, provided these are otherwise acceptable from agronomic point of view.
- Another approach can be growing of late blight resistant cultivars with different *Rpi* genes in different seasons, so that *P. infestans* strains prevalent in one season are not able to perpetuate in the next season and eventually perish.

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