Potato J. 38 (1): 1-8, 2011

EVOLUTION OF VERTICAL AND HORIZONTAL RESISTANCE AND ITS APPLICATION IN BREEDING RESISTANCE TO POTATO LATE BLIGHT

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ABSTRACT: The concepts of vertical and horizontal resistance in plants are based on the number of "major" genes involved in the defence mechanisms of the host. Vertical resistance is monogenic or only few genes activate the defence reactions, while horizontal resistance is polygenic. In potatoes these approaches were identified in early 1900's in *Solanum demissum*. However, in the 1950's resistant American and European clones were infected by local races of *Phytophthora infestans* (Mont) de Bary in the highlands of central México, alerting breeders and pathologists to "new" races of the pathogen previously unknown to science. In addition, characterization of such races with recent molecular tools creates a new paradigm for variation in the pathogen independent of the old gene-for-gene concept of host/pathogen resistance/virulence. Sexually derived progenies, great number of propagules, short life cycles, and high frequency of genetic changes, provide *P. infestans* with enough pathogenic plasticity to easily breakdown genetic resistance of the host. This leads to a continuous struggle to generate more and better potato clones resistant to the pathogen including the transgenic approach.

INTRODUCTION

Vertical resistance is when a plant variety exhibits a high degree of resistance to a single race or strain of a pathogen. This ability usually is controlled by one or a few number of plant genes. Horizontal resistance, on the other hand, protects the plant against several strains of a pathogen, although the protection is not complete. It involves more resistance genes that the vertical resistance (12). Vertical resistance is also known as specific, qualitative, monogenic, and non-durable. The plant with this type of resistance hardly exhibits symptoms. It results in the death of the infected cells, restricting the establishment of the pathogen (55). The R genes (R stands for resistance) of the plant correspond to the avirulent genes of the pathogen. This is known as the "gene-for-gene" interaction (13). These R genes in a given potato variety with vertical resistance prevent the infection and development of the late blight disease if the race of the pathogen contains the avirulent genes correspondent to the ones of the variety. Horizontal resistance, in contrast, is equivalent to non-specific, quantitative, multigenic, durable, and field resistance, and some times infection and disease onset is possible, but this type of resistance protects from many virulent races of the pathogen. These concepts were not known after the Irish famine in the 1840's. In those times biologists were busy searching for the causes of the problem (5). The Irish famine was followed by very active and aggressive empiric breeding programme in the northern hemisphere. It is estimated that in the US about 380 varieties were created or introduced between 1851 and 1910, while in Ireland 255 new potato genotypes were registered as up to 1885. Nevertheless, none of such genetic materials carried resistance genes from wild *Solanum* species. The cultivated potato, *Solanum tuberosum,* was the only species available for crossings, and none of the new varieties

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was better than cv. Champion, traditionally resistant in England (11, 2, 9, 7, 48).

In the early 1900's breeders discovered that wild *Solanum* species from the highlands of central Mexico were resistant with vertical, major R genes. Distinguished breeders such as K.O. Muller from Germany, Donald Reddick from the USA, William Black from Scotland, and Cees Mastenbroek from the Netherlands, incorporated those R genes into cultivated potatoes. The initial positive results led to the creation of many breeding programs between 1925-1935, to the point to propose the eradication of the disease (47, 41, 43, 39, 42, 53). It worked well against local races of *P. infestans* in Europe and the United States, but in the late 1940's, John S. Niederhauser and collaborators, working for the Rockefeller Foundation in the highlands of Mexico, realized that such resistance did not work in the Toluca Valley of México. Resistant varieties or clones from other countries were easily infected by *P. infestans* in Toluca, and they concluded that more virulent races were present in the Valley additional to the common ones already known in other places (35, 37). The resistant, successful potato clones and varieties from several countries were overwhelmed by the *P. infestans* plasticity or capacity to diversify into a wide range of different pathotypes, regardless the R genes constitution of the potato genotypes. Breeders and pathologists learned that the local population of the pathogen was more complex and with higher pathogenicity in Toluca than the races known in other countries at that time. To overcome the challenge, Niederhauser and his colleagues combined a broader resistance obtained from local wild *Solanum* species, *Solanum demissum* in particular, with the already present R genes, resulting in a new series of clones with more durable, horizontal resistance (37, 14, 38). Simultaneously, in the early 1950's Dr. Black proposed a classification of pathogenic races based on host-specific or indicator plants (6). The new era of horizontal resistance of potatoes and the study of the complexity of *Phytophthora infestans* was born. All the races of the pathogen are present in every growing cycle in Toluca (32, **Fig. 1**). Nevertheless, we usually include a set of the indicator plants in our trials in the Valley, to monitor the sequence of incidence (**Fig. 2**).

Actually, many wild *Solanum* species have been identified possessing R genes, like *S. demissum, S. hougassi, S. bulbocastanum, S. edinense, S. cardiophyllum, S. oxycarpum, S. stoloniferum,* and many other (14, 38, 8, 45, 28). However, the introduction of their resistance genes into cultivated potatoes is not enough to keep durable resistance. The capacity of diversification of *P. infestans* can overcome such resistance leading to the collapse of R-gene based potato germplasm, making breeding programs a continuous and intense activity.

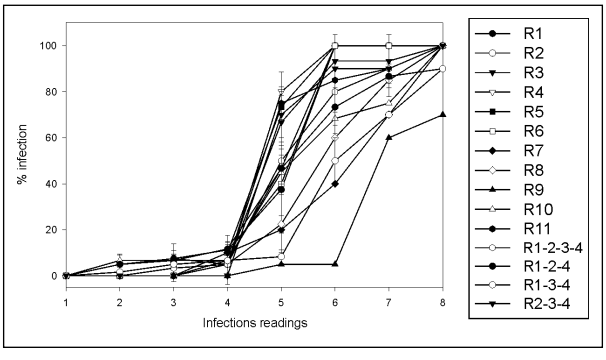

Fig. 1. Common disease progress curve in indicator plants for Phytophthora infestans pathogenic races in the Toluca Valley, México (32).

Breaking down resistance

According to McDonald and Linde (34), there are four aspects that the pathogen should have in order to breakdown host's resistance, and *P. infestans* has all four such requisites. All four are simultaneously present in the

oomycete in many potato growing areas of the world.

Mixed reproduction system: *P. infestans* has both, asexual (sporangia and zoospores) and sexual reproduction (oospores) (18), and sexually derived progenies have more variability than the asexual ones regarding race identification (52). The presence of the two mating types of the oomycete leads to genetic changes in its populations (22).

Fig. 2. Indicator plants for races of Phytophthora infestans in the Toluca Valley, México (Back, Drs. Kenneth Deahl, USA, and Louise R. Cooke, Northern Ireland).

Fig. 3. Transgenic potato plants with RB genes from S. bulbocastanum (Dr. John P. Helgeson inspecting). Susceptible clones (empty labels) at back. Toluca Valley, México.

High potential for genotype flow: Regardless the controversy on the proposed origin and direction of the genetic flow, either from central México (17) or South America (19), the spread of the A2 mating type to the rest of the world in the 1970's and 1980's is an excellent example of the great potential of *P. infestans* for genotype flow worldwide. In fact, it derived into a continuous increase in the variation of the pathogen (22, 20, 16, 21,).

Large effective population sizes: Under optimal conditions, *P. infestans* releases thousands of sporangia per week. In the Toluca Valley, the range goes from approximately 6,000 sporangia/cm^2 in resistant cv. Norteña, to 168,000 sporangia/cm^2 in susceptible cv. Alpha in 84 h incubation period (31).

High mutation rates: This last aspect is difficult to quantify, but it may not be necessary considering the great chances of recombinations among the numerous races of the oomycete in nature. Nevertheless, mutations are reported either for fungicide resistance (44) or for evolution studies (26, 3).

Phytophthora infestans: races vs. genotypes

The race identity is based in the specificity of the pathotype of *P. infestans* infecting the set of indicator plants (6). In recent years, in addition to the race concept, the idea of pathogen genotypes is of common use, thanks to new molecular tools to trace specific genes and their products of the oomycete. This information has devolved into other classifications of *P. infestans*, like the US series (15). On this respect, the basic data required for identifying a genotype are:

Mating type (A1 and A2): The unknown isolate is challenged to both mating types in separate dishes. When mycelia of both isolates

meet (unknown and known strains), oospore formation indicates that the unknown belongs to the mating type opposite of the one to which it was challenged. When oospores are formed when the unknown is paired with both mating types, the unknown is homothallic (18, 20).

Dilocus allozyme patterns for peptidase (PEP) and glucose-phosphate-isomerase (GPI): Allozymes are variants of the same protein but which may differ in their amino acid constitution. When separated by electrophoresis on the basis of mainly electric charges, the resulting band pattern identifies a particular genotype (23).

Restriction fragment length polymorphism (RFLP): When an endonuclease cuts a nucleic acid in specific, selective restriction sites, and a fragment of DNA is dispersed throughout the genome, probing a southern blot of the digested DNA with the labeled fragment produces a specific pattern. In the early 1990's Goodwin and co-workers standardized the method with the RG57 probe for genetic fingerprinting of *P. infestans* for population and genetic analysis (15, 24). Now a days, RFLP has the disadvantage of requiring standardization with specific probes for comparative studies from different research groups.

Mitochondrial DNA haplotypes: Haplotype stands for haploid genotype. It is a combination of alleles at multiple loci in the same mitochondrial DNA. Multiple studies on of *P. infestans* haplotypes not only provide a more sound characterization of the oomycete, but also suggest evidence of migration and genetic links of the pathogen from different geographic areas (19, 3, 15).

Metalaxyl resistance: This and other phenylamide systemic fungicides were introduced in Europe and north America in 1979. The following year resistant strains of *P. infestans* were reported in both continents and few years later phenylamide chemicals were banned or restricted to use with other contact fungicides (54, 10). Even though the genetics of the resistance is not clear (heterozygous, single genes, minor genes, incomplete dominance) (29), insensitivity/ sensitivity is still a valid character included in the description of *P. infestans* genotypes.

According to our data, races and genotypes of the pathogen are independent concepts. In the Toluca Valley, México, usually the early arrival of the pathogen corresponds to races (R1,2,3,4; R1,2,3; R2,3,4) followed by the sequence of the serial number (R1 to R11), except for R9, R5, and R2 (**Fig. 1**). These races are the last ones to show up (32, 4). The American US-1 genotype is reported as A1, 86/100, 92/100 (mating type, *GPI, PEP*) (15), but in our studies it corresponded to R3 in 2006 and to R10 and R1,2,3 in 2007. Also, the most frequent genotype in those two years was A2, 100/100, 100/100, but it corresponded to R5 and R8 in 2006, and to R8 only in 2007 (32, 4). Other clear example of the independence of the two concepts is that in Toluca all the races show up along the growing season every year, which in theory should correspond to the 56 genotypes (25).

Metabolic pathways for vertical and horizontal resistance

Vertical resistance follows the shikimic acid-phenylpropanoid pathway, as precursor for salicylic acid. It makes sense if we consider that this pathway induces hypersensitive responses and/or systemic acquired resistance, which is typical of vertical, monogenic resistance (1). Callose deposition and extracellular globules with phenolic compounds to strengthening cell walls are other consequences of this type of mechanism (55). Also, serine and aspartic-rich proteases in the plant are reported to elicit hypersensitive response and cellular necrosis (40). Horizontal defence, on the

other hand, includes the mevalonic acid pathway, that leads to the activation of both, defence genes (phenylalanine ammonia lyase, PAL, lipoxigenases) and protectant genes (glutathione-S-transferase, GST, peroxidases). It also stimulates more phytoalexins, chitinase and glucanase synthesis, as well as jasmonic acids and terpenes (1, 51, 33; Roy Navarre, USDA, Prosser, WA, USA, personal communication). No wonder why horizontal resistance is also called muti or polygenic resistance.

More molecularization

The discovery, isolation, and utilization of restriction enzymes (endonucleases), as well as the proposal of DNA structure in the early 1950's, launched a revolution in the molecularization of research focused on nucleic acid-based methods. The potato-late blight interaction was no exception. Turner (53) identifies three factors that drive the late blight science to molecular methods: (a) massive population displacements and spontaneous genetic changes of the oomycete, (b) adoption of molecular methods as tools, and (c) integration of a broader research body and transition to genomics (54).

Molecular techniques have enabled the cloning, sequencing and generation of new transformants of commercial potato varieties. One example of this gene manipulation is the transfer of resistance genes from *S. bulbocastanum* to *S. tuberosum*. The former is a wild species common in the highlands of Mexico, but its diploid condition does not allow traditional crosses with the tetraploid *S. tuberosum*. This challenge was overcome by J.P. Helgeson and his team by using protoplast fusion to obtain somatic hybrids that were highly resistant. These were used in a series of back crosses to *S. tuberosum* to obtain several highly resistant clones (27). Resistance of the clones was validated successfully in the Toluca Valley for several years. We inferred that such resistance was of the horizontal type because of 21 different *P.infestans* strains gradually infected the plants along the growing season (30). It was later reported that the resistance gene for late blight was mapped to chromosome 8 in *S. bulbocastanum*. This finding was possible by using RAPD and RFLP markers (36). As a follow up to this research, the same group cloned the resistance RB gene (RB stands for resistance-*bulbocastanum*). The resulting transgenic plants were highly resistant to the disease in Toluca (**Fig. 3**) (49). More recently several resistance gene clusters were located in chromosome 4 in this species (50). By using an AFLP marker this team found that the cluster cosegregated in progeny plants of an intraspecific mapping population, and working with other markers they learned that four genes are located in the same R-gene cluster on chromosome 4, "and likely belong to the same gene family". Resistance was also located in chromosome 5 for the genus *Solanum* in general, and "a previously identified cluster of three race-specific R genes on chromosome 11 was not associated with polygenic resistance" (46). This researcher found evidence of vertical resistance or race-specific QTL (Quantitative Trait Loci).

CONCLUSIONS

For the last 80-90 years, the evolution on the concept and the knowledge of vertical-horizontal resistance as applicable to the potato-late blight interaction, has gone through three periods:

a) From 1920's to 1940's. This period was characterized by potato breeding based on vertical, highly resistant genes, mainly from *Solanum demissum,* against specific races of *P. infestans*. There were only few pathotypes dispersed in the world outside México.

b) From 1950's to date. Breeding programs are using a combination of vertical and horizontal resistance from several wild *Solanums*, creating varieties with moderate but durable resistance. Indicator plants are of common use for identification of pathogenic races.

c) From the early 1980's to date, the molecularization era. Although knowledge of defence mechanisms has not contributed to development of resistant cultivars, the study of secondary metabolites explain, at least, the two defence mechanisms (vertical, horizontal). Nucleic acid-based studies, on the other hand, are focused in better characterization of the host for location and manipulation of resistance in specific chromosomes and genes of wild *Solanum* species, resulting in transgenic plants currently and successfully proved resistant to late blight.

On the basis of the vegetative propagation of potatoes, we should expect durable and uniform resistance of new potato varieties. However, considering the high genetic plasticity of *P. infestans*, chances are that because of the sexual reproduction of the pathogen new emerging pathotypes of the oomycete overcome host resistance, and the getting better host genotypes resistant to the new *P. infestans* is a permanent activity for breeders, pathologists, and molecular biologists.

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MS Received: 26 March 2009; Accepted: 8 November 2010