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The Never-Ending Story: Breeding Potato for Late Blight Resistance

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Editorial

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Among plant pathogens, *Phytophthora infestans* (Mont.) de Bary 1876 is the pathogen with the most important impact on human history. It was the responsible agent of the famous Irish famine in the 1840s. With direct results death of 1 million people and migration of 1.5 million others^[1]. Since then, it still causes problems to farmers, plant breeders, and to plant pathologists.

P. infestans belongs to *Oomycetes*. It adopts a hemibiotrophic lifestyle. In the first infection stage, it requires living cells (biotrophic); but in the second, it switches to saprophytic/necrotrophic lifestyle.

Introduced at first into Europe by Spanish explorers in the 16th century from South America, and then from Europe to the world. Cultivated potato (Solanum tuberosum L.) is member of the Solanaceae family. It includes beside potato, tomato, eggplant, pepper, and tobacco. With a total production of more than 373 MT in 2011, potato is the 4th worldwide most important crop after rice, corn, and wheat. However, many pathogens and pests threaten potato, for instance *Ralstonia solanacearum, Clavibacter michiganensis, Alternaria solani, Rhizoctonia solani, PVY, PVX, Globodera pallida, G. rostochiensis, Leptinotarsa decemlineata... among these pathogens and pests, <i>P. infestans* is the most threatful. Control of *P. infestans* is still problematic. Actually, the most used control method is fungicide application. In a season generally, the farmers make between 12-17 applications. The most promising alternative control method is resistance breeding.

An outstanding aspect of resistance breeding is its durability. Unlike in the case of resistance of potato to virus X, resistance durability in the pathosystem *P. infestans-Solanum* spp. still a dream. The problem is due to the pathogen ability to evolve quickly, and overcome the resistance gene deployed and also to be resistant to fungicide used to its control. The problem is amplified with the possibility of sexual reproduction, after the discovery of the A2 mating type in Europe, and other places in the world. The sexual reproduction offers the pathogen more flexibility and ability to evolve rapidly. It will be easier to overcome host resistance, to be resistant to fungicides, to resist to unfavorable meteorological/climatic conditions.

From its first massive attack, in 1840s and the resulted Irish famine, until now many resistant genotypes were deployed. At first potato resistance breeding against *P. infestans* was based on the introgression of R genes from the wild relative species *S. demissum*. Eleven R genes were identified (R1-R11). From these 11 R genes, 5 (R1, R2, R3, R4, and R11) were the most popular between potato breeders ^[2]. The R genes originated from *S. demissum* have the disadvantage of being easy to overcome by isolates of *P. infestans* after few years of their release in the fields.

After the defeat of R genes originated from S. *demissum*, the search for other resistance source became more and more urgent. So the interest of researcher and potato breeders was oriented to other potato wild species than S. *demissum*. A multitude of Rpi genes were discovered and from a diversity of wild species: S. *bulbocastanum*, S. *stoloniferum*, S. *phureja*, S. *verrucosum*, S. *schenckii*, S. *venturii*, ^[3] a total of more than 20 R genes were isolated and cloned ^[4].

Resistance gene introgression was based at first on the conventional hybridization. However, with some other wild potato species, it is not possible such as *S. bulbocastanum*. Many alternatives were adopted to overcome this obstacle, for instance: somatic hybridization, genetic transformation.

Breeding with broad-spectrum resistance is advanced as the best strategy ^[5]. Broad-spectrum resistance is thought to durable resistance, but until now there is no evidence that these R genes are more durable than race specific R genes. However, it is an important step toward this goal. And what if many genes are gathered in the same plant?

Gene pyramiding, also called gene stacking, is "the accumulation of R genes into a single genotype or cultivar and it can be achieved using major *Rpi* genes, defeated *Rpi* genes, different alleles of one gene, or the same allele (allele dosage)" ^[6]. Gene stacking can be performed with different technologies, starting by the conventional natural hybridization, somatic hybridization, until genetic transformation (cis- and transgenesis). Genetic transformation has the advantage over these techniques, it quicker, more accurate, and actually, we can introduce more than one gene at a time ^[7], the most important inconvenient is with the consumer acceptance.

Breeding field resistance is more durable^[8], but not only that, the Combination of quantitative resistance improves the durability and efficiency of race specific R genes^[9]. According to Vleeshouwers et al.^[5] stacking broad-spectrum R genes promises more durable potato resistance to *P. infestans*.

Zhu et al.^[7] succeeded to introduce three wide spectrum *R* genes into Desiree: *Rpi-sto1* from *S*. *stoloniferum*, *Rpi-vnt1* from *S*. *venturii*, and *Rpi-blb3* from *S*. *bulbocastanum*. The resulted resistance was the sum of the individual R genes effects ^[7]. The potato cultivar with the most durable resistance against *P*. *infestans*, Sarpo Mira, carries at least 5 R genes (*R3a*, *R3b*, *R4*, *Rpi-Smira1*, and *Rpi-Smira2*) that confer qualitative and quantitative resistance ^[10].

And what if we could help the plant to evolve quickly as the pathogen does? It is feasible according to Vleeshouwers et al.^[2] via artiicial evolution by Random Mutagenesis. Segretin et al.^[4] succeeded to expand potato resistance to other strains of *P. infestans* via mutation of a single amino acid.

Here I cited only a part of the techniques used. Many other techniques exist and are helpful. The problem of *P. infestans* cannot be solved only by a technology or a practice; it needs an integrated system that takes in consideration the plant, the pathogen, and the environment (including the human factor).

REFERENCES

- 1. Park TH, et al. Molecular breeding for resistance to *Phytophthora infestans* (Mont.) de Bary in potato (Solanum tuberosum L.) a perspective of cisgenesis. Plant Breeding. 2009;128:109-117.
- 2. Vleeshouwers VGAA, et al. Understanding and exploiting late blight resistance in the age of effectors. Annu Rev Phytopathol. 2011;49:507-531.
- 3. Khavkin EE. Potato late blight as a model of pathogen-host plant coevolution. Russian journal of plant physiology. 2015;62:408-418
- 4. Segretin ME, et al. Single amino acid mutations in the potato immune receptor R3a expand response to Phytophthora infestans. MPMI. 2014;27:624-637.
- 5. Vleeshouwers VGAA, et al. Effector genomics accelerates discovery and functional profiling of potato disease resistance and *Phytophthora infestans* avirulence genes. Plos One 3. 2008;8:e2875.
- 6. Tan MYA, et al. The effect of pyramiding *Phytophthora infestans* resistance genes $R_{pi-mcd1}$ and $R_{pi-mcd1}$ in potato. Theor Appl Genet. 2010;121:117-125.
- 7. Zhu S, et al. Functional stacking of three resistance genes against *Phytophthora infestans* in potato. Transgenic Res. 2012;21:89-99.
- 8. Gebhardt C. Bridging the gap between genome analysis and precision breeding in potato. Trends in Genetics. 2013;29:248-256.
- 9. Wulff BBH, et al. Improving immunity in crops new tactics in an old game. Current Opinion in Plant Biology. 2011;14:468–476.
- 10. Rietman H, et al. Qualitative and quantitative late blight resistance in the potato cultivar Sarpo Mira is determined by the perception of five distinct RXLR effectors. MPMI. 2012;25:910-919.