Host and non-host resistance against *Phytophthora infestans*, the causal organism of late blight of potatoes and tomatoes

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Phytophthora infestans (Mont.) de Bary, the causal organism of late blight, attacks potato and tomato crops on a worldwide scale. This pathogen belongs to the family of the *Pythiaceae*, which together with the *Peronosporaceae* and *Albuginaceae*, makes out the order of the *Peronosporales*. These three families are marked by a great number of reputed pathogens. *P. infestans* is only known as a pathogen to attack potatoes since the period of 1842 to 1845 when it showed up to cause serious crop losses in North America and Europe. Its centre of origin and diversity is most likely in the highlands of central Mexico, but there is a small group of researchers which point at the Andes as an possible alternative centre of origin.

Up to the 1980s, a single RFLP-fingerprint clone (in *P. infestans* referred to as the US1 clonal lineage) of the mating type A1 was the only representative of *P. infestans* outside Mexico, whereas in the Toluca valley of Mexico a sexually reproducing population with great genetic diversity existed. Since *P. infestans* is a oomycete featuring a bipolar, heterothallic mating system, the US1 clonal lineage of *P. infestans* consequently did not produce sexually and hence did not form oospores tot survive crop free periods. Instead the pathogen survived crop free periods in infected tubers in storage.

The situation changed dramatically during the 1980s when a global migration of new clonal lineages and even sexually reproducing populations took place. In the Netherlands (and many neighbouring countries in Western Europe), a sexual population of *P. infestans* is now present in a large number of potato growing areas. The introduction of these new populations has had and still has a major impact on late blight epidemiology and disease management. In addition to the presence of oospores as a persistent infectious survival structure, genetic variation, adaptive abilities, aggressiveness and virulence has boosted. Fortunately to growers and wholly contrarily to most other *Phytophthora* species, *P. infestans* does not infect roots. Infections through oospores are mainly through leaflets and young stem tissues. For oospores to germinate and to infect, a 24 hours period of puddles in the field appears to be necessary. Compared to infection by asexually produced zoosporangia and zoospores, oospore initiated infections are relatively rare. However for a pathogen with an extremely rapid spreading capacity as *P. infestans*, this does not appear to be a major constraint.

For many reasons resistance is a basic part of control strategies for many pathogens. This is certainly the case with broad-leaved vegetables, where the customer does not allow any residues of pesticides. Also potato growing is under great pressure to minimize pesticide inputs because of concern about environment and health aspects and resulting government imposed rules. In addition, the increase of aggressiveness of the pathogen requires higher levels of resistance to counterbalance the increased risk of attack. Unfortunately, moderate levels of resistance may imply also increased oospore production.

Resistance breeding against late blight in potatoes started in the twenties of last century with the introduction of R-genes which in a short period were neutralized by new virulent strains of the pathogen. For many years, resistance breeding continued introducing new R-genes to fail again. Alternative resistance strategies incorporating field resistance, horizontal resist-

ance, polygenic resistance and partial resistance, presented as more durable forms of resistance emerged.

Durability of resistance is an important feature but an operational definition is not easy to present in case of potato breeding. Durability is associated with the economic life span of cultivars. For most cases, the effective life span of R-genes does not exceed five to seven years. For quite a number of crops this duration is long enough to be considered as durable. As long there are enough R-genes available, the problem appears manageable. The life span of potatoes is fairly long, anyhow much longer than the period that R-genes continue to be effective. Hence R-genes did not provide durable resistance with potatoes against *P. infestans*. Deliberate breeding for high level R-gene based resistance was abandoned and instead breeding for partial resistance was embraced. Unfortunately due to insufficient knowledge on the genetic background of partial resistance in general and on the genetic base of the partial resistance present in potato progenitors in particular, breeders still are introducing cultivars with high level partial resistance basically mastered by one or few genes. This is partly due to the fact that breeding for new potato cultivars and their introduction takes a long time, but also because breeders aim for high levels of partial resistance with a high heritability, which in case of polygenic resistance is a contradiction. Nevertheless it is to be questioned whether in a single potato clone high levels of polygenic resistance may be ever combined with the high quality standard and high yielding capacity of modern potato cultivars, which are also thought to be under polygenic control.

Introducing non-host resistance is often regarded as an attractive solution to the constraints involved with traditional R-gene and polygenic resistance. There are many pathogenic organisms, but each of them attacks a limited number plants species. A plant species not affected by certain pathogens is considered as a non-host for those pathogens and the resistance expressed as non-host resistance. In fact each plant species is non-host resistant to almost all pathogens except the relatively few ones which attack it. Species specific R-genes and QTL's are possible candidates to govern non-host resistance in a number of pathosystems. For closely related species this may be true as results of recent research concerning *Solanum* species and *P. infestans* and *Lactuca* species, and *Bremia lactucae* suggest. A question to consider is whether the introduction of resistance genes from a non-host plant species to a more or less related host species may not build a bridge for target pathogens to the non-host species. Building such bridges may form a much greater risk than using such genes from host species. To answer this question, basic knowledge has to be gathered on the nature and similarities of non-host genes, R-genes and QTL's and their function in plants. Similarly, functional analysis of virulence and avirulence genes and genes governing aggressiveness is still lacking. Before opening a possible box of Pandora we should first make a risk assessment about what is actually inside the box.

Questions can be raised about the validity of the non-host theorem in case no disease is found on a certain plant species in a specified region. Based on such assumptions research has been initiated in Ecuador by Plant Research International and the International Potato Center (CIP) in the centre of origin of a several solanaceous host and non-host species of *P. infestans*. Tree tomato, *S. betaceum*, was up to the eighties not affected by *P. infestans*, but in the last 20 years became severely affected by late blight. Other host species became more severely attacked. Results obtained so far give an interesting view in the kitchen of rapid evolution of host specificity and speciation in *P. infestans.*