Host Plant Resistance to Diseases: Potential and Limitations

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Abstract

Of several control measures available, host-plant resistance has been a strong choice for economical and effective management of plant diseases. During the natural host-pathogen interaction process, some pathogens have evolved to parasitize large number of hosts while others have remained specific to certain hosts and developed pathotypes and races that are specific to cultivars within a host form. Based on the genetic and biochemical parameters, resistance in plants has been classified into monogenic, oligogenic, polygenic, acquired resistance and post-transcriptional gene silencing. The conventional breeding for disease resistance in most crop plants has mostly utilized major resistance (R) *genes based on the classical gene-for-gene system that has often been accompanied by rapid breakdown of resistance. Polygenic or quantitative resistance has been used much less frequently than the major gene resistance and thus in many cases resistance has been nondurable. Following developments in molecular techniques, a number of R genes against different pathogens have now been cloned from different plant species that encode specific proteins and this has enhanced the possibilities of efficient resistance breeding. Many* R *genes lack durability because they can be defeated by a single loss-of-function mutation in the corresponding avirulence* (Avr) *gene. Pyramiding several R genes effective against multiple races of a pathogen using marker-assisted breeding and transgenic use of R genes and their clusters resistance provide future prospect for breeding cultivars for durable resistance.*

Keywords: Diseases, host plant, resistance, limitations

Introduction

The natural life forms can be classified into three categories as producers (green plants), consumers (organisms exploiting other organisms) and decomposers (organism using dead organisms) (Ribeiro do Vale *et al.*, 2001). Green plants, including agricultural crops are extensively used by multitude of consumers including vast array of insect pests and plant pathogens. During the course of continued evolution these green plants have developed broad based defense mechanisms to protect themselves from their consumers. These defense mechanisms are based on avoidance, tolerance or resistance. Studies have shown that host resistance by far is the most important defense mechanism that can be used to control diseases of agricultural crops caused by large number of pathogens, including fungi, bacteria, viruses and nematodes.

Despite substantial advances made in developing plant disease management strategies, the global food security is threatened by multitude of pathogens and pests causing about 30% production losses annually. Of several control measures available, chemical pesticides provide effective protection but their application is compromised by environmental effects and by the emergence of resistant pathogen strains. In addition, chemical control of plant diseases is beyond the means of resource-limited farmers of developing nations. In recent times biological control measure, based on microbial antagonism, has received prominent attention, but its effective application has been limited due to quality and supply problem, and the complex soil-microbe-climate interactions under field conditions. For these reasons, host-plant resistance has been a strong choice for economical and effective management of plant diseases. Disease management through host-plant resistance is based on simple and easily transferable seed-based technology that does not cost additional amount to farmers, although it is a time consuming and expensive exercise for the researchers. The process of developing a disease resistant cultivar involves a multidisciplinary approach involving plant pathologist, plant breeder, molecular breeder, plant physiologist and

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agronomist at different stages of research and development. Sound knowledge of biology and epidemiology of disease, host-pathogen interactions, development of effective and rapid screening technique for identification of resistance, utilization of resistance to develop agronomically desirable breeding lines and varieties and evaluating their agroecological adaptation and yield potential are the major steps involved in developing a disease resistant cultivar. Impressive progress has been made in managing several diseases through deploying resistance genes in the background of high yielding cultivars in a number of crops both at global and regional levels. However, the constant struggle between host-pathogen continues towards the natural evolutionary process leading to the cycle of boom and bust in most crop-pathogen systems.

Why a certain pathogen causes disease in one host plant and not in another has always intrigued plant pathologists. In nature resistance is a rule and susceptibility an exception. This is because plants have an innate ability to recognize potential invading pathogens and prepare for their defense. In contrast, successful pathogens cause disease because they are able to evade recognition or suppress host defense mechanisms. During the never-ending interaction between host and pathogen, some pathogens have evolved to parasitize large number of hosts while some others have remained specific to certain hosts and developed pathotypes and races that are specific to varieties within a host form.

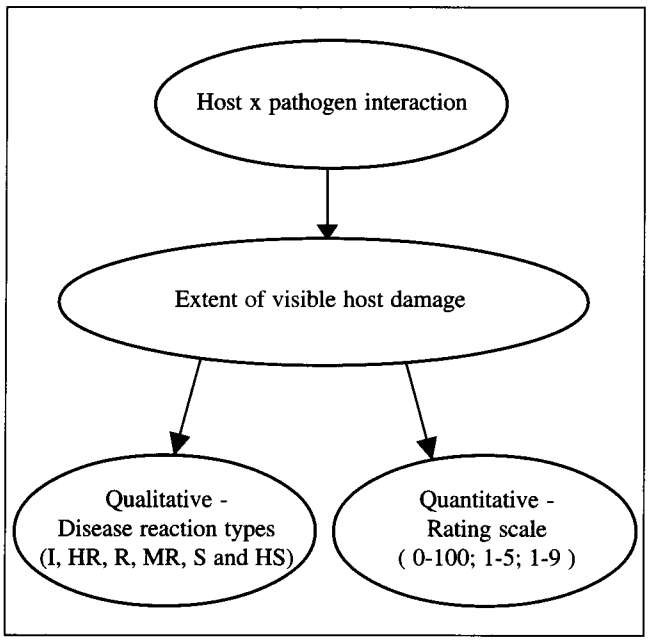

Figure 1. Host-pathogen interaction and measurement of qualitative and quantitative resistance in relation to standard susceptible and resistant checks

Types of resistance operating in crop plants

We come across various terms reported in literature for resistance types operating in different plant-pathogen systems. Based on the genetics of resistance these are basically of two types: a) monogenic, oligogenic – dominant/ single gene/major gene/vertical resistance/qualitative/race-specific/strong and non-durable; b) polygenic- recessive/ minor/multiplegenes/horizontal/partial/residual/ quantitative/race non-specific/weak and durable. The third kind of resistance that provides the biochemical basis of resistance emanating through biological control system is variously called as: acquired, induced, systemic, systemic acquired resistance (SAR), and others. Research on production of antifungal compounds, such as phytoalexins as a result of interaction of plants to external agents provided some insights into induced resistance. Certain fungal pathogens in genera of *Alternaria* and *Cochliobolus* are known to produce host-selective toxins (HSTs) as agents of virulence or pathogenicity (Khomoto and Otani, 1991). These HSTs have been shown to suppress host defences and making the host susceptible. Post-transcriptional gene silencing (PTGS) is another host defense system recognized in plants that operates against viruses (Cogoni and Macino, 2000).

Measuring resistance

The extent of visible host damage resulting from the complex host-pathogen interaction is measured to classify the plants into resistance or susceptibility group. Disease rating scale provides the measure of resistance and susceptibility of plants. Disease reaction types (immune, highly resistant, resistant, moderately resistant, susceptible, and highly susceptible) provide the qualitative assessment of resistance, while severity and incidence scales (such as 1-5; 1-9; 0-100) the quantitative assessment (Fig. 1). A simple and realistic disease rating scale is important to quantify and discern genetic differences between lines and plants.

Resistance is expressed in relative terms in reference to standard checks (susceptible and resistant). Various factors that influence resistance expression are: weather variables, inoculum density, interplot interference, disease symptoms, crop maturity, plant habitat, agronomic practices etc. The expression of resistance genes often get modified by the action of other genes. Adult plant resistance in wheat to rusts (Ribeiro do Vale *et al.*, 2001), recovery resistance in pearl millet to downy mildew (*Sclerospora graminicola*) (Singh and King, 1988) and foliar disease resistance at seedling stage in several crops are some of the examples of variable expression of resistance genes. There are temperature-sensitive resistance genes, such as L-genes in flax to flax rust (Islam and Mayo, 1990) and Sr and Lr genes in wheat to wheat rusts (Browder, 1985).

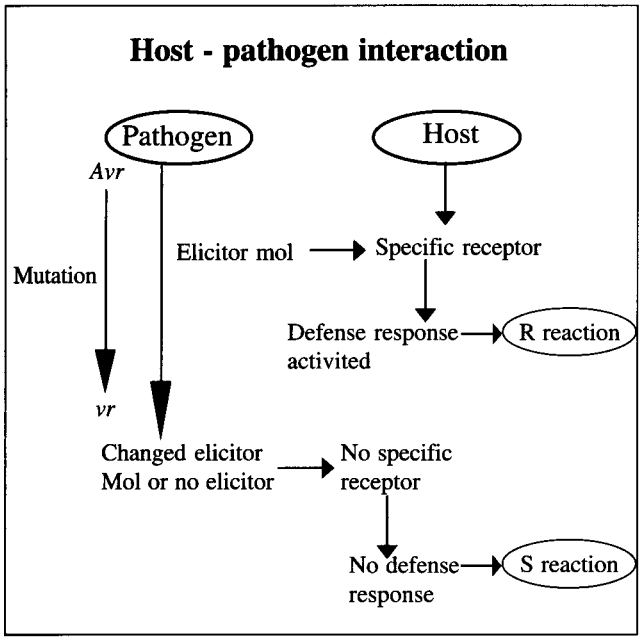

Figure 2. A conceptual framework of host-pathogen interaction at cellular level leading to expression of resistant or susceptible reaction

Genetics of host-pathogen interaction

With the classical work of Flor (1971) on genetics of interaction between flax and flax rust (*Malampsora lini*) we gained substantial understanding of the gene-for-gene hypothesis. The hypothesis states that plant contains single dominant resistance genes (*R* genes) that specifically recognize the complementary avirulence genes (*Avr* genes) of the pathogens. Avirulence genes in the pathogen encode a protein product that is recognized by the complementary *R* genes of the plants, which results in induction of defense gene expression (hypersensitive reaction - HR) and inhibition of pathogen growth (incompatible reaction). However, if the plants do not contain the *R* gene, the pathogen will be able to grow and infect them (compatible reaction), even though it contain *Avr* gene (Fig. 2). The modern molecular work is based on this classical concept of gene-for-gene relationship.

The HR is known to result from the specific interaction at the cellular level of the product of *R* gene and product of the *Avr* gene. If one of the two products is absent there are no incompatibility and susceptibility results (Staskawicz, 2001). Virulence is actually is the absence of avirulence, and genetically there is nothing like a virulence gene. This gene-for-gene system occurs more clearly and frequently in biotrophic pathosystems, such as rusts, smuts, powdery mildews and downy mildews of cereals. The resistance of these systems is typically race-specific and also get easily neutralized by new races of the pathogen. However, there are evidences to suggest that disease resistance in plants is a complex trait determined by multiple loci and that environmental effects and fitness trade-offs should be considered for the coevolutionary host-pathogen dynamics (Kover and Caicedo, 2001).

Identification and cloning of *R* genes

The Flor's gene-for-gene concept discussed above formed the basis of subsequent molecular cloning of pathogen avirulence genes and plant resistance genes. A number of *R* genes against different host-pathogen systems have been identified, their genetics studied and utilized in breeding programs. In some cases, *R* genes have been mapped and they have been found to cluster on few chromosome arms, some times they are linked tightly and known as complex loci. In flax-rust pathosystem 34 *R*-genes have been identified in seven regions and some of these regions are closely linked (Ribeiro do Vale *et al.*, 2001). A major breakthrough was the cloning and characterization of the maize Hm1 *R* gene controlling resistance to certain strains of *Cochliobolus carbonum* using transposon (*Mu*) tagging (Johal and Briggs, 1992). The Hm1 gene encoded an NADPH-dependent reductase that inactivates the toxin produced by the pathogen. Further studies revealed that plants share common mechanisms for disease resistance to diverse pathogens, and the R genes share common protein motifs.

A number of *R* genes against different pathogens have now been cloned from different plant species that encode proteins, which can be grouped into several super families based on protein domains (Dangl and McDowell, 2006). The vast majority of genes belong to the (nucleotide binding site-leucine rich repeats) NBS-LRR, eLRR, or LRR-kinase super families initially identified in tomato, tobacco, rice and *Arabidopsis* by map-based cloning or transposon tagging (Dangl and Jones, 2001; Wisser *et al.*, 2005). Resistance gene analogs (RGAs) can now be easily identified by sequence similarity, and genetic studies have revealed that the above R proteins are ubiquitous in plants. Thus, RGAs can be cloned by PCR-based approaches and/or by using genomic information and then genetically mapped. It is becoming practical to clone *R* genes from a variety of cops or their wild relatives and to rapidly transfer them into elite cultivars (McDowell and Woffenden, 2003). This is a major technology advance from the conventional breeding.

Breeding for resistance

Resistance breeding program has often used *R* genes using pedigree and backcross methods for controlling various plant diseases. Soon after a disease resistant cultivar is released farmers are confronted with problems of resistance breakdown or neutralization of resistance by new forms of the pathogen. Rapid increase in utilizing major resistance

genes in breeding programs has been accompanied by rapid breakdown of resistance. Polygenic or quantitative resistance (QR) has been used much less frequently than the major gene resistance and thus in many cases resistance has been nondurable (Parlevliet, 1995).

The classical resistance breeding uses well refined highly effective field and greenhouse disease screening techniques employing heavy inoculum load that generally identify major gene resistance but is inadequate to identify QR, unless the disease scores are taken on a quantitative scale. These screening approaches together with the belief that polygenic resistance is difficult to select for and might not give a good level of resistance has led to the greater exploitation of *R* genes than those of *QR* genes, QTLs. Although QR occurs in most crop species and more often under desirable and adapted cultivars and germplasm, it has not been exploited adequately. Using recurrent selection approach QR level can be improved much the same way as selection for higher yields and this resistance could likely be more durable than most major gene resistance (Ribeiro do Vale *et al.*, 2001). At ICRISAT, QTLs effective against specific pathotypes of pearl millet downy mildew have been identified and marker-assisted backcross breeding has been successfully used to develop a resistant hybrid "HHB 67-new" (Thakur and Hash, 2004) that is being grown commercially in Haryana and Rajasthan.

With the currently available molecular techniques the possibilities of efficient resistance breeding have been enhanced. The tissue culture and transformation techniques can transfer *R* genes a) between plant species, b) transfer genes from pathogens (pathogen-derived resistance); and c) production of new genetic constructs to enhance *R* gene expression in the plants. At ICRISAT, a coat-protein gene from the virus has been introduced in peanut variety JL 24 to ward off the *Indian peanut clump virus*, and chitinase gene from rice and glucanase gene from pea are being introduced into peanut varieties for providing protection against *Aspergillus flavus* (ICRISAT, 2005). Thus molecular techniques of transferring genetic material with the aim to obtaining resistance are enormously diverse and promising.

Prospects for durable resistance

Resistance is considered durable if it remains effective when used for many years over a substantial area (Johnson, 1984). Many *R* genes lack durability because they can be defeated by a single loss-of–function mutation in the corresponding *Avr* gene. Because individual *Avr* genes often make incremental contributions to virulence, pathogens can afford to alter or discard an *Avr* gene with little or no fitness penalty (Leach *et al.*, 2001). As an alternative to single-gene deployment, multiple *R* genes can be bred (gene pyramiding) into individual plant lines (Pink, 2002). In principle, the pathogen will have to accumulate multiple mutations in *Avr* genes to escape detection. This is unlikely to occur if the mutations have strong cumulative effects on virulence. Another approach is to use multilines - the mixture of lines each possessing different *R* genes in the same plot, and a susceptible line can be used in the mixture to reduce the selection pressure for mutation in the *Avr* genes (Mundt, 2002). Both pyramiding and multilines deployments have not been used widely owing to the time required for breeding assortments of *R* genes into elite cultivars.

Transgenic use of *R* genes that are known for durable resistance provides another prospect for breeding for durable resistance. For example, the pepper gene Bs2 has provided long lasting resistance against bacterial spot (*X. campestris*), and Bs2 has been cloned from pepper and shown to encode a nucleotide-binding site-leucine-rich repeat (NBs-LRR) protein (Tai *et al.*, 1999). The *Bs2* transgene works effectively in tomato against the same pathogen.

Non-host resistance genes have become more accessible to genetic dissection using molecular tools (Heath, 2000). For example, *Arabidopsis* and tobacco are uniformly resistant to many microbes that are serious pathogens of many crops. A maize *R* gene, *Rxo1* (governing resistance to some specific diseases) recognizes a rice pathogen *Xanthomonas oryzae* pv. *oryzicola* causing bacterial streak in rice but not in maize (Zhao *et al.*, 2005). Surprisingly, this gene also controls resistance to the unrelated pathogen *Burkholderia andropogonis* that causes bacterial stripe of sorghum and maize. Thus the same gene *Rxo1* controls resistance reaction to both pathogens and non-pathogens of maize. *Rxo1* has a NBS-LRR structure, similar to many such identified genes. *Rxo1* functions as a transgene to rice demonstrating the feasibility of nonhost *R* gene transfer between cereals (Zhao *et al.*, 2005). However, transfer of *R* genes from model species to distantly related species might be hampered by a phenomenon called "restricted taxonomic functionality" which needs to be better understood to help overcome this problem.

Naturally occurring defense-inducing compounds, such as synthetic analog of salicylic acid is being marketed as a foliar spray to control pathogens by inducing SAR in crops (Görlach *et al.*, 1996). A protein called Harpin, which regulates Type III secretion of virulence factor in pathogenic bacteria, can also induce SAR when applied as spray (Moffat, 2001).

Conclusion

Given the diversity of strategies that pathogens use and their ability to rapidly adapt to new cultivars and environment, it would unreasonable to think the development of a magical strategy for durable, broad-spectrum resistance. However,

with increasing knowledge of host-pathogen interaction and disease epidemiology, and rapidly available molecular tools it should be possible to provide better protection of crops against diseases. Both classical and molecular breeding approaches can be followed to utilize *R* genes and *QR* (QTLs) that are mostly present in cultivated species and their wild relatives, developing cultivars with multiple *R* genes and use of multilines and cultivar mixtures. Cloning additional *R* genes in model plants for their applicability, transforming genes or gene constructs encoded for resistance and application of functional genomic tools to disease resistance will provide new insights into interactions between defense signalling and other plant processes. Enhanced understanding of the structural basis of recognition to design R proteins that recognize essential virulence factors and elucidation of the biochemical functions of the Avr proteins and R proteins would be more useful. There is also a strong need for a very cohesive interdisciplinary team efforts to carry out various components of research and the disease resistant cultivars thus developed be properly and adequately evaluated under farmers' field conditions in different agroecological regions for resistance expression and its durability. Monitoring cultivar for resistance stability and durability is critical for appropriately guiding the resistance breeding program.

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