Resistance in Plants, Concepts and Mechanisms

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Abstract— *In contrast to most animals, plants are sessile organisms that they have not a circulatory system. So they have an innate immune system in each cell. In fact interaction between plants and their pathogens is based on systemic signaling capability from infection sites. In plant pathogen interaction sometimes pathogens produce elicitor and sometimes produce effector. On this basis, resistance in plants is divided to host and non host. On the other hand the plant responses depend not only on the recognition mechanisms but also very much on the biology of the interactions and genetic characteristics of plants and their pathogens. Gene – for – gene and the matching allel are two basic models for explanting of genetic basis of interaction between plants and their pathogens.*

Keywords— Effector, Elicitor, Qualitative Resistance, Quantitative Resistance, Plant Resistance.

I. INTRODUCTION

Have you ever thought that which of the two are more dangerous for humankind? Plant diseases or human diseases? Have you ever thought that all of kind of organisms from very small living things which have not even complete structural cell to human who is masterpiece of creation use green plants? So plants or green plants have a lot of consumers. If plants cannot defense against this wide spread range of consumers the human health is threatened. The role of plants not only in our foods but also in our dressing and or in bioproduct production either under field conditions or in storage is so clear that it dose not require additional explanation. So plants as benefactors of world must be able to combat with their risk factors and use from defense mechanisms that are principally based on avoidance, resistance or tolerance [11].

Avoidance is mainly active against animal parasites and includes such diverse mechanisms as volatile repellents, mimicry and morphological features like hairs, thorns and resin ducts. Resistance is usually of a chemical nature. Little is known of tolerance; it is very difficult to measure and is usually confounded with quantitative forms of resistance [27].

Resistance in plants can include two basic form, quantitative and qualitative [3]. Also the other forms that all of them are based on the genetic characteristics of plants and their pathogen agents especially the interactions of these two factors. On the other hand the study of these genetic traits is very important because genetic improvement of plants is the best way to manage of damage caused by plant diseases.

II. BASIC CONCEPTS

2.1 Elicitors and Effectors

Before any explanation about these basic concepts it must be clear that in contrast to most animals, plants are sessile organisms, they lack a circulatory system and their cells are framed with a rigid cell wall. These evolutionary constraints have resulted in the evolution of a primary cell – autonomous immune system [56]. In fact they have innate immune system in each cell with systemic signaling capability from infection sites [21].

In plant pathogen interaction, sometimes pathogens produce elicitor and sometimes produce effector. Elicitors such as peptides, metabolites, cell wall components, enzymes, and toxins are produced for suppressing plant defense [78]; [60]; [50] and [46] and are called pathogen/microbe – associated molecular patterns (PAMP/MAMP). These elicitors produce non – host disease resistance and have the ability to reduce the disease severity of actual pathogens (basal disease resistance) [57]. In this kind of resistance, following pathogen attack plant signal molecules are produced by damaged host that are named damage – associated molecular patterns (DAMP) [78]. These elicitors or PAMP/ MAMP/DAMP are recognized by the pattern recognition receptors (PRRs) that are biosynthesized in endoplasmic reticulum and transported to plasma membrane [47]. As a first line of defense response, the PAMP/MAMP trigger downstream genes resulting in no symptoms or race – non – specific hypersensitive response, generally referred to as the PAMP/pattern – triggered immunity (PTI) or non – host resistance [2]; [78]; [60]; [6] ; [76]; [45] and [7].

So elicitors induce similar defense responses in plants as induced by the pathogen infection [61].

And also nonhost resistance is a broad – spectrum plant defense that provides immunity to all members of a plant species against all isolates of a microorganism that is pathogenic to other plant species. Upon landing on the surface of a nonhost plant species, a potential bacterial pathogen initially encounters preformed and, later, induced plant defenses. This nonhost resistance response often results in a hypersensitive response (HR) at the infection site [53].

III. NON HOST RESISTANCE AND HYPERSENSITIVE RESPONSE (HR)

Plant immunity against the majority of microbial pathogens is conveyed by a phenomenon known as non – host resistance (NHR) [49]. This defence mechanism affords durable protection to plant species against given species of pathogens. This contrasts with the well – studied host resistance, mediated by the products of plant resistance (R) genes, which establish pathogen race – or cultivar – specific resistance. Whereas NHR routinely provides durable crop protection in the field, the effectiveness of host – resistance is characteristically transient.

The broad – spectrum nature of NHR closely parallels that exhibited by the innate immune system of animals. NHR, however, has proved difficult to characterise as a result of the absence of a tractable genetic system. It is thought to be genetically complex, involving the deployment of both constitutive and inducible defence responses, in combination with a host physiology that may be routinely incompatible with pathogenesis.

One of the most dramatic visible phenotypes that is frequently (but not always) associated with plant resistance is rapid localized cell death, the hypersensitive response (HR), at the site of infection, which is often compared with animal programmed cell death. This is an especially effective process in limiting pathogens that require living host cells [30].

The HR occurs in plants in response in infection by plant pathogenic fungi, bacteria and viruses. When an HR occurs the plant dose not succumb to infection and damage to the plant is limited to the cells in HR lesion. The mechnisms involved in generation the HR and ultimately causing resistance have been the subjects of intensive research. Much of research has been carried out with simplified experimental systems e.g. with fungal components called elicitors that they was mentioned above. These components cause necrosis in whole plant tissues or plant cell suspension cultures. The HR caused by plant pathogenic bacteria has also been studied. Historically this was because of the possibility of separating prokaryotic (pathogen) metabolism from eukaryotic (host) metabolism by using selective antibiotics and more recently because of the relative ease of using molecular genetics methodology on the pathogen. The HR can also be induced by viruses and a similar phenomenon has been reported in resistance of plants to some nematodes. Information about the HR has been obtained by studying many different host plants, such as *Arabidopsis*, barley, bean, cucumber, lettuce and tomato in response to viruses, bacteria, fungi, or a whole range of different elicitor molecules. The HR has been investigated in whole plants or in cell culture system. Thus it is perhaps wise to treat generalizations with caution while trying at the same time to sift out a unified picture of the HR as far as possible from the mass of information available from different experimental systems [80].

IV. EFFECTORS AND HOST RESISTANCE IN PLANTS

As it was mentioned earlier in plant pathogen interaction sometimes pathogens produce effector. Specialized pathogens produce race – specific intracellular elicitors called effectors, produced by specific avirulence (AVR) genes [78] and [69]. Though these are considered to be specific to biotrophs, several necrotrophs also produce effectors [78]. These effectors suppress other PAMPs and also the host resistance genes to become more virulent [44]. The effectors, depending on their domains, are recognized by plant – produced specific receptors (R proteins), encoded by R genes [78]; [22]; [21] and [58]. As a second line of defense response, the effectors trigger downstream genes resulting in race – specific hypersensitive response to contain the pathogen, generally referred to as the effector triggered immunity (ETI), qualitative resistance, or vertical resistance [78] and [50]. Such a resistance is considered to be monogenic and spawned the gene – for – gene hypothesis [18]. However, these effector recognition receptor genes are just surveillance genes and the real resistance genes that induce hypersensitive response are NADPH oxidase, callose synthase, etc., The genotypes rendered susceptible are considered to vary in basal resistance, partial resistance, or horizontal resistance [32]; [68] and [60].

V. HOST AND NONHOST RESISTANCE – SIMILARITIES AND DIFFERENCES

Given that host pathogens and nonhost pathogens can be recognised by similar mechanisms, it is not surprising that numerous studies have documented that defence responses to host pathogens and nonhost pathogens are also similar. However, the plant responses depend not only on the recognition mechanisms but also very much on the biology of the interactions, and so it is difficult to make useful comparisons between an incompatible host – pathogen interaction and a

nonhost – pathogen interaction. Any statement on the similarity of responses should therefore be based on closely related host and nonhost pathogens. A novel study of the expression of about 8000 *Arabidopsis* genes in response to different strains of *Pseudomonas syringae* [82] would seem to meet this criterion. There is a problem, however, in that the nonhost pathogen used in this study, *Pseudomonas syringae* pv. *phaseolicola*, does not activate HR [52], unlike the avirulent host pathogens that were used for comparison. Nevertheless, changes in the expression of approximately 2000 response genes were largely shared among two incompatible interactions, which were mediated by single R genes, and the nonhost interaction [82]. Currently, comprehensive gene expression arrays are being generated for barley and wheat, and these will allow interesting comparisons of host and nonhost resistance. Closely related fungal pathogens that have apparently identical infection biology exist for these cereals, but they are strictly separated in host and nonhost pathogens. The best example may be the formae speciales of the powdery mildew fungus.

Efforts have been made to elucidate whether the defence signalling steps known from host resistance are shared by nonhost resistance. The ENHANCED DISEASE SUSCEPTIBILITY1 (EDS1)[1] protein, which is necessary for R – gene – mediated resistance to many pathogens in *Arabidopsis* [55] is also important for nonhost resistance to isolates of *Peronospora parasitica* and, which are pathogenic on closely related Brassicas [24]. Furthermore, a recent study of the role of the plant SUPPRESSOR OF G2 ALLELE OF SKP1 (SGT1)[2] protein in resistance to different diseases supports the idea that host and nonhost plants share certain defence signaling components [36]. In yeast, SGT1 plays a role in SCF (Skp1 Cullins[3] F – box proteins) E3 – ligase – mediated ubiquitylation and may regulate a number of R – protein functions [59]. As a result of silencing studies of SGT1 in *Nicotiana benthamiana*, this list was expanded to include the non LRR (Lucin rich repeat) R gene Pto[4] [36]. Interestingly, nonhost resistance to two bacterial pathogens also requires the SGT1 protein [36]. Nevertheless, resistance to the nonhost bacterium *Xanthomonas campestris* pv. *campestris* and to Cauliflower mosaic virus (CaMV), a nonhost virus, were not affected by the silencing of SGT1. Salicylic acid (SA) signaling in host resistance has received much attention over the years [35], but the extent to which it is involved in nonhost resistance remains unclear. Degradation of SA in *Arabidopsis* salicylate hydroxylase (NahG) lines confers susceptibility to the nonhost bacterium *P. syringae* pv. *phaseolicola* [52]. New observations suggest, however, that this is caused by the catechol that is produced during the degradation of SA [74]. Hence, even though this nonhost bacterium has nonhost avr – genes [37] that may have corresponding R genes in *Arabidopsis*, the interaction of these genes may be overruled by strong general elicitors that activate innate immunity. Interestingly, *Arabidopsis* manifests no HR to infection by *P. syringae pv. phaseolicola* [55], neither does it manifest an HR during general elicitor mediated innate immunity [42]. However, this innate immunity does seem to involve signalling components that are shared with R – gene – mediated signaling [43]. An *Arabidopsis* mutant, nho1 (for nonhost resistance1), has been isolated on which *P. syringae* pv. *phaseolicola* grows and causes disease symptoms [52]. It is significant that this mutant is also compromised in R – gene – mediated resistance to *P. syringae*. These observations should be kept in mind when interpreting the recent genomic study which shows that the response of 2000 *Arabidopsis* genes to *P. syringae* pv. *phaseolicola* is slowed and reduced as a result of NahG –mediated degradation of SA [82].

VI. GENETIC BASIS OF HOST AND NONHOST RESISTANCE

6.1 Avirulence genes and resistance genes

Virulence gene is a gene that, during the disease process, codifes the production of factors that alter host – cell structures and functions [14].

Avirulence genes in pathogens are those genes that confer the ability to be recognized by a resistant host plant [30]. A number of avirulence genes have been cloned from plant pathogens, particularly viral and bacterial avirulence genes [62]. Viral avirulence genes encode a range of functions including capsid proteins and replicase proteins. Comparison of the gene products of bacterial avirulence genes shows that they are mostly unrelated and their function is largely unknown. There is

[1] - Positive regulator of basal resistance and of effector-triggered immunity specifically mediated by resistance proteins such as TIR-NB-LRR (TNL) that is Toll interleukin-1 receptor-like nucleotide-binding site leucine-rich repeat

[2] - an iated disease resistanceessential component of *R* gene-med in plants

[3] - are a family of hydrophobic scaffold proteins which provide support for ubiquitin ligases (E3). All eukaryotes appear to have cullins. They combine with RING proteins to form Cullin-RING ubiquitin ligases (CRLs) that are highly diverse and play a role in myriad cellular processes, most notably protein degradation by ubiquitination [8].

[4] - The Pto gene encodes a serine-threonine kinase that confers resistance in tomato to *Pseudomonas syringae* pv tomato strains expressing the avirulence geneavrPto.

now evidence that the bacterial avirulence gene products are introduced into plant cells by a type III secretion mechanism[5] [48]. Further evidence suggests that these gene products are involved in enhancement of bacterial virulence (in the absence of the corresponding host resistance gene) and so are analogous to the virulence effector proteins delivered to animal cells by mammalian bacterial pathogens [83]. Only a few fungal avirulence genes have been cloned owing to the more complex genomes of fungi. The products of these genes include small, secreted proteins of unknown function [62]. In one case, a fungal avirulence protein from the rice blast fungus has similarity to a zinc protease [12]. Pathogens require signals from the plant to induce cell differentiation and express essential pathogenicity genes. This requirement for cues from the plant is obvious for rust fungi, in which hyphal differentiation is induced by the surface topography of the plant [15]. A recent study of the barley powdery mildew fungus (*Blumeria graminis* f. sp. *hordei* [Bgh]) suggested that the composition of the surface wax is important in activating the development of a differentiated appressorium [54]. Wax composition could therefore be involved in determining whether plants are hosts or nonhosts. Presence of preformed barriers is often claimed to be a first line of plant defence. Conceptually, preformed plant cell walls, antimicrobial enzymes, and secondary metabolites would be ideal early obstacles for the pathogen. These barriers are undoubtedly important in defence against many host and nonhost pathogens, but their success in preventing disease probably depends on the degree of co – evolution of the pathogen and host. However, little documentation is available on the role of preformed defences. Nevertheless, one example in which a host pathogen is adapted to overcome a preformed antimicrobial compound, whereas a closely related nonhost pathogen is not, clearly confirms that preformed barriers can provide important defences. The wheat root pathogen *Gaeumannomyces graminis* var. *tritici* is not adapted to attack oats successfully and cannot cause disease on oat roots. The related oat root pathogen *G. graminis* var. *avenae* depends on its ability to detoxify avenacin A – 1, an antimicrobial saponin, to proceed in its life cycle. A mutant oat line, which no longer expresses avenacin A – 1, is susceptible to *G. graminis* var. *tritici.* It appears therefore that *G. graminis* var. *tritici* is a nonhost pathogen of oats because it lacks the enzyme that detoxifies avenacin [41]. Plants can mount several barriers in response to attack by both host and nonhost pathogens, and these responses can be independent of the genotype of the individual pathogen. 'General elicitors' may be released during attacks by both host pathogens and nonhost pathogens, and the barriers that are activated in response to these elicitors contribute to resistance towards both types of microbes. Flagellin, a protein of the bacterial flagella, serves as such an elicitor. This protein activates defence through a pathway that involves FLAGELLIN INSENSITIVE2 (FLS2), a leucine – rich repeat (LRR) receptor kinase and a mitogen – activated protein (MAP) kinase cascade [77]. Other general elicitors are released during the enzymatic degradation of pathogen cell – wall polymers, when the pathogen is making its way into the host. This group of elicitors include oligomers of chitin and glucans. Flagellin and oligomers, released from pathogen cell walls, typically activate the production of antimicrobial proteins and phytoalexins (antimicrobial secondary metabolites) in the plant tissue. Such general elicitors are often indispensable to the microbe, and plants are suggested to exploit them during recognition in the same way as animals recognise PAMPs [24]. The use of general elicitors together with the involvement of an LRR – receptor kinase and a MAP kinase[6] cascade make this defence mechanism reminiscent of animal 'innate immunity' systems, suggesting a shared ancient system for combating invaders [77]. Other examples of defence mechanisms that have similarities to innate immunity are the activation of defence in parsley and potato nonhost plants by Pep – 13 , a peptide fragment from *Phytophthora sojae* [10], and the activation of defences in nonhost tobacco by the 'harpin' protein of *Pseudomonas syringae* [33]. These plant responses certainly impede pathogen proliferation in a quantitative manner, but their contribution to nonhost resistance is not entirely clear. An example of an inducible structural barrier, which is potentially activated by general elicitors, is the papilla. This local cell wall fortification is formed on the inner side of plant cell walls at the site of fungal penetration. The papillae formed in response to powdery mildews are well studied and serve a significant role in keeping the fungus out [16] and [65]. A case of papilla – based resistance, which is unusual because it gives complete protection, is the barley mlo[7] – resistance to the *Blumerie graminis* f. sp. *hordei* (Bgh) host pathogen. MLO

[5] - (often written Type III secretion system and abbreviated TTSS or T3SS, also called Injectisome) is a protein appendage found in several Gram-negative bacteria. In pathogenic bacteria, the needle-like structure is used as a sensory probe to detect the presence of eukaryotic organisms and secrete proteins that help the bacteria infect them. The secreted effector proteins are secreted directly from the bacterial cell into the eukaryotic (host) cell, where they exert a number of effects that help the pathogen to survive and to escape an immune response.

[6] - A mitogen-activated protein kinase (MAPK or MAP kinase) is a type of protein kinase that is specific to the amino acids serine and threonine.

7- The Mlo-related proteins are a family of plant integral membrane proteins, first discovered in barley. Mutants lacking wild-type Mlo proteins show broad spectrum resistance to the powdery mildew fungus, and dysregulated cell death control, with spontaneous cell death in response to developmental or abiotic stimuli.

negatively regulates papilla formation, and so mutations in this gene cause resistance [5]. Papillae are also important in nonhost resistance. In wild type *Arabidopsis*, about 80% of the conidia of the nonhost pathogen Bgh are stopped in association with papillae. These mutants have been studied to study nonhost resistance. Mutations in the PENETRATION1 (PEN1) and PEN2 genes reduced the plants' ability to arrest Bgh – conidia to about 20% of that of wild type plants. The identity of PEN1 strongly suggests the importance of vesicle trafficking in penetration resistance. (Mutation in PEN2 cause constitutive cell wall changes, and therefore this gene may represent a defence mechanism [16]. The *pen* mutants are paralleled by the required for MLO – specified resistance1 (ror[8]1) and ror2 mutants in barley. These ror mutants were isolated as suppressors of mlo – resistance and, like the *pen* mutants, they reduce penetration resistance to Bgh significantly [5]. Significantly, the barley ROR2 gene is a functional homologue of the *Arabidopsis* PEN1 gene [16]. This discovery strongly suggests that elements of papilla – related vesicle trafficking are conserved between host and nonhost resistance, even in these distantly related plant species.

Many ideas have been documented in an attempt to explain the genetic basis of host resistance to pathogens. The gene – for – gene model refers to a specific genetic interaction between a host and its pathogen. It states that a resistance gene (*R* gene) in the host and an avirulence gene (*Avr* gene) in the pathogen must be present for the host to be resistant. *R* genes in the host recognize *Avr* genes in the pathogen and a defense response is activated preventing the establishment of the pathogen in the host. [18]. was the first to discover and present this matching specificity between host and pathogen genes. His work in selective crop breeding with flax lead to the rather unintentional detection of this interaction between this plant and its fungal pathogen, flax rust. He showed that for every gene in the plant that conditioned resistance, a corresponding and complementary gene in the pathogen that conditioned avirulence existed [64]. Since the initial proposal of this gene – for – gene idea evidence for its presence has been presented for over 25 different plant host – pathogen pairs [72]. However, this proposed gene – for – gene interaction between host and pathogen is controversial. Evidence for this interaction has almost exclusively been found in plants and more specifically agricultural crops [1] and for many reasons this model, although it exists, may not be applicable to a wide selection of host – pathogen interactions. Crop plants are subject to strong artificial selection. Interestingly there is increasing evidence to suggest that the intensity of selection may influence the number of genes involved in [34]; [20] and [38]. So it is possible that evidence for a simple gene – for – gene interaction in crop plants is the result of artificial selection of only a small subset of genes which would have been selected to defend plants from pathogens under natural conditions. In addition, most crops are grown in stable and often optimized conditions and exposed to minimal competition with other species [19]. The size of crop plant populations are also controlled and therefore fluctuate less through time than natural populations [19]. It is important to consider the contribution that both artificial selection and growing conditions may have on the nature of the genetic interaction between hosts and pathogens when assessing the wider applicability of the gene – for – gene view. The lack of evidence for this interaction in natural populations supports the observation that this model may have limited application outside crop plant – pathogen interactions. Indeed the attraction of this theory may be its simplicity rather than its accuracy [71]. Investigation of the genetic basis of host resistance to pathogens continued following the initial proposal of the gene – for – gene view by [18] leading to the development of more sophisticated models. Another view termed the matching – allele model has been presented to explain the genetics underlying host resistance [72]. This model suggests each host allele confers resistance to one parasite allele. Resistance in the host occurs when its genotype and that of its pathogen match exactly. For successful infection by the pathogen to occur the genotype of the host and pathogen must not match. In contrast to the gene – for – gene model, which predicts that parasite host range will vary greatly, the matching – allele model predicts that all parasites can attack the same number of hosts [23]. Whereas the gene – for – gene model has been important in the understanding of host pathogen interactions in plants, the matching – allele model is favored by zoologists [1]. A matching – allele interaction has been documented in both studies with invertebrates [66] and vertebrates [31]. However, overall there is little experimental support for this alternative genetic scenario [23] and [39]. Although the gene – for – gene model and the matching – allele model have been considered independent in much of the literature it has been suggested that they are in fact two ends of a continuum [1]. Assessment of allele frequency dynamics along this continuum revealed that the variance of allele frequencies under a gene – for – gene system can strongly approach those predicted by matching – allele models if the costs of virulence and resistance are great [1]. In light of such findings there may be doubt as to how the two models can be discriminated between given certain data [73] and [13]) and it is possible that results of past studies based on allele frequency variance may be misinterpreted and

8- RAR-related orphan receptors (RORs) are members of the nuclear receptor family of intracellular transcription factors.

fitted to the wrong model [73]. Rather than considering both models as being equally likely to explain the results, it may be that if the results fit what could be the outcome of the gene – for – gene model the matching – allele model will not be considered. The consideration of both models in explaining empirical data is necessary given their convergence under certain conditions. According to neodarwinian theory traits which evolve at the highest rate will be those that are controlled by genes at many loci which each have a small effect [34]. Evidence suggests that commonly the evolution of resistance to pathogens is relatively rapid [79]. The gene – for – gene model and the matching – allele model suggested to account for host resistance are based on the interaction at one genetic locus only and therefore contrast this prediction [70].

VII. OTHER FORMS OF DIVISION OF PLANT RESISTANCE

7.1 Qualitative and quantitative resistance

Resistance, like other traits, occurs in a qualitative or in a quantitative way. With the former the different genotypes in a population occur as discernible phenotypes; it is usually controlled by a major gene. Quantitative resistance (QR) is defined as a resistance that varies in a continuous way between the various phenotypes of the host population, from almost imperceptible (only a slight reduction in the growth of the pathogen) to quite strong (little growth of the pathogen). This resistance is often indicated with other terms such as partial, residual and field resistance or even (wrongly) with tolerance. QR occurs at various levels to nearly all important pathogens in most cultivars of our crops. For example barley and barley leaf rust, *Puccinia hordei*. The QR to this pathogen inherits polygenically [26] and all cultivars in Western Europe, including the very susceptible cultivars, carry at least some QR. Most cultivars, though, carry considerable levels of QR, preventing the barley leaf rust from becoming a major pathogen in Western Europe [27]. In Ethiopia the barley landraces represent a centre of diversity. Barley genotypes without any QR are very rare [4]. Also Wheat/yellow rust, wheat/leaf rust, and barley/powdery mildew. Breeding in Western Europe is directed at selecting major genes of the non – durable type to protect against these three major pathogens. In this way, new recommended cultivars tend to enter the recommended cultivar list with high scores for resistance. After a number of years these scores are much lower as the major gene resistance is not effective any more. After the resistance "breaks down", QR becomes visible if present. All cultivars selected for their major gene resistance appear to carry moderate to fair levels of QR hidden behind that major gene. This hidden QR is sometimes indicated as residual resistance.

Therefore, QR is present almost everywhere. Cultivars without any QR are very rare. For this type of resistance breeders do not need to look for primitive genotypes from centres of diversity nor to related wild species. The resistance is near at hand in adapted cultivars, a fortunate situation as it makes breeding easier. [63] concluded that the ideal sources of resistance are those present in closely related, commercial genotypes, and any effort to transfer resistance from related species and genera should be considered long term. QR is, except for a few cases of monogenic, incomplete inheritance, inherited oligogenically or polygenically. Examples of the former are the QR of maize to *Puccinia sorghi* Schw [75] and of wheat to *Puccinia recondita* [42], based on a few (two or three) additive genes. Polygenic QR is exemplified by the field resistance of potato to *Phytophthora infestans* [81] the QR of maize to [*Cochliobolus heterostrophus* (Drechsler)] and [*Setosphaeria turcica* (Luttr)] [40], in rice to bacterial blight [51] and of barley to *Rhynchosporium secalis* (Oud.) Davis. [67] and to *Puccinia hordei* [26], and of rice to [*Magnaporthe grisea* (T.T. Hebert) Yagashi and Udagawa] [9]. Polygenic QR is usually supposed to be non race specific, but does not appear to be so. In the polygenic pathosystems mentioned above, small race – specific effects have been reported [25] and it is probable that polygenic resistance to specialized pathogens often goes together with small race – specific effects. [28] described this in the following way: When resistance in the host and aggressiveness in the pathogen interact on a polygene – for – polygene basis and several host cultivars are tested against a series of pathogen isolates the general impression is that of non – race – specificity. Most variation is between cultivars and between isolates. If the accuracy of the experiment is sufficiently high small but significant race – specific effects can be observed.

VIII. BREEDING AND RESISTANCE

In order to reduce costs and to increase the efficiency of identifying resistant plants or lines in segregating populations, breeders developed screening methods in which plants as young as possible were exposed to high concentrations of, preferably, a specified inoculum. This efficiently identifies complete resistance based on major genes but is inadequate for recognizing small differences in resistance. These screening approaches, together with the belief that polygenic resistance is difficult to select for and might not give a good level of resistance, led to the present situation where major gene resistance has been exploited very well, while QR has been used only sparingly. This is unfortunate as there is so much QR available. Quantitative resistance occurs to most of our important pathogens at various levels in nearly all our crops as discussed under ''Quantitative Resistance.'' Since this QR occurs in the cultivars grown, it is genetic material that is related to what the

breeders' desire. For this type of resistance breeders do not need to look for primitive genotypes from centres of diversity nor to related wild species. The resistance is near at hand in adapted cultivars, a fortunate situation as it makes breeding easier. [63] concluded that the ideal sources of resistance are those present in closely related, commercial genotypes, and any effort to transfer resistance from related species and genera should be considered long term. To select for QR means accumulating QR in much the same way as selecting for higher yields. The breeder selects the plants or lines with the lower levels of disease severity and by doing that continuously over the seasons, the level of QR will increase fairly rapidly as [29] showed. There is, however, one complication. If there is also non – durable major gene resistance around, it has to be taken into account. The QR is not visible when such an effective major gene is present. By using, preferably, local material, the frequency of such non – durable still effective major genes is often low, as the local pathogen population has adapted to these genes. Introducing plant material from elsewhere, especially from the centres of diversity, increases the frequency of such non – durable effective major genes considerably, as the local pathogen population has not yet adapted to the newly introduced resistance. Therefore, to select QR stick as much as possible to local material as they will almost certainly carry QR. One can also avoid ending up with non – durable major resistance in the selected material by selecting against susceptibility, i.e. removing the most susceptible plants and lines all the time [29]. Plants or lines with complete resistance should also be removed in case of resistance breeding against specialized fungal pathogens, as such resistances can be assumed to be non – durable. In case of non – specialized pathogen and virus's one may use any resistance [11].

IX. CONCLUSION

In nature organisms are divided to producers and consumers. Green plant are the most important of producers so that without them life in planet earth is impossible. All of our efforts for understanding of mechanisms of interaction between plants and their pathogens are for production of more food and better food. For this purpose we need to more and better understanding of mechanisms that interfer in plant resistance. These studies are in different levels. At this time the study of basis genetic of resistance (of plants against pathogen) is very important. With the possibility of isolating specific genes that they have desirable characteristics it becomes possible to make genetic constructs by combining genes from different origins or even by changing the isolated genes. The possibilities are almost infinite and each construct has to be tested.

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