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Invited Expert Review

Disease Resistance in Maize and the Role of Molecular Breeding in Defending Against Global Threat

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Abstract

Diseases are a potential threat to global food security but plants have evolved an extensive array of methodologies to cope with the invading pathogens. Non-host resistance and quantitative resistance are broad spectrum forms of resistance, and all kinds of resistances are controlled by extremely diverse genes called "R-genes". R-genes follow different mechanisms to defend plants and PAMP-induced defenses in susceptible host plants are referred to as basal resistance. Genetic and phenotypic diversity are vital in maize (*Zea mays* L.); as such, genome wide association study (GWAS) along with certain other methodologies can explore the maximum means of genetic diversity. Exploring the complete genetic architecture to manipulate maize genetically reduces the losses from hazardous diseases. Genomic studies can reveal the interaction between different genes and their pathways. By confirming the specific role of these genes and protein-protein interaction (proteomics) via advanced molecular and bioinformatics tools, we can shed a light on the most complicated and abstruse phenomena of resistance.

Keywords: Maize; concept of pathology; R-genes; types of resistance; genetic diversity; genome wide association study.

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Introduction

Global production of all cereals is not sufficient to feed the total population and meanwhile a declining trend in total agricultural population over the past decade was observed (**Tables 1** and **2**, http://faostat.fao.org). Plant diseases are a potential threat to global food security and endure as the focus of extensive research using a wide range of methodologies. It has been assessed that in the United States only, regardless of the prevention measures practiced, each year, crops worth $ 9.1 billion were lost to disease (Agrios 2005). Loss of crops from plant diseases may result in hunger and starvation, especially in developing countries where access to disease-control methods is limited and annual losses of 30 to 50 percent are common for major crops. Maize (*Zea mays* L.) is the world's most extensively grown crop with an annual worldwide production of 822 and 817 million tons in 2008 and 2009, respectively (http://faostat.fao.org). Maize is affected by an average of

Table 1. The total population of the world and agricultural population along with annual percentage

Year	1999–2001	2003–05	2008	2009	2010
Agric. (000) ha	2 571 119	2 598 516	2 617 264	2 620 710	2 623 741
Total population (000)	6 115 333	6 432 972	6 750 057	6 829 362	6 908 685
% Agric. Pop	42.04	40.39	38.77	38.37	37.97

Table 2. The production of cereals crops worldwide and area harvested in thousands

Year	1999–2001	2003–05	2007	2008	2009
Area (000) ha	672 096	679 725	698 029	712 226	708 495
Productivity (000) tons	2 084 449	2 212 315	2 353 652	2 520 700	2 489 302

almost 100 pathogens but only a fraction of diseases are present in a given location depending upon various factors and rarely do the number of these diseases become severe. The major diseases of maize along with their causal organism are listed in **Table 3**. Furthermore, wherever available, the minimum and maximum losses in one growing season along with their references are given for specific diseases. The most important and destructive diseases are leaf blights, stalk rots, ear and kernel rots, seedling diseases, and sometimes bacterial and viral diseases also cause economic losses to total production of maize crop. Common smut and crazy top are the example of miscellaneous diseases but are not usually destructive. Maize disease can occur at any growth stage (Ali et al. 2011a) i.e., from germination to maturity and for this purpose two types of scales (0–9 and 0–5) have been developed by scientists (Poland et al. 2011; Kump et al. 2011; Ali et al. 2011b) to describe losses at different developmental stages. To date, the overall losses of maize diseases have never been published and can't be sourced but it has been broadly mentioned that different diseases cause different economic losses in different regions of the world. The most important one in history is the destruction of southern corn leaf blight fungus (*Helminthosporium*

Table 3. Some important diseases of maize crop along with their losses and causual agent

S.No	Disease	Causal agent	% Losses	Reference
1	Northern corn leaf blight	*Setosphaeria turcica*	13–50	Tefferi et al.1996
2	Southern corn leaf blight	*Cochliobolus heterotropus*	15–46	Zwonitzer et al. 2009
3	Gray leaf spot	*Cercospora zeae*	5–30	Ward et al. 1999
4	Curvularia leaf spot	*Cochliobolus lunatus*	10–60	Akinbode 2010
5	Brown spot	*Physoderma maydis*	6–20	Lal and Chakarvati 1976
6	Southern corn rust	*Puccinia polysora*	20–80	Liang and Wu 1993
7	Common corn rust	*Puccinia sorghi*	18–49	Groth et al. 1983
8	Eye spot	*Aureobasidium zeae*	14–44	Chang and Hudon 1990
9	*Alternaria* leaf spot	*Alternaria tenuissima*	3–7	–
10	Top rot	*Gibberella subglutinans*	–	–
11	Head smut	*Sporisorium reilianum*	Up to 30	Njuguna 2001
12	Common smut	*Ustilago zeae*	40–100	Pope et al.1992
13	Ear rot	*Fusarium verticillioides*	5–15	Ako et al. 2003
14	Crazy top downy mildew	*Sclerophthora macrospora*	Significant	
15	Banded leaf and sheath blight	*Rhizoctonia cerealis*	0–60	Tang et al. 2004
16	Stalk rot	*Pythium inflatum*	25%	–
17	Root rot	*Fusarium graminearum*	25–30	Hebbar et al. 1992
18	Maize dwarf mosaic	*Maize dwarf mosaic virus*	0–90	Goldberg and Brakke 1987
19	Maize rough dwarf	*Maize rough dwarf virus*	10–70	Dovas et al. 2004
20	Bacterial stalk rot	*Pseudomonas zeae*	85	Thinda and Payakab 1985

Note: In case of occurrence of disease the minimum and maximum losses are given and only one scientific name of the causing organism is given for simplicity as other synonyms are also used in many articles for different pathogens.

maydis race T), which caused about one billion dollars losses in 1970. Most recently, it was reported from Ohio State University that only 30 different diseases affect maize in Ohio, and only about half cause economic losses. Even so, 5 percent to 15 percent of the maize crop is lost to disease each year, which amounts to nearly $ 100 million in lost farm income. The diseases of Ohio are divided into five groups for identification and listed in **Table 4**. Categorization of maize diseases based on the underlying pathogen phenomena will facilitate the formation of valid conclusions by the scientific community concerning the mechanisms of disease. The estimated global losses in maize due to some diseases (not including insects or viruses) were about 9% in 2001–3 (Oerke 2005). These losses varied significantly by region with estimates of 4% in northern Europe and 14% in western Africa and South Asia (http://www.cabicompendium.org/cpc/economic.asp). All together in the world the total amount of losses from maize diseases will cost billions of dollars, though its economic costs are extremely hard to estimate. Maize diseases are extremely difficult to identify because of the involvement of several factors such as herbicide injury, nutrient deficiency or excess, soil pH, soil compaction, genetic abnormality, and weather-induced injury. Therefore, a detailed laboratory examination is required most of the time. “How do pathogens attack plants? How do plants defend themselves? Why does a pathogen infect one species but not the other? And how to stop losses from diseases?” are the fundamental and utmost significant queries of the scientific community.

Recently, scientists have rapidly used the powerful technologies of modern molecular biology such as association mapping and joint linkage and association mapping to confirm genes for different traits (Lin et al. 2011). These techniques will facilitate the scientists in making profound improvements on the basic understanding of plant disease resistance and improving the genetic make-up of plants. Relatively little is known about the genetic architecture of many plant traits (Mackay et al. 2009) because the phenotypic variation in most traits is the outcome of several to many genes involved in the biological system and each gene has a small to moderate effect on phenotype. In exploring the mechanisms of disease resistance, several studies showed that different genes are involved in regulating the pathways that control either plant growth or activation of defense responses against pathogens (Darvill et al. 1992; Cote and Hahn 1994; John et al. 1997; Esquerre-Tugaye et al. 2000).

The present review will discuss the basic terminologies used in disease resistance in crop plants and will mainly focus on resistance in maize. The review describes the whole mechanism of the disease resistance and will try to focus on the future perspectives of disease resistance along with the role of genetic diversity, association mapping, combined linkage and association mapping and genomic selection in overcoming this devastating global problem.

The Phenomenon of Resistance in the Light of History

During the last several decades of its history, plant disease research has focused on understanding the interactions of and developing of more effective means to control disease (Sequeira 1993; Keen 2000; Degefu and Hanif 2003). Plant diseases are controlled by adopting certain techniques like quarantine measures, cultural practices, application of chemicals and most importantly development of disease-resistant genotypes by genetic methods (Strange 2005).

Since the early 20th century, classical breeding for disease resistance in plants has been the primary method for controlling plant diseases. The process of inheritance of resistance to pathogens started from the work of Biffen in 1905 by crossing resistant and susceptible wheat cultivars and observing segregation in the F_2 generations (Biffen 1905). He obtained his results by growing the parents and their subsequent generation, under natural infection and concluded that there was one recessive gene that was responsible for resistance. The collection of the data in early stages showed that there were basically three kind of plants, namely, those severely rusted, those essentially rust free, and those with an intermediate amount of rust with the ratio of F_2 plants being 1:1:2 (severely rusted : rust free : intermediate), respectively. At maturity there were only severely rusted and essentially free of rust plants at a ratio of 3:1, respectively. According to the interpretation of data from the end of the season, Biffen concluded that the resistance was due to one recessive gene. Had he chosen to interpret the data collected during the development of the epidemic, he might have concluded that resistance was partially dominant (Ellingboe 1981). These events are mentioned to show the importance of the interpretation of data in understanding inheritance of disease resistance. As it was a novel study for its time, the description was not sufficiently clear and there was not enough evidence was present to support the results but advanced techniques in molecular biology and specially achievements in association mapping will provide more evidence and reliable results for elaborating the phenomenon of disease resistance.

A substantial understanding of the genetic interactions that control disease resistance in plants was explained by Flor in the 1940s. He published his seminal work on the genetics of the interaction between flax and its obligate rust pathogen, *Melampsora lini* (Loegering and Ellingboe 1987). Flor’s work was novel, insightful, and under-appreciated at the time as he concurrently studied the inheritance of resistance in the host and virulence in the pathogen (Staskawicz 2001). The conclusion of Flor’s work is that for each resistance gene in the host there was a corresponding avirulence gene in the pathogen (Flor 1946, 1947, 1955) and his results clearly showed that the number of genes that distinguished two cultivars depended on the isolate of the pathogen. **Table 5** explains the

Table 4. All the major diseases of maize causing significant losses in Ohio (USA) and different races of different pathogens

Leaf blights	Stalk rots	Ear and kernel rots	Seedling diseases	Virus	Miscellaneous
Northern corn leaf blight race 0, 1	Gibberella	Gibberella ear rot	Pythium	Maize dwarf mosaic	Common smut
Southern corn leaf blight race T, O	Anthracnose	Diplodia ear rot	Fusarium	Maize chlorotic dwarf	Crazy top
Northern leaf spot race 1, 2, 3	Fusarium	Fusarium kernel rot	Gibberella	Wheat streak mosaic	Nematode diseases
Stewart's bacterial wilt and leaf blight	Diplodia	Nigrospora ear and cob rot	Bipolaris		
Anthracnose	Bipolaris	Penicillium rot			
Eyespot	Bacterial	Aspergillus rot			
Common Rust	Nigrospora				
Gray leaf spot					
Yellow leaf blight					
Holcus spot					
Bacterial stripe					
Physoderma brown spot					
Southern Rust					

Table 5. The phenomenon of relationship with two genes in the pathogen and two genes in the host

	Pathogen	Host			
		A	B	C	D
		R1R1 R2R2	R1R1r2r2	r1r1 R2R2	r1r1 r2r2
A	P1P1P2P2	resistant/Avir	resistant/Avir	resistant/Avir	susceptible/Vir
B	P1P1p2p2	resistant/Avir	resistant/Avir	susceptible/vir	susceptible/Vir
C	P1p1P2P2	resistant/Avir	susceptible/Vir	resistant/Avir	susceptible/Vir
D	P1p1p2p2	susceptible/Vir	susceptible/Vir	susceptible/Vir	susceptible/Vir

host-pathogen relationship during the involvement of two genes in host responding to two genes in the pathogen. It is now possible to understand why other investigators, who had crossed the same cultivar, had come to different conclusions. We can easily conclude that the numbers of genes present in both host and pathogen can explain the exact type of resistance and if these genes segregate in next generation, the host will respond in a different way depending upon the genes it carries.

During the 1950s scientists came across a problem that high levels of resistant cultivar became susceptible having a single major gene of resistance (Ellingbao 1981) and it was observed that the pathogens have the potential of more severe racial changes. They were near to find out another phenomenon as they came to know that different cultivars have different levels of resistance and susceptibility in epiphytotics. It was supposed that this type of resistance did not follow the gene-for-gene hypothesis and a lot of different terms were used for this phenomenon (Person 1959). This puzzle was solved in 1960s by Van der Planck by suggesting two type of disease resistance. One is vertical resistance which follows the pattern of gene-for-gene hypothesis and is being controlled by only a few race-specific genes. It is obvious that this resistance which is based on one or few genes is temporal and the racial change of the pathogen will overcome the hurdle of resistance. The other type of resistance is horizontal resistance which is controlled by many minor genes, each of which contributed a small effect to restrict all races of pathogens from infection and was considered as permanent resistance (Van der Planck 1963; Ellinboe 1981). The review of McMullen and Simcox showed the genomic organization of disease resistance genes in maize. These scientists divided maize genome into 100 "bins" of approximately 20 cM each (Gardiner 1993; Davis et al. 1999) and were nominated by the chromosome number and a two-digit decimal (e.g., 1.01, 1.02, etc.). Furthermore, they

found out evidence of nonrandom distribution of resistance genes by summarizing the positions of reported resistance loci according to those bins.

Investigating the root cause of disease resistance, in the 1990s the main focus of research was "the role of cell wall in disease resistance". During the interaction of the pathogen and host, the forefront barrier is a plant's cell wall. The degradation of cell wall is pivotal for the pathogen to penetrate and colonize in the host for causing losses to the concerned plant. The various polymers of which it is comprised may assist as substrates to the various enzymes secreted by microbial pathogens, providing them nutrients (Walton and Cervone 1990; Degefu et al. 1995). The plant's cell wall is not an inert and static structure but a vital extension of the cytoplasm (Robinson 1991). The cell wall comprises of components for signaling and communications by simplistic continuity through plasmodesmata (Carpita 1996; Ebel and Mithöfer 1998). Signals from the cell wall elicited by pathogen and insect attack activate the defense response genes against these attacks (Albersheim and Darvill 1985; Ryan 1990; Ryan and Farmer 1991; Darvill et al. 1992). There are powerful signaling molecules responsible for acting at minimal concentration to deliver information to the plant under attack (Ryan and Farmer 1991; Shibuya and Minami 2001). In response to this information (the elicitor) the plant defense response is activated often through activation of genes that encode enzymes responsible for the synthesis of phytoalexins (Peck et al. 2001).

Selective pressure on host plants exerted by virulent pathogens has resulted in the coevolution of plant resistance (*R*) genes, which specifically recognize pathogen strain- or race-specific factors and allow the establishment of pathogen race/plant cultivar-specific disease resistance (Abramovitch and Martin 2004; Chang et al. 2004; Espinosa and Alfano 2004; Jones and Takemoto 2004). The pathogen growth is arrested by the *R* gene product's ability to recognize avirulence-dependent signals; this phenomenon triggers a chain of signal transduction events that culminates in activation of defense mechanisms. Genetic overlap between specific and basal resistance responses suggested that one function of the R-mediated signaling is to more rapidly and effectively activate defense mechanisms that are shared by both pathways (Glazebrook 1997; Yang 1997; McDowell and Dangl 2000; Dangl and Jones 2001). *R*-genes can be mapped through Mendelian genetics and have been cloned through many methods. Their modes of action along with complete description of signal transduction pathways have been defined in previous findings (Glazebrook 2001; Hammond-Kosack and Parker 2003; Rathjen and Moffett 2003; Wisser, et al. 2006). This type of resistance conforms to the gene-for-gene hypothesis and is genetically determined by complementary pairs of pathogen-encoded avirulence (*avr*) genes and plant resistance genes (homologous plant-microbe interaction; specific incompatibility) (Gabriel and Rolfe 1990; Prell and Day 2000). Gene-for-gene disease resistance is economically important as it is used in numerous crops to confer extremely effective disease resistance (Russell 1978; Lucas 1998; Simmonds and Smartt 1999). Plants have numerous *R* genes and pathogens have many *Avr* genes. Simply described, disease resistance is observed if the product of any particular *R* gene has recognition specificity for a compound produced due to a particular pathogen *Avr* gene. Most *Avr* proteins are considered to be virulence factors required for the colonization of host plants, which (upon recognition by resistant host plant cultivars) act as pathogen race-specific elicitors of plant defense and thereby deceive the microbe to the plant's surveillance system (Abramovitch and Martin 2004; Alfano and Collmer 2004; Jones and Takemoto 2004).

Earlier findings about *R* genes have been widely reviewed (Bent 1996; Hammond and Jones 1997; Dang and Jones 2001;Jones and Dang 2006). The immense *R* genes encode proteins that carry a leucine-rich repeat (LRR) domain, either as part of intracellular NB-LRR proteins that also carry a nucleotide binding (NB) site and other conserved domains, as an extracellular LRR in transmembrane receptor-kinase proteins, or in "receptor-like proteins" that have an extracellular LRR and a transmembrane domain but then very little at the intracellular C terminus of the protein (Bent and Mackey 2007).

Summarizing the diverse *R* genes, most of them share a striking degree of homology on conserved motifs. They mainly include a nucleotide-blinding site (NBS), leucine-rich repeat (LRR), a motif with homology to the cytoplasmic domains of the Drosophila Toll protein and the mammalian interleukin-1 receptor (TIR), a coiled-coil (CC) or leucine zipper (LZ) structure, transmembrane domain (TM), and protein kinase domain (PK). According to these features, at least four classes are distinguished among most R genes as follows: NBS-LRR, Receptor-like kinase (RLK), LRR-TM and TM-CC (Jones and Dang 2006). The NBS-LRR genes represent the largest class of R genes, and encode proteins with a variable N-terminal domain of approximately 200 amino acids (aa), connected by a predicted NBS domain of approximately 300 aa and a more variable tandem array of approximately 10 to 40 short LRR motifs. Furthermore, the NBS-LRR genes are categorized into three subgroups based on the motif within their N-terminus: TIR group, CC or LZ group and non-motif group (Bent and Mackey 2007).

The modern methods of biotechnology and genetic engineering are the easiest and less time-consuming methods to develop resistant varieties (Staskawicz 2001). Wissser et al. (2006) summarized previous publications on the mapping of maize disease resistance loci and reported the locations of 437 quantitative trait loci for disease, 17 resistance genes and 25 R-gene analogs. Most recently, the maximum number of disease resistance QTLs was identified through linkage-association

mapping and all the genes were annotated to different kinds of proteins (Poland et al. 2011; Kump et al. 2011).

Basic Concepts of Pathology

The lines of attack of most plant pathogens are extremely diverse and involved different phenomenon followed by pathogen and host. Pathogenic bacteria proliferate in intercellular spaces (the apoplast) after entering through gas or water pores (stomata and hydathodes, respectively), or succeed to enter through wounds. Nematodes and aphids feed directly by inserting their stylet into the plant cells. The most important and largest group of plant pathogen, fungi, can directly invade plant epidermal cells, or spread hyphae on surface of, between, or through plant cells. Pathogenic and symbiotic fungi and oomycetes have the ability to produce special feeding structures (haustoria), into the host cell plasma membrane. All the pathogen classes use their specific way of invasion into the plant for successful enhancement (Jones and Dangl 2006). Basically the pathogens are divided into two main classes, necrotrophic (non-obligate parasite) and biotrophic (obligate parasite). The necrotrophs kill the host plant and feed on the content while the biotrophs require a living host for their survival and completion of life cycle (Dangl and Jones 2001). The immune responses of plant show high polymorphism is used to identify and respond to biotrophs (Dangl and Jones 2001).

The basic mechanism of disease can't be completed in the absence of pathogen, host and environmental conditions. This phenomenon is referred to as disease triangle, determined by the above mentioned three-way interaction of the pathogen, the plant, and the environmental conditions (Lucas 1998). Each pathogen requires a specific environmental condition to cause maximum destruction to the host (Fry et al. 1993).

Pathogenesis

Maize pathogens have plenty of pathogenicity genes that are required for infection or for enhancing host virulence. As the pathogen comes in contact with the host, pathogenesis starts. Pathogenesis is the series of events that occurs in the host-pathogen interaction, including infection and colonization of the host, and development and dissemination of the pathogen (Chung et al. 2010). The pathogenic capacity of an organism is determined by its virulence factors. Virulence is by definition the degree of pathogenicity within a group or species of microorganisms or viruses as indicated by case fatality rates and/or the ability of the organism to invade the tissues of the host. Avirulence genes (*Avr*) can be defined as genes in the pathogen that encode a protein product that is conditionally recognized directly or indirectly only by those plants that contain the complementary resistance gene. A specific interaction was governed in plant pathogen interaction that is the pathogen avirulence gene correspond with the resistance *R* genes of the host plant; if the both these genes are present, the plant resist the disease and if one is missing disease results (Flor 1971; Dangl and Jones 2001). Mitogen-activated protein kinases (MAPKs) are in the family of serine/threonine protein kinases and have been determined to be vital for pathogenesis of many pathogenic fungi including the natural pathogen of maize, *Cochliobolus heterostrophus* causing southern corn leaf blight (Takano et al. 2000; Zheng et al. 2000).

A successful pathogen has the ability to cause disease in the plants (Collinge et al. 2010) irrespective of the host genetic nature or they are able to evade recognition or suppress host defense mechanisms or both, while a successful plant must withstand adverse conditions irrespective of the genetic background of the pathogen, even if the environmental conditions favor the pathogen's growth and development (Staskawicz 2001). However, if the host plant's genetic background does not contain the *R* gene, the invader is still capable of initiating disease in that plant even though it still contains the avirulence gene (Dangl and Jones 2001). These diverse steps are involved in the development of diseases and the resistant plants combat the invading pathogen in the beginning of the process to survive successfully. Sometimes it is hard to find out the exact interaction between pathogen and host, i.e the pathogen-host relationship.

Epiphytotics

Epiphytotics affect a high proportion of the host plant population (Agrios 2005), sometimes across an extensive area. They may be mild or destructive and can be local or regional in occurrence. Epiphytotics result from various combinations of factors, including the right combination of climatic circumstances and monoculture. An epiphytotic may take place when a pathogen is introduced into a zone in which it had not formerly existed (Ullstrup 1972). Epiphytotics can also occur when host plants are cultivated in large acreages where previously little or no land was devoted to that specific crop plant. The environmental features that may affect growth and development of plant diseases and determine whether they become epiphytotic include temperature, relative humidity, soil moisture, soil pH, soil type, and soil fertility. Many pathogens, especially among the bacteria and fungi, spend part of their life cycles as pathogens and the remainder as saprophytes (Van der Planck 1963).

Types of Resistance

Resistance to disease varies among plants i.e, sometimes the plant shows immunity to a specific pathogen and

sometimes a plant is tolerant to a pathogen, suffering minimal injury (Van der Planck 1963). Naturally plants may be infected by several pathogens at a time (Strange 2005). Any plant that must contend with nutrient deficiency or an imbalance between soil moisture and oxygen is often more susceptible to several pathogens. Most of the plants when infected by one pathogen become prone to the invasion of secondary pathogens (Bent 1996). The combinations of all pathogens that affect a plant one after the other make the disease extremely complex. For a disease to be recognized, a scientist must be well aware of normal growth habits, varietal characteristics and normal variability of plants within species. Furthermore, a variety of terms have been used by different scientists for explaining the phenomenon of resistance in detail which include complete resistance versus partial resistance, horizontal versus vertical, monogenic versus oligo/polygenic, qualitative versus quantitative, major-gene versus minor-gene and narrow-spectrum versus broad spectrum. This diversity of terms adds an element of confusion to the literature because some terms are used in different ways by different authors. On the other hand, it shows the range of interests and assumptions made by the respective authors. We will try to give a brief description to the reader in order to understand all the names used in the field of disease resistance and to clearly distinguish the basic types of resistance.

Non-host and Host Resistances

Potentially phytopathogenic microorganisms incapable of infecting any cultivar of a given plant species are referred to as heterologous pathogens, while plants that are resistant to all isolates of a given pathogen species are called non-host plants (heterologous plant-microbe interaction; basic incompatibility) (Gabriel and Rolfe 1990; Prell and Day 2000). Non-host resistance is the most durable type of resistance and is defined as resistance of an entire plant species to all isolates of specific microbial species. This broad-spectrum defense strategy is of prodigious potential for agricultural applications. Even though this mechanism is less understood, still it is obvious that this type of resistance is more durable and provides resistance against pathogens throughout all members of a plant species (Mysore and Ryu 2004). Interplay of both constitutive barriers and inducible reactions comprises the basis for this most durable form of plant disease resistance (Thordal-Christensen 2003; Jones and Takemoto 2004; Mysore and Ryu 2004; Nurberger and Lapka 2005).

Certain components are responsible for the non-host resistance and disruption of any of these components leads to loss of non-host resistance against certain pathogens. The first hurdle for a pathogen before invading the plant is its passive defense mechanism. Two basics things (plant cytoskeleton and actin microfilaments) have been implicated in playing a role in defense against fungal penetration (Mysore and Ryu 2004). The second obstacle an invading pathogen has to face is the inducible plant defense mechanisms. It has been mentioned that phytoalexins (low molecular weight antimicrobial compounds that are synthesized *de novo* in response to pathogen attacks) are responsible for this component. The third component involved in plant defense signaling comprises of certain hormones like ethylene and salicylic acid (Knoester et al. 1998; Dempsey et al. 1999). Ethylene perception has been mentioned as required for basal resistance against pathogens and it can also induce disease resistance in plants. Furthermore, salicylic acid has been implicated in playing crucial role in non-host resistance (Mysore and Ryu 2004). Wound-induced protein kinase (WIPK), salicylic acid-induced protein kinase (SIPK) and heat shock protein (Hsps) have been previously implicated as signaling components of plant defense reactions (Zhang and Klessig 2001; Kanzaki et al. 2003). Broad spectrum disease resistance genes are the fourth important component involved in defense of plants. Several non-host disease resistance genes have now been identified and they are required for non-host resistance against certain non-host pathogens. An *Arabidopsis* non-host resistance gene, NHO1, was recently identified and subsequently cloned (Lu et al. 2001; Kang et al. 2003).

Mysore and Ryu (2004) classified the non-host resistance against bacteria, fungi and oomycetes into two types: type I and type II. The type I non-host resistance does not produce any visible symptoms (necrosis) and the type II non-host resistance was always associated with rapid localized necrosis or localized cell death. A single pathogen species can trigger both type I and type II non-host resistances in different plant species. For example, *P. syringae* pv. *phaseolicola* triggers type I non-host resistance in *Arabidopsis* and type II non-host resistance in tobacco (Lindgren et al. 1986; Lu et al. 2001). The purpose of describing non-host resistance is to find out genes in non-host species (which will be definitely of major effect) and transfer the gene(s) to the host. In control environments these studies have been carried out successfully (Zhao et al. 2004; Zhao et al. 2005) but the important step will be field application of these advanced techniques in field experiment.

A large number of plants are non-hosts for several pathogens because of no relation, but when a host-pathogen relationship does exist, susceptibility is more common than resistance (Fry 1982). The use of host resistance is a more reliable and eco-friendly way to reduce losses. The ability of a microorganism to produce disease can be evaluated only in terms of the host reaction, and conversely the resistance, or immunity, of the host can be judged only with regard to its effect on the microorganism. Host-plant resistance describes a range of adaptations evolved by plants which improve their survival and reproduction by reducing the infection of pathogens. Heath

(2000) and Kamoun (2001) stated that infrequent changes in the host range of phytopathogenic micro-organisms are indication of the stability of plant species resistance. It is believed that this particular type of resistance relies on multiple protective mechanisms that comprise of both constitutive barriers and inducible reactions (Heath 2000; Kamoun 2001; Thordal-Christensen 2003; Mysore and Ryu 2004; Nürnberger et al. 2004). Preformed physical or chemical barriers constitutively present on the plant surface (wax layers, rigid cell walls, antimicrobial secondary metabolites, such as phytoanticipins) may initially stop establishment of infection structures (Heath 2000; Dixon 2001; Kamoun 2001; Nürnberger et al. 2004). During evolution, plant species resistance was overcome by individual races or strains of a given pathogen species through the acquisition of virulence factors, which enabled them either to evade or to suppress plant defense mechanisms (Abramovitch and Martin 2004; Alfano and Collmer 2004; Chang et al. 2004). Such plants are considered hosts that were rendered susceptible to colonization by so-called homologous pathogens (homologous plant-microbe interaction; basic compatibility) (Gabriel and Rolfe 1990; Prell and Day 2000). Most of the crop plants have evolved two approaches to identify pathogens (Chisholm et al. 2006; Jones and Dangl 2006). On the external surface of the host cell, conserved microbial elicitors called pathogen associated molecular patterns (PAMPs) are recognized by receptor proteins called pattern recognition receptors (PRRs, Boller and Felix 2009). PAMPs are typically essential components of whole classes of pathogens, such as bacterial flagellum or fungal chitin (Dodds and Rathjen 2010). Plants also react to endogenous molecules released by pathogen attack, such as cell wall or cuticular fragments called danger-associated molecular patterns (DAMPs). Stimulation of PRRs leads to PAMP-triggered immunity (PTI). The second class of perception involves recognition by intracellular receptors of pathogen virulence molecules called effectors; this recognition induces effector-triggered immunity (ETI) (Dodds and Rathjen 2010). PAMP-induced defense in susceptible host plants is insufficient to stop infection; nonetheless, it is referred to as basal resistance. The PAMPs recognized by plants are multifarious and comprise proteins, carbohydrates, lipids and minor molecules, such as ATP (Boller and Felix 2009).

Quantitative Disease Resistance (QDR) and Multiple Disease Resistance (MDR)

Host plants use several strategies to defend themselves against damage, caused by several pathogens. In plants most disease resistance is quantitative rather than qualitative in nature (Wisser 2006). Chung et al. (2010) divided broad-spectrum resistance into two classes: (1) resistance effective against all known variants of a given pathogen ("race non-specific resistance") and (2) resistance effective against more than one pathogen ("multiple disease resistance"). Broad spectrum resistance to several diseases may be more durable than simply inherited resistance because of the evolution of novel races of pathogens (Van der Planck 1968).

R-genes falls in the category of complete resistance but still the response mediated by resistance genes has often proven short lived, temporary and race specific (Chung et al. 2010). The best known *R*-genes can provide high levels of resistance or even complete immunity (Chung et al. 2010). *R*-gene-mediated resistance is initiated through a gene-for-gene interaction; the recognition of a pathogen effecter by a host protein encoded by the *R*-gene leads to the induction of the hypersensitive response (HR), the production of antimicrobial metabolites such as phytoalexins and the expression of pathogenesis-related (PR) proteins (Jones and Dangl 2006). This type of interaction, typically resulting in a highly effective but race-specific defense response against pathogenic invasion, is sometimes known as qualitative resistance.

Qualitative disease resistance is monogenic, usually controlled by one gene or a few genes with major effects, whereas quantitative disease resistance (QDR) is generally polygenic, controlled by many minor genes (Van der Plank 1963; Ross 1986). Quantitative resistance confers intermediate levels of resistance and is believed to be controlled by a set of genes distinct from, or partially overlapping with, those involved in qualitative resistance (Wesser et al. 2005; Fu et al. 2009). Quantitative resistance is presumably more durable because multiple genes with minor effects lead to lower selection pressure and greater complexity to overcome (Parlevliet 2002). A large number of quantitative trait loci (QTL) for disease resistance have been mapped in plants (Young 1996; Poland 2009), but little is known about the underlying genetic basis or defense mechanisms involved.

Although rapid development has been achieved in recent years in the genetic classification of quantitative disease resistance (Bent and Mackey 2007), improvement in the understanding of the genetic and physiological processes underlying QDR has been restricted due to their complexity, incompleteness and variable expression (Geiger and Heun 1989; Young 1996; Kelly and Vallejo 2006). QDR tends to be associated with more durable resistance (Poland et al. 2011), because a pathogen strain that overcomes a single allele of small effect does not gain a large selective advantage, and loss of effectiveness of a single gene does not leave the host completely susceptible (Ayliffe et al. 2008; Rosewarne et al. 2008; Poland et al. 2009).

Mapping quantitative trait loci (QTLs) is a powerful tool for genetic dissection of QDR. A range of mechanisms have been associated with QDR, some of which are broader in spectrum and more durable than others. Traditionally, partial resistance

has been more difficult to transfer than simply inherited resistance due to its presumed multi-genic nature. Molecular mapping techniques in combination with marker-assisted selection, however, may enable breeders to identify and exploit these forms of resistance more effectively (Young 1996). Some major resistance genes have been observed to confer moderate levels of either race-specific resistance e.g. *Rp1* in maize (Smith and Hulbert 2005) or race-nonspecific resistance e.g. *RB* in potato (Song et al. 2003). Similarly in rice, the recessive allele of a susceptibility gene *Pi21*, encoding a proline-rich protein with putative heavy-metal binding and protein-protein interaction motifs, contributes resistance to blast disease (Fukuoka et al. 2009). The resistance of these non-*R*-genes has thus far been unwavering. Disease non-specific QDR have been found to be controlled by genes intricate in basal resistance, systemic acquired resistance, and defense signaling pathways (e.g. *RPW8.1* and *RPW8.2* in *Arabidopsis* (Wang et al. 2007); *npr1* in *Arabidopsis* (Cao et al. 1998)). Agriculturally important genes of this type, including *Lr34* in wheat (Krattinger et al. 2009) and *mlo* in barley (Buschges et al. 1997) have been shown to confer durable resistance to a number of obligate pathogens. Several studies have been carried out to map R-genes, resistance gene analogs (RGAs), and loci conditioning QDR (quantitative trait loci for disease, or disease QTL) in plants. DNA markers tightly linked to QRLs controlling QDR can be used for marker-assisted selection (MAS) to incorporate valuable traits.

Multiple disease resistance (MDR) is another form of resistance, in which the same locus is responsible for resistance to several pathogens, is both practically and conceptually significant and yet is also below par understood (Zwonitzer et al. 2010). Incomplete confirmation is available concerning quantitative trait loci (QTL) conditioning MDR. The revealing of clusters of QTL conferring resistance to multiple diseases is consistent with but does not prove the hypothesis that MDR genes exist in plants (Wisser et al. 2005; Wisser et al. 2006). More direct evidence for MDR is the observation of pleiotropic effects on multiple diseases revealed with certain induced gene mutations (Cao et al. 1997; Century et al. 1997; Lee et al. 2002; Nurmberg et al. 2007). Mitchell-Olds (1995) investigated genetic correlations among levels of disease resistance of *Brassica rapa* to three fungal pathogens: *Peronospora parasitica, Albugo candida*, and *Leptosphaeria maculans.* He reported heritable genetic variation for resistance to all three concerned pathogens and positive, statistically significant genetic correlations between resistance to *L. maculans* and *P. parasitica* in populations in which selection was directed at only one of the pathogens. Recently, Balint-Kurti et al. (2010) observed highly significant correlations between resistances to southern leaf blight (SLB), gray leaf spot (GLS), and northern leaf blight (NLB) in the maize intermated B73 × Mo17 (IBM) population, although they did not spot any disease resistance QTL associated with resistance to all three diseases. Zwonitzer et al. (2010) described that analysis of complex trait inheritance in single population can only provide a partial understanding of its genetic architecture; however, because of the potential genetic heterogeneity of such traits across diverse germplasm, tremendous space is available for innovation in this field. Therefore, to understand the complete phenomenon of MDR and the phenomenon underlying, explanatory and evidential experiments are required to explore the idea. This will require genetic exploration of MDR in different mapping populations and through different techniques of molecular biology and bioinformatics.

Plants possess several preformed defense strategies but also activate species-level resistance, race-specific resistance and race-non-specific resistance, which vary within species and between species. Certain types of resistance are well understood and summarized here, while MDR needs to be confirmed that either it works against a specific strain of a pathogen or all the strains of the concerned species of the different pathogens (**Figure 1**).

Need for Genetic Diversity

Throughout the history, it is obvious that monoculture or similar genetic background of crop plants is the basic cause of epidemics in different parts of the world. Plant diseases can be minimized by the reduction of the pathogen's inoculum, inhibition of its virulence mechanisms, and most importantly, the promotion of genetic diversity in the crop (Strange 2005). The cultivation of maize with male-sterile cytoplasm grown on 60 million acres in the United States, a classical example of monoculture, was confronted in 1970 by a virulent new race of the southern corn leaf blight fungus (*Helminthosporium maydis* race T), causing damage to about 700 million bushels of corn (Ullstrup 1972). A projected loss of one billion dollars was attributed to the 15% drop in total production due to SLB race T (Tatum 1971; Hooker 1972). This new race then disseminated widely and was more recently reported in maize growing areas worldwide. Southern corn leaf blight is a disease of global importance and is a considerable threat to the maize-growing areas (Kump et al. 2011). The major gene for resistance to pathogen attack has been widely used for protection of field crops but up till now no known gene confers complete resistance and the breeding community solely depends upon quantitative resistance (Kump et al. 2011). To avoid destruction of the entire maize crop and cope with different types of pathogens at the same time, the prevailing thing in breeding maize is genetic diversity. The utmost vital and extensively exploited plant sources of disease resistance can be found at the crop's center of origin, where crop diversity is the highest.

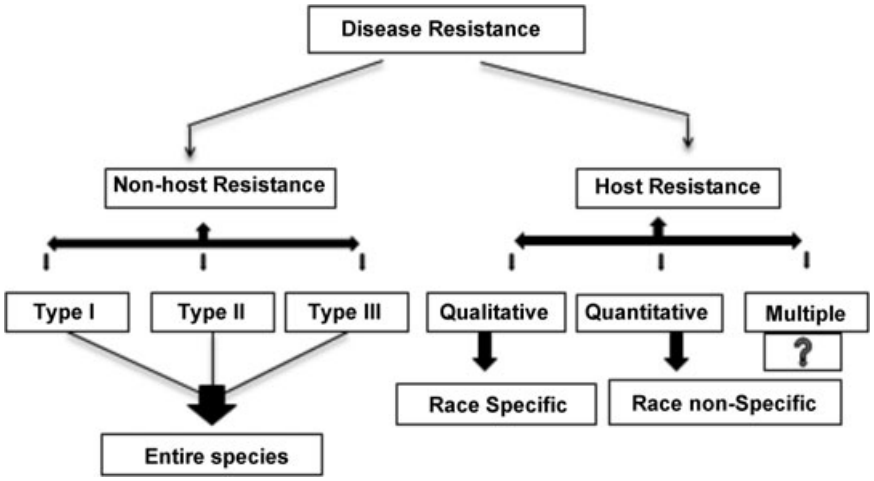

Figure 1. Basic concept of disease resistance.

Qualitative = Qualitative disease resistance (mostly control by single major gene); Quantitative = Quantitative disease resistance (several to many minor genes); Multiple = Multiple disease resistance (defense of plants against several diseases).

Cultivated species along with their wild relatives represent imperative genetic sources of potential resistance to plant pathogens (Flor 1971). Maize shows an astonishing amount of phenotypic diversity: plant height can vary from 0.5 to 5 meters at maturity; flowering dates fluctuate from 2 to 11 months after planting; the ear and kernels differ in color, length, size, shape, etc. (Sprague et al. 1988; Yan et al. 2011). For nearly every trait of economic or agronomic importance, there are measurable phenotypic differences within the global maize germplasm pool (Yan et al. 2011). The extent of variation and severity of diseases differs from mild lesions to complete destruction of the plants by different pathogens. The number, length and width of lesion on each leaf are different for the same plant. The lesions on the leaf can coalesce to cover the whole leaf and destroy the photosynthetically active area of the plant, starving the plant to death. To overcome the problem of finding exact genes for a specific disease, accurate phenotyping is essential. For this purpose, number of lesions per leaf, length of lesion and width of lesion should be measured along with multiple times scoring for calculation of area under disease progress curve. Along with phenotypic variation maize is a particularly diverse crop at all levels of resolution because large insertion and deletion are common and also contain tandem repeat clusters, transposons and abundant retroelements (Rafalski and Anaiev 2009). It has been assessed that throughout the maize genomic sequence, polymorphism exists between two diverse lines every 44 bp (Gore et al. 2009), and that the divergence between two maize inbred lines is even greater than between human and chimpanzees, which diverged as independent species 3.5 million years ago (Buckler and Stevens 2005). Plant diseases can be minimized by the reduction of the pathogen's inoculum, inhibition of its virulence mechanisms, and most importantly the promotion of genetic diversity in the crop.

It has been mentioned that genes involved in defense against pathogens and pests, evolved rapidly and exhibit frequently high allelic diversity (Rose et al. 2004; Tiffin et al. 2004; Rafalski and Anaiev 2009). Once the genes of economic importance have been identified and mapped, it will become easy for the plant breeders to develop resistant varieties and hybrids in the shortest possible time to combat the invading pathogen's races. The key molecular phenomenon involved in the evolution of maize genetic diversity was summarized (**Table 6**, Rafalski and Anaiev 2009).

Impact of Joint Linkage and Genome-wide Association Mapping in Elevating Disease Resistance

Association analysis is a powerful tool to identify QTL and has the capability of identifying a single polymorphism within a gene that is responsible for the phenotypic variation of a specific trait. The first association study of a quantitative trait based on candidate gene approach was performed in maize for flowering time and the *dwarf8* (*d8*) gene (Thornsberry et al. 2001). Genome-wide association studies (GWAS) have emerged as an influential tactic for detecting genes underlying complex diseases at an exceptional rate. In contrast to linkage mapping, association mapping can explore to the greatest extent recombination events and mutations in a given population with a higher resolution. Furthermore, GWAS can explore maximum polymorphism in the whole genome of maize that is the root cause of genetic diversity and once these points are

Table 6. Factors contributing genome diversity in maize

1) Genome or large genome segment duplications followed by structural and functional diversification.
2) Gene duplications, followed by diversification of DNA sequence and gene function.
3) Mutational processes including those associated with recombination and DNA replication, including gene conversion events.
4) Insertion and loss of DNA transposons.
5) Insertion and partial loss of retroelements.
6) Capture and translocation of gene segments by specialized classes of transposons (Pack-Mules and Helitrons).
7) Expansion and contraction of simple sequence repeats (SSRs).
8) Expansion and contraction of tandemly repeated sequences.
9) Possible gene flow between maize and teosinte.

Rafalski and Anaiev, 2009

completely explored, the genotypes will be easily manipulated genetically and ideal phenotypes will be developed to overcome the global problem of diseases. The choice of germplasm for association mapping, composed of elite inbred lines, diverse inbred lines or land races, is the vital concern for success of association analysis (Flint-Garcia et al. 2003; Breseghello and Sorrells 2006; Yu and Buckler 2006; Zhu et al. 2008; Yang et al. 2010). The best association mapping panel should harbor as much genetic diversity as possible and be used to resolve complex genetic traits (Yang et al. 2010). Recently, Kump et al. (2011) and Poland et al. (2011) have observed 32 and 29 QTLs for the two most prevailing diseases in maize in the world, southern corn leaf blight and northern corn leaf blight, respectively. These scientists have divulged a large number of loci having small additive effect that are involved in controlling the outsized phenotypic variation for disease resistance in maize crop. For northern leaf blight among the 29 QTLs, 3 QTL alleles had an estimated effect larger than ±5% while for southern corn leaf blight, the mentioned QTLs jointly explained 80% of the phenotypic and 93% of the genotypic variation. During my personal communication with Edward S Buckler about the uses and benefits of association mapping and its field implementation for development of diseases resistance varieties/hybrids, or transformation of all the known genes in nested association mapping (NAM) panel, he suggested the use of association mapping and genomic selection in the near future for resolving the problem of disease resistance. Yan et al. (2011) reviewed the current progress and strengths of association mapping in maize and the requirements for its effective use in enhancing maize genetic improvement for all traits of interest.

Recently a new approach of combined linkage and association analyses for fine mapping genes has been mentioned by Li et al. (2011). They have presented a reliable, cost effective and comprehensive protocol of combined linkage and association mapping. This process has the advantage of current and historical recombination events for QTL cloning in those species with an available reference genome. Furthermore, the recommended four steps while using the combined strategy of linkage and association mapping is extremely simple, easy to follow and needs a short period of time for complete validation of target genes by different bioinformatics tools. Once the development of an appropriate population required for combined linkage and association mapping has been completed, several genes of economic importance can be fine mapped with this easy approach. Using the four steps approach (Li et al. 2011), major genes and QTLs can be easily identified for different diseases in maize and the gigantic global losses can be abridged by developing genetically resistant varieties harboring major genes against the concerned pathogens.

Molecular Breeding for Disease-resistant Maize: Process and Challenge

Losses from many of the key diseases have been reduced significantly due the effective use of conventional breeding activities, though a thorough understanding of the basis of resistance is often lacking. Population improvement activities over several cycles of selection, has significantly improved performance of the germplasm both for agronomic traits as well as quantitative resistance to maize diseases. Resistance to the foliar diseases including southern and northern leaf blights, gray leaf spot, *polysora* and common rust, and downy mildew are all diseases effectively controlled through conventional breeding, where under disease pressure the susceptible genotypes could be eliminated before recombining the germplasm. The diseases where less progress has been achieved are banded leaf and sheath blight, post flowering stalk rots and ear rots. Based on the problem of conventional breeding, the modern molecular breeding gives complete insight into the diseases and cost effective PCR based markers can be easily used for development of disease resistant varieties and hybrids. Molecular breeding offers an integrative summary of subjects from basic theories to their applications for crop improvement and comprises of molecular marker technology,

gene mapping, genetic transformation, quantitative genetics and breeding strategies. Several studies have been reported for identifying disease resistance QTLs and exploring the mechanism through molecular breeding i.e. SCMV (Sugarcane Mosaic Virus) resistance (Zhang et al. 2003), MDMV (Maize Dwarf Mosaic Virus) resistance (Liu et al. 2006), common smut resistance (Ding et al. 2008), head smut resistance (Li et al. 2008), *Fusarium moniliforme* ear rot resistance (Zhang et al. 2006), banded leaf and sheath blight (BLSB) resistance (Zhao et al. 2006). Prasanna et al. (2010) documented well several important and successful studies about several diseases and other related traits through molecular marker-assisted breeding techniques. All disease QTLs are not easy to identify and clone, so there are several reasons that prevent scientists from reaching this goal of putting molecular assisted selection into an effective breeding program. Many reasons can account for this including a limited capacity to identify small effect QTLs, large genotype × environment interactions, and not being able to fine map the resistance QTLs. High throughput genotyping platforms are currently available and when linked with precision phenotyping in the field, can provide the information needed to effectively use marker assisted selection in a breeding program for complex traits. Current genotyping costs are falling and will make this a method more adapted for use in breeding programs and also the use of double haploid production will speed up the breeding process. The ultimate goal will be the breeder ready marker for marker assisted selection to upgrade the economic value of maize and assure global food safety.

Perspectives

Disease resistance in maize is normally accomplished by screening germplasm for identification of resistant lines or accessions, and then utilizing a backcross breeding scheme to introgress resistance from the donor parent into an agronomically superior, adapted line or inbred. For this purpose the first thing to explore will be the complete genetic architecture of maize crop and to find out all the genes playing any minor or major role in the disease resistance phenomenon. To study the structure and function of whole genomes, with the advent of fast and relatively economical sequencing methods, scientists have been able to obtain the base sequence of complete genomes. The B73 is not a resistant line and we are lacking information about the genes and QTLs controlling disease resistance, as the reason why the exact identification of resistance QTLs is extremely challenging. To solve this problem more reference genomes will be required to exactly identify the disease QTLs and especially a BAC library of resistance source will be tremendously helpful. Wild maize is a good gene bank for the breeding community because of the high genetic diversity in maize crop. As mentioned earlier, maize is affected by more than 100 pathogens, so an entire genetic pool must be available which represents the entire genetic variability or diversity available in maize. It will require a reliable and trustworthy germplasm, which includes land races, cultivars, varieties, hybrids, wild species and relatives of maize crop from all centers of diversity, gene banks, gene sanctuaries, farmers' fields, markets and seed companies. Disease resistance is a complex phenomenon and different genes play different roles in helping out the plant against the pathogens. Different kinds of protein-protein interactions are involved at different growth stages. The interaction of different genes and their pathways must be revealed, to confirm the exact role of genes and then to summarize all the genes based on their product; if genes are hard to find for a specific pathogen, we must be able to supplement different kinds of protein at different stages to overcome the losses at threshold level. For this purpose, we must study the properties and activities of all the proteins that a plant makes during its lifespan, i.e. proteomics will be the most important step. We should find out different and accurate ways to use the information from genomics and proteomics, for exploring maximum information about the genetics and function of different proteins. The data set obtained for genomics will be very large therefore complete knowledge about bioinformatics will be required for mining the massive amount of genomic information for meaningful knowledge about the structure and expression of gene.

Crossing of adaptive maize with its progenitor (*Teosinte*) will give us the opportunity to find the origin and evolutionary mechanism followed either by artificial selection or natural evolution of desirable genes responsible for resistance. Furthermore, we should understand the allelic frequencies and spatial distribution of pathogen effector in wild ecosystems, to clarify the evolution of basal immune system and by what means we might organize this information more efficiently to control disease. Another approach will be to make the maize crop more diverse by inserting different genes from other species and make maize crop as non-host to the most important pathogens as we know that large insertion and deletion is relatively common in maize crop compared to other main cereals (Rafalski and Anaiev 2009). As the process of disease resistance is extremely complex and complicated so, the scientists from different disciplines must interact and share breeding material with each other for smooth progress in this field. The other aspect includes the development of high throughput precision phenotyping systems for disease identification, scoring, rating and area under disease progress curve (AUDPC) with complete understanding of genotype by environment interaction. For this purpose we are developing a new technique for precise

phenotyping based on bar coding, which is more reliable than manual scoring and avoids the laborious work of punching data and checking. Along with this, precision phenotyping includes the use of an appropriate field design and statistical analysis, providing multiple optimal environmental conditions for disease development, having virulent pathogens, and the capacity to record the most appropriate phenotypic traits associated with resistance at the optimum time. We recognized precision phenotyping is a limitation frequently encountered in working with complex biotic and abiotic stress traits in maize, and globally there must be activities to improve phenotyping within the global maize community. Furthermore, we encourage the development of publicly available computational tools and proper statistical design tailored to study all the types at the same time with a perfect model and make the process available at the fingertips for scientists, breeders, students and especially the farming community.

However, numerous emerging questions need to be answered. Do the genes responsible for qualitative or quantitative resistance behave in the similar fashion like non-host resistance, and is there any difference in the regulation mechanism of the genes showing complete resistance in the host and genes responsible for non-host resistance? What will be the foremost intimidations in disease resistance over the imminent few decades? Will we be able to explore the complete number of genes through advanced molecular techniques, responsible for disease resistance in maize crop; if so, then what will be our strategy to combat the newly evolved pathogen at that time? How we will stop the survival of the fittest pathogen as the pathogens will be under tremendous pressure of survival and how much will it influence the human race? Which kind of characteristic will be required in the newly developed cultivars to cope with a super pathogen race? To what extent will the modern techniques of molecular biology be ready to lend a hand in the production of new varieties having idealistic traits?

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