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Broad-spectrum and durability: understanding of quantitative disease resistance

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Although quantitative resistance loci provide partial and durable resistance to a range of pathogen species in different crops, the molecular mechanism of quantitative disease resistance has remained largely unknown. Recent advances in characterization of the genes contributing to quantitative disease resistance and plant–pathogen interactions at the molecular level provide clues to the molecular bases of broad-spectrum resistance and durable resistance. This emerging knowledge will help in identifying genes involved in quantitative broad-spectrum resistance and durable resistance leading to formulation of efficient ways for using these genetic resources for crop improvement. This knowledge is also turning quantitative resistance genes with minor effects into a productive resource for crop protection via biotechnological approaches.

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Introduction

Plant diseases are one of the major limiting factors in crop production. Broad-spectrum resistance and durable resistance to diseases are desirable for crop improvement. Broad-spectrum resistance (BSR) refers to resistance against two or more types of pathogen species or the majority of races of the same pathogen species [1]. Durable resistance (DR) refers to resistance that remains effective during its prolonged and widespread use in environments favorable to the pathogen or disease spread [2].

Plant disease resistance can be classified into two categories: qualitative resistance conferred by a single resistance (*R*) gene and quantitative resistance (QR) mediated by multiple genes or quantitative trait loci (QTLs) with each providing a partial increase in resistance. Other terms have also been used for these genetically distinguishable resistances [3••,4••]. Compared with qualitative resistance, QR is characterized by a partial and durable effect of resistance that is generally pathogen species-nonspecific or race-nonspecific but pathogen species-specific [1,3••,5]. It is the most important or only form of resistance to necrotrophic pathogens and even some biotrophic pathogens (e.g., *Xanthomonas oryzae* pv. *oryzicola* causing rice bacterial streak) [3••,6]. Although QR has been widely used in breeding programs of different crops [7–12], the genes underlying QR are largely unknown. This review focuses on emerging clues underlying the molecular bases of quantitative BSR and DR on the basis of the characterized resistance QTLs and genes mediating partial resistance, and the current molecular model for plant–pathogen interaction. The perspective of applying this knowledge in crop improvement through biotechnology approaches is also highlighted.

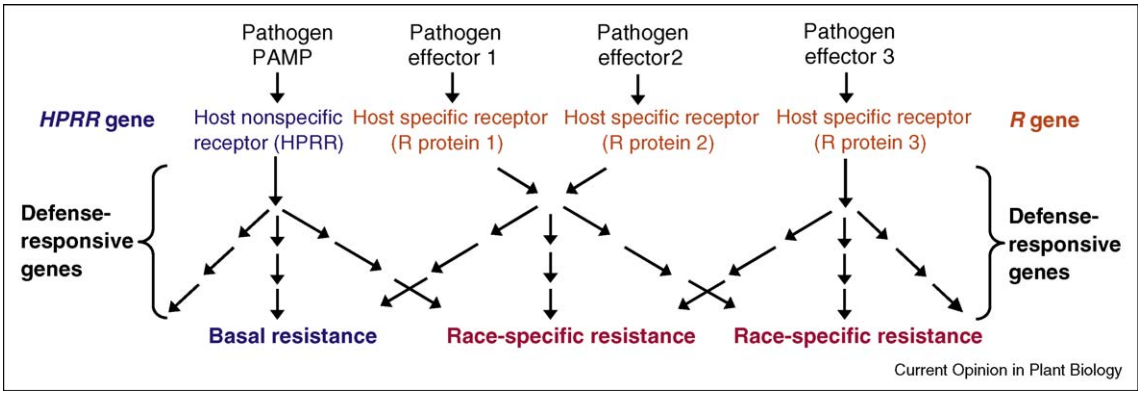
Two classes of genes in disease resistance

On the basis of the current model, plants respond to pathogen infection through two types of immune responses: basal resistance and race-specific resistance [13] (Figure 1). Plant–pathogen recognition initiates the signal transduction pathways that interact with each other to form a complex network leading to defense responses [14]. We can simply divide the genes involved in disease resistance into two classes, the receptor genes, which include *R* genes and host pattern recognition receptor (HPRR) genes, and defense-responsive or defense-related genes (Figure 1). The latter are characterized by responding to a pathogen attack via changing expression levels or posttranslationally modifying their encoding proteins [15,16]. There are large numbers of defense-responsive genes in a given species [1,15,17]. The encoding proteins of defense-responsive genes function either as activators or as suppressors in defense responses. The following evidence suggests that defense-responsive genes and HPRR-type genes are important resources for quantitative BSR and DR.

Genes contributing to quantitative BSR and DR

QTLs detected for a given disease always have variant effects in resistance. We group the QTLs that explain >10% of the phenotypic variation as major QTLs and those that explain <10% of the phenotypic variation as minor QTLs. Recently, three major QTLs against fungal pathogens were isolated by map-based cloning. Wheat

Figure 1



Cross-talks between basal and race-specific resistance pathways and between different race-specific resistance pathways. Basal resistance is initiated by the interaction between host pattern recognition receptors (HPRRs) and evolutionarily conserved pathogen-associated molecular patterns (PAMPs), which is a relative nonspecific defense response. Race-specific or gene-for-gene resistance is triggered by the direct or indirect interaction of host resistance (R) proteins and complementary pathogen effector molecules.

QTL *Lr34* confers BSR and DR to leaf rust, stripe rust, and powdery mildew [18••,19]. It encodes a protein resembling plasma-membrane ATP-binding cassette transporters. Wheat *Yr36* provides high temperature-dependent QR to diverse stripe rust races and encodes a kinase-START protein [20••,21]. Rice QTL *pi21* is a recessive gene conferring DR to blast disease, which encodes a loss-of-function mutation in a cytoplasmic proline-rich protein consisting of a putative heavy metal-binding domain and putative protein–protein interaction motifs [22••,23]. The characteristic of the three genes in quantitative BSR and/or DR and the features of their encoding proteins suggest that they may belong to the defense-responsive gene class or HPRR gene group.

Most of the identified resistance QTLs have small effect on disease resistance. Map-based cloning is not the best choice to isolate minor resistance QTLs, because of the difficulty in fine mapping. Four defense-responsive genes contributing to four minor resistance QTLs against rice diseases have been identified by a strategy of validation and functional analysis of the QTL [4••]. *OsWRKY13* encodes a transcription regulator, which positively regulates rice resistance to bacterial blight and blast diseases. *OsDR8* encodes a thiamine synthesis-related protein that positively regulates rice resistance to bacterial blight and blast. *GH3-8* encodes an auxin-amido-synthetase, which regulates rice resistance to bacterial blight by inhibiting auxin activity. *OsMPK6* encodes a mitogen-activated protein kinase. Interestingly, it positively regulates resistance to bacterial streak and blast, but regulates both negatively and positively resistance to bacterial blight [4••,6]. A major rice blast resistance QTL was also characterized using a similar approach [24••,25]. This QTL is associated with several physically clustered defense-responsive members of the germin-like protein (*OsGLP*) gene family, which function collectively in resistance to both blast and sheath blight diseases.

Although only a few genes underlying resistance QTLs have been characterized, several HPRR-type genes and a number of defense-responsive genes have also been suggested as contributing to QR [3••,26] and some of them mediate BSR. For instance, a mutation of Arabidopsis *LysM RLK1*, encoding a HPRR-type protein, partially reduced resistance to necrotrophic and biotrophic fungi [27]. A pepper defense-responsive gene *CaAMP1*, encoding an antimicrobial protein, mediates BSR against biotrophic, hemibiotrophic, and necrotrophic pathogens [28]. The potato *StAOS2*, encoding an enzyme for jasmonic acid synthesis, quantitatively mediates resistance against oomycete and bacterial pathogens [29]. Only a few functional studies support that QR may also be contributed by *R* genes that have residual effects against virulent pathogens or defeated *R* genes [3••], although *R* gene loci frequently colocalize with resistance QTLs [1,30].

The alleles contributing to resistant loci can be grouped by comparison with their corresponding susceptible alleles. The first group includes those whose functions are owing to their encoding proteins being different from those of susceptible alleles, like *Lr34*, *pi21*, *OsWRKY13*, and *StAOS2* [4••,18••,22••,29,31]. The second group includes ones that are lost in the susceptible plants, such as the case of *Yr36* [20••]. The third group of resistant alleles results from expressional or posttranslational difference during host–pathogen interaction. The resistant allele may be more rapidly activated (for a positive defense regulator) or suppressed (for a negative defense regulator). The differential expression

or posttranslational modification of resistant alleles depends on upstream defense signaling. The examples include *CaAMP1*, *OsNPR1/NH1*, *MPK3*, and *MPK6*, which were more efficiently induced or activated in resistance reaction than in susceptible reaction [28,32,33]. Suppression or activation of *CaAMP1* and *OsNPR1/NH1* quantitatively increased susceptibility or resistance to virulent pathogens [28,32]. Arabidopsis MPK3 and MPK6 kinase activities associated disease resistance depend on activation of HPRR receptor FLS2 [33]. The knowledge of grouping resistant alleles will be helpful for choosing an appropriate approach to use these resources in breeding programs.

Molecular bases of BSR and DR

The above listed genes are confirmed or suggested to function in either basal or *R*-gene mediated resistance [4••,20••,24••,27,33,34]. Defense signaling pathways leading to basal and race-specific resistance often cross-talk [14,35] (Figure 1). On the basis of the current model of host–pathogen interaction and the features of the characterized genes contributing to QR, we may propose models to elucidate the molecular mechanisms of quantitative BSR and DR. Race-specific resistance QTLs have been identified in different crops [36–40]. Thus, the quantitative BSR of a given plant may be enhanced by the cooperation of multiple genes functioning in pathways leading to resistance against different pathogen species or different races of the same species. The above evidence also suggests that quantitative BSR can be conferred by a single gene. In this case, the gene may function in a basal-resistance pathway, in overlapping pathways between different race-specific resistances, or in the cross-talk point of different defense pathways (Figure 1). All the three situations can include both pathogen species-nonspecific resistance and race-nonspecific resistance. This one-gene model is supported by *CaAMP1*, *OsWRKY13*, and *OsDR8*. *CaAMP1* may function in basal resistance [28]. *OsWRKY13* and *OsDR8* function in resistances mediated by two different *R* genes [4••,6].

Quantitative DR may be controlled by one or multiple genes, whose encoding proteins do not interact with rapidly evolving pathogen factors that play essential roles in pathogenicity on plants [41]. On the basis of this hypothesis, we may predict, to certain extent, which gene in QR may mediate DR. Cytochrome P450 enzyme StAOS2 is essential for biosynthesis of the defense signaling molecule jasmonic acid [29]. Thus *StAOS2* fits this hypothesis as a candidate for quantitative DR gene to pathogens whose infection can be suppressed by host jasmonate-dependent pathway. Transcription regulator OsWRKY13 functions by directly or indirectly regulating the expression of host defense-responsive genes [4••,6]. Thus *OsWRKY13* may be a quantitative DR gene. However, whether the known DR QTLs, *Lr34*, *Yr36*, and *pi21*, also fit the above hypothesis would await the characterization of the biochemical functions of their encoding proteins. It may also be likely that a DR gene may not be durable infinitively. A new effector may become adapted to the resistance in evolutionary time [41,42]. The polygenic control underlying QR is proposed to make it more difficult for pathogens to overcome these multiple resistances [3••,43,44], which constitute more effective DR.

Approaches for breeding BSR and DR crops

Marker-assisted selection (MAS) has been widely applied in breeding programs for targeted transferring and pyramiding resistance loci in different crops [8–12,45,46]. MAS will provide an important approach for breeding BSR and DR corps, although in the past QR has been used in crop improvement by conventional breeding without the knowledge of resistance loci [7]. With the knowledge of genes underlying resistance loci, MAS can improve the efficiency by using gene-specific or closely linked markers to avoid bringing undesired traits into an improved cultivar owing to linkage drag [22••,47]. MAS may be more effective for the selection of the first and second groups of resistant loci mentioned above, whose functions depend on the specificities of encoding proteins. MAS may be less effective for selection of the third group of resistant loci, whose functions are initiated by upstream signaling thus depend on the genetic background. This prediction is supported by the reports that MAS is less effective for selection of some quantitative traits other than disease resistance [48,49].

Characterized QR genes can also be used more efficiently for crop improvement with strategies involving transgenics. *Lr34* confers only adult plant resistance, which appears to be associated with its increased expression in the adult stage [18••]. *Yr36*-mediated high temperature-dependent resistance is due to high temperature induction of *Yr36* [20••], suggesting that the function of *Yr36* may also be associated with its expression level. Constitutive expressing *Xa3/Xa26* and *Xa21* can change adult-stage resistance to whole-growth-stage resistance [6]. It will be interesting to see whether *Lr34* and *Yr36* can confer a whole-growth-stage or temperature-independent resistance if regulated using a pathogen-induced or tissue-specific strong promoter. The use of a recessive gene in hybrid crop production is not convenient. QTL *pi21* is a recessive gene. However, it can be used in breeding programs by suppressing its dominant (susceptible) allele by means of an RNA interference strategy [22••]. There are other advantages of improving crop resistance by manipulating the expression of QR genes using appropriate promoters. This approach may enable the utilization of QR genes whose functions depend on upstream signaling and may enhance the effect of a single gene in QR, which has small effect driven by the native promoter, for improving BSR or DR [4••].

Conclusions

Defense-responsive genes and HPRR-type genes are important resources for quantitative BSR and DR. The natures of quantitative BSR and DR may not be as complex as previously speculated. A single locus can confer BSR or DR or both. Current biotechnological approaches offer various opportunities to efficiently use QR loci in crop improvement. MAS of resistance loci will provide an important approach for improving crop resistance. By manipulating the expression of the genes contributing to QR, we may use a minor resistance allele for improving BSR and DR of crops, and transfer a developmentally controlled or environment factor-dependent resistance allele to a whole-growth-stage or environment factor-independent resistance gene in crop breeding. Furthermore, the time consuming work routinely practiced by most researchers and breeders will not be the only way to identify a DR gene, as we may explore quantitative DR genes by their positions in defense signaling pathways. The emerging knowledge of BSR and DR is turning QR loci into a more productive resource for protecting crop production.

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