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Review

Mechanisms of quantitative disease resistance in plants

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ABSTRACT

Quantitative disease resistance (QDR) causes the reduction, but not absence, of disease, and is a major type of disease resistance for many crop species. QDR results in a continuous distribution of disease scores across a segregating population, and is typically due to many genes with small effects. It may also be a source of durable resistance. The past decade has seen significant progress in cloning genes underlying QDR. In this review, we focus on these recently cloned genes and identify new themes of QDR emerging from these studies.

Contents

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1. Introduction

1.1. What is quantitative disease resistance?

Plant disease resistance responses are complex and multilayered (reviewed in Refs. [1–5]). Historically, plant immunity has been divided broadly into two categories: incomplete resistance provided by quantitative disease resistance (QDR) genes and complete resistance mediated by resistance (R) proteins [1–3]. More recently, recognition of the role of microbial elicitors and their host receptors led to the idea of 'microbial triggered immunity' (MTI). The zig-zag model of plant immunity accounts for the latter two categories, incorporating general pathogen elicitors, their host receptors, and R-protein mediated resistance into a single model [4]. In this model, one of the first lines of plant defense is recognition of microbial elicitors such as flagellin and chitin. These Microbe Associated Molecular Patterns (MAMPs) are recognized by pattern recognition receptors (PRR) at the cell surface that initiate a signaling cascade leading to generally weak defense responses and MTI. Some pathogens can overcome this level of resistance by secreting effector proteins that interfere with host metabolism and act to promote pathogen virulence. Plants have evolved to recognize and defend themselves against such effectors; this immunity is known as Effector-Triggered Immunity, or ETI, and typically leads to full resistance with no disease symptoms. Plant proteins that recognize effectors are termed R proteins, and are frequently in a class of proteins known as Nucleotide Binding Site Leucine Rich Receptors (NBS-LRRs).

Although the zig-zag model has been extremely useful for thinking about two seemingly separate parts of plant immunity – MTI and ETI – the model is limited largely to describing interactions between hosts and biotrophic pathogens. It is less suitable for understanding host-necrotrophic interactions, and does not (by design) account for the complexities of host-pathogen interactions that lead to a wide range of host immune responses [5,6]. The Invasion Model, which describes plant immunity as a surveillance system that continually evolves to detect microbial invasion, may be more useful for describing the nuanced layers of plant defense [6]. In this model, plants recognize invasion patterns (IP) that are derived from microbes (such as MAMPS or effectors) or endogenous elicitors that result from infection, such as Damage Associated Molecular Patterns or DAMPs. IPs are recognized by IP—triggered receptors (IPTRs). MTI and ETI are viewed less as strictly contrasting responses and instead as continuous immune outputs resulting from variation between different IPs and IPTRs.

Such a model accounts for Quantitative Disease Resistance (QDR). QDR has been traditionally recognized but is less well understood than MTI or ETI [6–8]. QDR refers to host plant resistance that leads to a reduction in disease, but not the absence of disease [1,2,7]. As a quantitative trait, QDR is controlled by multiple genes that can interact with the environment and with each other. Recent work has shown however, that the cumulative effects of pyramiding many QDR loci can result in high levels of resistance [8–11]. Phenotypically, QDR exhibits a continuous distribution of resistance values that do not fit Mendelian segregation ratios [1,7]. This is in contrast to a qualitative trait, in which the variation is typically due to differences at one locus, and the effects of different alleles at the locus are large relative to the environment. An important point is that a resistance phenotype can vary within a population for reasons not due to genes controlling QDR [7]. For example, varying levels of resistance within a population could result from the effects of one gene with low heritability or low penetrance [7]. Alternatively, a host population may contain a series of *R* genes that each provides complete resistance against one strain of a pathogen. However, when infected with a highly complex pathogen population, such a host population may exhibit a continuous distribution of resistance [7]. Genetic analyses must be used to determine if observed variable levels of resistance are due to QDR. Additionally, care must be taken with the phenotyping approach used for such analyses, as this may affect the outcome. For example, the use of a 5-point scale for disease severity could potentially make a continuous distribution of disease severity appear to be discrete and may result in errant conclusions.

Recent work suggests that QDR can result from quantitative variation in the components of either MTI or ETI [1,2], as well as through completely different mechanisms ([1,2] and references in this review). This fits within the framework of the Invasion Model, and supports the idea of plant immunity as a continuum with quantitative variation in both pathogenic elicitors and host responses leading to a spectrum from disease to resistance [6]. Consistent with this, tolerance – the host's ability to withstand high pathogen load with limited disease symptoms or fitness cost – appears to be a component of QDR in several pathosystems, including Arabidopsis – *R. solanacearum* [12], and Arabidopsis – *Pseudomonas syringae* [13].

The potential mechanisms underlying QDR have frequently been deliberated [1–3,7,14]. Recent years have seen a significant increase in the genes cloned that contribute to QDR. In this review we focus on these genes and discuss new insights into the mechanisms of QDR gained from their functions. Although QDR may result from the pathogen's ability to suppress immunity ('effector triggered susceptibility', [15]), our focus here is not on susceptibility alleles, but on plant genes that lead to QDR. For reviews and papers that highlight more historical concepts of QDR, the reader is directed to [1,7,14,16].

1.2. How important is QDR?

If ETI offers complete resistance, why is there interest in the genes underlying QDR, which provides only a decrease in disease? First, although ETI produces complete resistance, since this resistance is due primarily to one R protein, pathogen effector proteins can evolve to overcome the R protein and ETI-mediated resistance [17]. In contrast, because multiple genes underlie QDR, the evolutionary pressure on pathogens is significantly decreased. QDR may therefore be a good source of durable resistance (resistance that remains effective over a long period of time even with wide crop cultivation). Indeed, several genes underlying QDR have been used in breeding programs for more than half a century with no signs of increased pathogen virulence [18,19].

The second reason for interest in QDR is that ETI is most effective against biotrophic pathogens. ETI frequently results in a cell death known as the hypersensitive response (HR). The HR limits biotrophic pathogen growth and colonization and typically leads to full resistance against these pathogens. However, necrotrophic pathogens, which feed on dead tissues, exploit this cell death to increase their own virulence. In contrast to ETI, QDR provides an effective means of control for both biotrophic and necrotrophic pathogens.

Another reason for interest in QDR is that many QDR loci are effective against multiple races of a given pathogen, providing broad-spectrum resistance, or are effective against multiple pathogens [18]. However, QDR loci involved in race or isolate-specific resistance are becoming increasingly common [1,2]. Such loci have been identified for resistance to tomato bacterial wilt (*Ralstonia solanacearum*) [20]; vascular wilt in melon (*Fusarium oxysporum*) [21], stripe rust in wheat (*Puccinia striiformis* f. sp. *tritici*) [22], powdery mildew in grape (*Erysiphe necator*) [23], and barley leaf rust (*Puccinia hordei*) [24], among others. Isolate-specific QTL may represent evidence for the minor-gene-for-minor-gene model of QDR, originally conceptualized by[16].