Innate Immunity of Plants, Animals and Humans

Bearbeitet von
Holger Heine

ISBN 978 3 540 73929 6
Format (B x L): 15,5 x 23,5 cm
Gewicht: 549 g

Weitere Fachgebiete > Chemie, Biowissenschaften, Agrarwissenschaften > Biowissenschaften allgemein > Evolutionsbiologie

Zu Inhaltsverzeichnis

schnell und portofrei erhältlich bei

beck-shop.de
DIE FACHBUCHHANDLUNG

Die Online-Fachbuchhandlung beck-shop.de ist spezialisiert auf Fachbücher, insbesondere Recht, Steuern und Wirtschaft. Im Sortiment finden Sie alle Medien (Bücher, Zeitschriften, CDs, eBooks, etc.) aller Verlage. Ergänzt wird das Programm durch Services wie Neuerscheinungsdienst oder Zusammenstellungen von Büchern zu Sonderpreisen. Der Shop führt mehr als 8 Millionen Produkte.
Chapter 1
Evolution of Resistance Genes in Plants

Shunyuan Xiao(✉), Wenming Wang, and Xiaohua Yang

Abstract  Potential pathogens deliver effector proteins into plant cells to suppress microbe-associated molecular pattern (MAMP)-triggered immunity in plants, resulting in host–pathogen coevolution. To counter pathogen suppression, plants evolved disease resistance (R) proteins to detect the presence of the pathogen effectors and trigger R-dependent defenses. Most isolated R genes encode proteins possessing a leucine-rich-repeat (LRR) domain, of which the majority also contain a nucleotide-binding site (NBS) domain. There is structural similarity and/or domain homology between plant R proteins and animal immunity proteins, suggesting a common origin or convergent evolution of the defense proteins. Two basic strategies have evolved for an R protein to recognize a pathogen effector (then called avirulence factor; Avr): direct physical interaction and indirect interaction via association with other host proteins targeted by the Avr factor. Direct R-Avr recognition leads to high genetic diversity at paired R and Avr loci due to diversifying selection, whereas indirect recognition leads to simple and stable polymorphism at the R and Avr loci due to balancing selection. Based on these two patterns of R-Avr coevolution, investigation of the sequence features at paired R and Avr may help infer the
R-Avr interaction mechanisms, assess the role and strength of natural selection at the molecular level in host–pathogen interactions and predict the durability of \( R \) gene-triggered resistance.

**Abbreviations**  
\( R \), resistance gene; \( Avr \), avirulence gene; HR, hypersensitive response; MAMP, microbe associated molecular patterns; MTI, MAMP-triggered immunity; ETI, Effector-triggered immunity; TIR, toll and interleukin receptor; NBS, nucleotide binding site; LRR, leucine rich repeat; RLP, receptor-like protein; RLK, receptor-like kinase.

1 Evolution of the Plant \( R \) Gene System

Plant innate immunity consists of preformed physical and chemical barriers (such as leaf hairs, rigid cell walls, pre-existing antimicrobial compounds) and induced defenses. Should an invading microbe successfully breach the preformed barriers, it may be recognized by the plant, resulting in the activation of cellular defense responses that stop or restrict further development of the invader (Nurnberger et al. 2004). Apart from virus-induced RNA silencing, an ancient, evolutionary conserved antiviral defense mechanism in both plants and animals (which is not discussed in this chapter), two evolutionarily interrelated mechanisms have evolved in plants for detection of the invading microbes. First, plants are able to recognize some conserved microbe-derived molecules which are collectively described as microbe-associated molecular pattern (MAMP) by cell-surface receptors and trigger immune response (Gomez-Gomez and Boller 2000; Zipfel et al. 2006). Evidence is accumulating that this so-called MAMP-triggered immunity (MTI) is evolutionarily ancient and may be a general feature of plant resistance against a broad-spectrum of potential pathogens (Nurnberger et al. 2004; He et al. 2006). This type of resistance occurs at or above the species level, and is often referred to as non-host resistance. It can be envisaged that microbes that successfully breached constitutive defensive barriers of plants but were restricted by MTI gradually evolved strategies to target and sabotage the MTI. Increasing evidence indicates that successful microbes suppressed MTI by sending effector proteins into the plant cell to interfere with the host defense system, resulting in the breakdown of non-host resistance and the establishment of a host–pathogen interaction. The “defeated” host then faced selection pressure imposed by the successful pathogen to evolve novel defense mechanism to survive. This led to the evolution of the second recognition mechanism for which plants evolved disease resistance (\( R \)) proteins to specifically detect the presence of the pathogen effectors [called avirulence factors (\( Avr \)) once recognized by \( R \) proteins] and subsequently trigger a much stronger defense response to counter the suppression of MTI by the pathogen (Chisholm et al. 2006). Thus, \( R \) gene-dependent, pathogen-effector-triggered host immunity (ETI) most likely evolved on top of MTI to fortify the plant immune system. Recent publications strongly support this
Evolution of Resistance Genes in Plants

1 Evolution of Resistance Genes in Plants

Inference (Kim et al. 2005; He et al. 2006; Nomura et al. 2006). For example, He and colleagues recently found that HopM1, a conserved effector protein of *Pseudomonas syringae*, targets an immunity-associated protein, AtMIN7 in *Arabidopsis thaliana*. HopM1 mediates the destruction of AtMIN7 via the host proteasome (Nomura et al. 2006). Sheen and colleagues found that AvrPto and AvrPtoB, two effector proteins of the bacterial pathogen *P. syringae* suppress MTI at an early step upstream of MAPK signaling (He et al. 2006). Both AvrPto and AvrPtoB are recognized by the plant R protein Pto in tomato, thereby triggering Pto-dependent resistance (Kim et al. 2002).

Evolution of the ETI system in plants marks a higher level of plant–pathogen coevolution in which the major players are plant *R* and pathogen *Avr* genes. Unlike MTI, which is expressed in all plants of a given species, ETI is often expressed in some but not all genotypes within a plant species. This correlates to the phenomenon that there are often two likely outcomes from a given host–pathogen interaction: (a) compatible interaction in which the pathogen is able to suppress host defenses and colonize the plant; (b) incompatible interaction in which the pathogen is detected by the plant containing an *R* gene and the plant is resistant. Therefore, genetically defined *R* genes are polymorphic determinants of host resistance against specific pathogens.

MTI in plants resembles the innate immune system of animals in that structurally similar cell-surface receptors are deployed to recognize MAMPs such as flagellin and lipopolysaccharides and the induction of host defenses involves MAPK signaling cascades (Nürnberger et al. 2004). Thus, MTI seems to be a highly conserved defense mechanism evolved in both plants and animals. Interestingly, so far there is no clear evidence to indicate the existence of ETI in animals. Therefore, it appears that the evolution of an elaborative plant ETI system in which a large array of *R* proteins function as receptors to recognize pathogen-specific effectors constitutes an important distinction between the plant and animal innate immune systems (Ausubel 2005). This probably reflects the consequence of adaptive evolution: plants are sessile, lack a circulating system and live relatively longer than most invertebrate animals; thus evolution of a greater capacity in every single cell to respond and mount effective defenses against numerous microbial invaders seems to be a logical choice for plants.

In the following sections, we focus our review on the current understanding of evolution and maintenance of plant *R* genes within the context of concomitant evolution of pathogen *Avr* genes that interact with *R* genes. For detailed molecular mechanisms of *R* gene evolution, we strongly recommend several excellent earlier review articles (Michelmore and Meyers 1998; Bergelson et al. 2001; Holub 2001; Meyers et al. 2005).

2 Conservation and Diversity of Plant *R* Genes

Since the isolation of the first plant *R* gene, *Hm1* in maize in 1992 (Johal and Briggs 1992), over 60 plant *R* genes controlling resistance against pathogens ranging from viruses, bacteria, fungi to nematodes have been isolated from different plant species
Most isolated R genes seem to activate common or overlapping sets of defense programs in local areas infected by pathogens. Those defense responses include transcriptional induction of pathogenesis-related (PR) genes, production of reactive oxygen species, fortification of the cell wall, synthesis of antimicrobial compounds and, in many cases, a hypersensitive response (HR) which is a form of plant programmed cell death analogous to animal apoptosis (Hammond-Kosack and Jones 1997; Dangl and Jones 2001; Nurnberger et al. 2004). The primary local resistance triggered by R genes may also lead to activation of a secondary defense termed systemic acquired resistance in the uninfected tissues, which is a more long-lasting immune response throughout the whole plant against a broad range of pathogens (Durrant and Dong 2004).

Based on features of the deduced domain structures and/or biochemical functions, R proteins can be divided into three classes (Table 1). The largest class contains a nucleotide-binding site (NBS) and leucine-rich-repeat (LRR) motifs (Hammond-Kosack and Jones 1997; Dangl and Jones 2001). These R proteins confer resistance to various pathogens and can be further subdivided into two groups, based on their N-terminal features. The first group contain an N-terminal domain resembling the cytoplasmic signaling domain of the Drosophila toll and human interleukin-1 receptors (TIR) and are called TIR-NBS-LRRs (Whitham et al. 1994; Lawrence et al. 1995). The second group contain (in most cases) a coiled-coil (CC) domain and thus often are referred to as CC-NBS-LRRs (Bent et al. 1994; Grant et al. 1995). An exceptional case in the TIR-NBS-LRR group is the Arabidopsis RRS1-R protein that has a WRKY domain attached to the LRR at the C-terminus (Deslandes et al. 2002). The WRKY domain is found in a group of transcription factors implicated in the signal transduction of R genes (Eulgem 2005). The structural feature of RRS1-R implies a direct link between Avr-recognition and the transcriptional activation of defense genes (Deslandes et al. 2003).

The second class of R proteins comprise cell surface receptor-like transmembrane proteins (RLP) and receptor-like kinases (RLK) (Table 1). The common feature of these proteins is that they possess an extracellular LRR (eLRR) domain. Representatives of RLP R proteins are tomato Cf proteins conferring resistance to the tomato fungal pathogen Cladosporium fulvum (Jones et al. 1994; Hammond-Kosack and Jones 1997) and Arabidopsis RPP27 conferring resistance to the oomycete Hyaloperonospora parasitica (Tor et al. 2004). RLK R proteins are represented by rice Xa21 and Xa26, both of which confer resistance to multiple strains of Xanthomonas oryzae pv. oryzae (Song et al. 1995; Sun et al. 2004).

The remaining R genes encode proteins that either resemble the overall structure or a domain of the above two classes with some degree of structural variation, or have a novel protein structure that does not show significant homology to any other R proteins (Table 1). Therefore, they are atypical R genes in comparison with the LRR-encoding R genes. For example, tomato Pto and Arabidopsis PBS1 encode members of a conserved protein kinase family (Martin et al. 1993; Swiderski and Innes 2001) that resemble the cytoplasmic protein kinase domain of RLK R proteins. The broad-spectrum powdery mildew R gene RPW8 from Arabidopsis encodes a small protein containing an N-terminal transmembrane domain and a CC
<table>
<thead>
<tr>
<th>R protein class</th>
<th>Schematic domain structure</th>
<th>Predicted function</th>
<th>Examples</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Receptor</td>
<td>RPM1, RPS2</td>
<td>Bent et al. (1994), Grant et al. (1995)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Receptor</td>
<td>RRS1-R</td>
<td>Deslands et al. (2002)</td>
</tr>
<tr>
<td>eLRR</td>
<td>![eLRR Diagram]</td>
<td>Receptor</td>
<td>Cf9; RPP27</td>
<td>Jones et al. (1994), Tor et al. (2004)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Receptor</td>
<td>Xa21, Xa26</td>
<td>Song et al. (1995), Sun et al. (2004)</td>
</tr>
<tr>
<td>Atypical</td>
<td>![Atypical Diagram]</td>
<td>Host target?</td>
<td>Pto, PBS1</td>
<td>Martin et al. (1993), Swiderski and Innes (2001)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>?</td>
<td>RPW8</td>
<td>Xiao et al. (2001)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>?</td>
<td>Xa27</td>
<td>Gu et al. (2005)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fertility</td>
<td>Xa13</td>
<td>Chu et al. (2006)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Negative regulator of PCD</td>
<td>MLO</td>
<td>Buschges et al. (1997)</td>
</tr>
</tbody>
</table>