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Mechanisms of aphid adaptation to host plant resistance

Ashley D Yates[1] and Andy Michel[1,2]

Host-plant resistant (HPR) crops can play a major role in preventing insect damage, but their durability is limited due to insect adaptation. Research in basal plant resistance provides a framework to investigate adaptation against HPR. Resistance and adaptation are predicted to follow the gene-for-gene and zigzag models of plant defense. These models also highlight the importance of insect effectors, which are small molecules that modulate host plant defense signaling. We highlight research in insect adaptation to plant resistance, and then draw parallels to virulence adaptation. We focus on virulent biotype evolution within the Aphididae, since this group has the highest number of described virulent biotypes. Understanding how virulence occurs will lead to more durable insect management strategies and enhance food production and security.

Addresses
[1] Center for Applied Plant Sciences, and The Ohio State Center for Soybean Research, USA
[2] Department of Entomology, Ohio Agricultural Research and Development Center, The Ohio State University, 1680 Madison Ave., Wooster, OH, USA

Corresponding author: Michel, Andy (michel.70@osu.edu)

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Insect-resistant plants and the adaptation problem

Plant resistance to insects enhances environmentally safe and profitable food production and ensures food security, whether the source of resistance is transgenic [1] or natural host plant resistance [2]. Host plant resistance (or HPR, see glossary) uses naturally occurring resistance that is traditionally bred into commercial varieties; to date >500 crop plants have insect resistance, with a value >US $2 Billion [2]. Despite these benefits, HPR durability is threatened by insect adaptation to overcome plant resistance.

Insects across several taxa have evolved the ability to survive and reproduce on crop plants with HPR (termed *virulent biotypes*, see glossary) [3,4]. Plant resistance durability could be improved with a more thorough understanding of how insects adapt to resistant crops, which necessitates knowledge of plant–insect interactions. We briefly review recent research that support models of basal plant resistance to insects, and then draw parallels to specific HPR systems in which insects have evolved virulence. We highlight virulence among aphids, since virulent biotypes are highest in this taxa by far [4]. (*Note*: other important virulence-HPR systems have recently been reviewed, see [5••,6].) Our goal is to compare model systems in aphid-basal plant resistance with HPR-virulence systems so that we can identify knowledge gaps in aphid virulence evolution and further expand the use of HPR for agricultural production.

Plant–insect interaction models

The gene-for-gene and zigzag hypotheses describe genetic and molecular interactions between plants and their pests (parasites, pathogens, nematodes and insects) [7–13]. In the gene-for-gene model, a pest or pathogen carries avirulence (*avr*, see glossary) genes that are recognized by specific plant resistance (R) genes (see glossary) to confer resistance in the plant. Resistance is lost when the *avr* gene is not recognized by the plant's cognate resistance gene [8]. Additional research indicates a more complex and diversified *avr* and *R* gene interaction, leading to the zigzag model [7]. The zigzag model also clarifies the type of molecule that plants recognize to mount a defense response: conserved molecules (i.e. microbial/pathogen/herbivore-associated molecular patterns, MAMPS/PAMPS/HAMPS), or 'effector' molecules (see glossary) that interact with R genes [7]. In the zigzag model, insect effectors can provide a potential path to adaptation (Figure 1).

Adaptation may occur in any step in the gene-for-gene or zigzag model. The plant recognizes conserved molecules deployed by insects during colonization via transmembrane pattern recognition receptors (PRR) and activates basal level PAMP-triggered immunity (PTI) [7,12]. To counteract PTI, an insect may secrete effector proteins, defined by their presence in saliva and ability to modulate plant defenses [10,12], which leads to effector-triggered susceptibility (ETS). However, a plant's R protein (deriving from the *R* gene) can recognize an insect effector and mount a counter-defense via effector-triggered immunity (ETI). Many *R* genes contain nucleotide-binding site and

Glossary

Host plant resistance (HPR): a plant cultivar with naturally occurring genetic traits that confer more resistance to pest damage than the susceptible genotype [2]. HPR is bred into many crops for protection against pests.

Biotype: an insect's phenotype when interacting with specific plant resistance genes. The term 'biotype' does not have a genetic basis, so it is possible that insects within a biotype display the same phenotype via different genetic mechanisms.

Virulent: insects that overcome HPR and survive on resistant plants.

Avirulence gene: a gene product that is recognized by an R gene product in specific plant genotypes, leading to effector-triggered immunity (ETI) [7]. The same avirulence gene can go undetected by R genes of other plant genotypes, resulting in effector-triggered susceptibility (ETS). Because 'avirulence gene' is specific to both the insect and plant genotypes, the term 'effector' may be used instead [12].

R gene: a plant resistance gene that can recognize an avirulence gene/effector, resulting in ETI [7].

Effector: a pathogen or insect molecule that modifies the host cell, triggering or suppressing plant defenses [12].

NBS-LRR: many R genes encode NBS-LRR proteins, which are characterized by nucleotide-binding sites and leucine-rich repeats [7]. NBS-LRR proteins recognize pests, including insects [14].

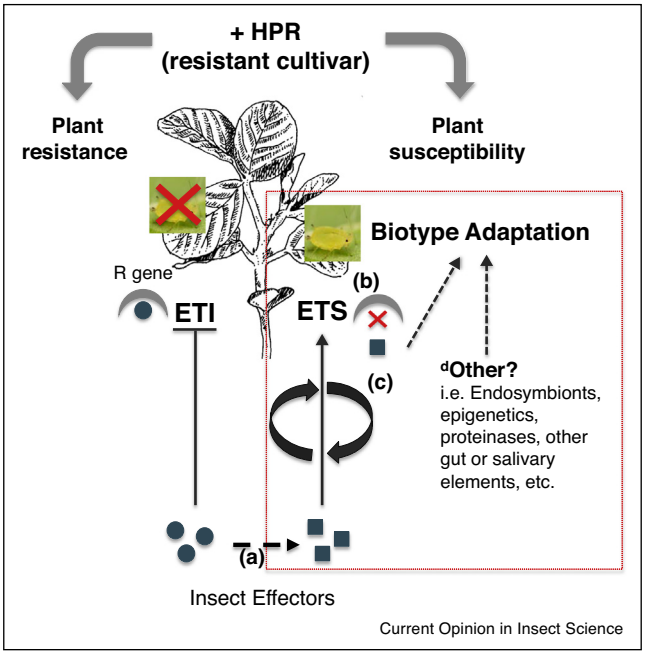
leucine-rich repeats (NBS-LRR, see glossary) [7,14], and several NBS-LRR genes provide (or are thought to provide) HPR to insects [15–18]. This cycle can continue, potentially leading to a diversified and complicated co-evolutionary interaction (Figure 1).

If plants are able to successively perceive an attack, then the signaling mechanisms that lead to both PTI and ETI plant resistance likely overlap [19]. Plants elicit multiple signaling pathways that include general stress responses or pest-specific responses. For aphids, general plant signaling and defensive mechanisms include production of H_2O_2 and reactive oxygen species (ROS), cascades of MAP kinases, callose deposition to reinforce cell walls, and calcium influx to help seal sieve elements and prevent phloem ingestion [2,3,20•,21–28]. Phytohormones such as salicylic acid (SA), jasmonic acid (JA), ethylene (ET), abscisic acid (ABA), and gibberellic acid (GA) also act as signaling mechanisms that lead to downstream defenses such as the production of plant secondary metabolites [25]. However, the phytohormones induced during aphid–host interactions are species-specific (reviewed in [25,29–32]). For example, SA signaling is important for potato aphid (*Macrosiphum euphorbiae)* resistance in tomato [33]. Plant resistance may also be overcome due to insect adaptations against these specific products of plant defense; soybean aphids (*Aphis glycines*) induce ABA-related genes, which may facilitate feeding on susceptible soybean cultivars [34•].

Insect effectors and plant resistance

In most plant–insect interactions, insect effectors play a major role in adapting to PTI and ETI (Table 1) [5••,10,11,13]. The characteristics and mechanisms of

Figure 1



Biotype adaptation within the zig-zag model of plant defense. Insects overcome basal plant resistance (PTI, see text) by inserting effector proteins that modulate defense signaling and lead to effector-triggered susceptibility (ETS) [7]. In some crops with HPR, R genes can recognize insect effectors, resulting in effector-triggered immunity (ETI) [7]. To counteract ETI, insects may evolve novel effectors[a] that are unrecognized by a R gene[b], again leading to ETS. Selection can lead to diversified R genes and insect effectors that can continue to counter-adapt to each other[c] [7]. Currently, we lack evidence that links aphid effectors with biotype adaptation to HPR. More research is required to understand how aphids adapt to HPR via effectors or other mechanisms, such as endosymbionts, epigenetics, or proteinases[d]. A better understanding of biotype adaptation will promote HPR durability in agriculture. The drawing of the zigzag model of plant defense is adapted from [7]. The soybean drawing is copyright of The Ohio State University, and the aphid photo is courtesy of MaLisa Spring.

effectors can be quite variable. For example, effector protein(s) in aphid saliva can bind calcium, which may prevent sieve element sealing during feeding [21,35]. Several effectors promote colonization, but only in specific, aphid–host interactions (see Table 1). It is also possible that some effectors act as a 'decoy' to elicit a specific plant defense, which suppresses a more effective defense [31,34•,36,37]. For example, expression of effector Me47 in tomato triggers PTI but ultimately promotes aphid colonization [37]. It is possible that PTI, which is not an effective defense response in this interaction, is elicited to avoid an alternative defense(s) [37]. Complete characterization of aphid effector mechanisms are challenging since most do not have a homolog outside of aphids [20•,38–40], and many systems are not amenable to functional genetics. Nonetheless, much research supports the major role of insect effectors in plant–insect