Plant–microbe interactions
Affairs of the plant: colonization, intolerance, exploitation and co-operation in plant–microbe interactions

Editorial overview
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Abbreviations
avr avirulence
R resistance

Plants are the primary carbon source for nearly all terrestrial non-photosynthetic organisms, including ourselves. Many species seek to tap this photosynthate, and plants are continuously resisting their strenuous and intimate advances. The review articles in this issue of Current Opinion in Plant Biology address recent progress in several relationships, some more polite and considerate than others, between plants and other organisms. (For reasons of space, most plant herbivore interactions were not addressed.)

Plant resistance to pathogens forms the subject for the first six reviews. Early this century wheat rust resistance was shown to segregate as a dominant Mendelian trait. In the 1940s, HH Flor showed that not only was this also true for a variety of alleles of flax rust resistance (R) genes, but also that virulence genes in the pathogen segregated and are recessive. He proposed the concept of dominant avirulence (avr) genes in the pathogen, which are recognized when the plant carries the corresponding R gene. Presumably these avr genes encode products that are part of a battery of molecules made by the pathogen as it tries to access photosynthate in the host without being noticed. Bacterial, and recently fungal, avr genes have been isolated, yet we know precious little about their mode of action. The last few years, however, have witnessed extraordinary progress in isolation of R genes. As has long been the case for pathogen avr genes, these R genes sequencially beg the question of how their products, upon avr protein recognition, activate the mechanisms of resistance.

The first two articles address what has been learned from this avalanche of R gene sequences. Jeff Ellis and David Jones (pp 288–293) provide a comprehensive review of the four main classes of R gene product structure, with critical commentary on the classification of some gene products. They note the enduring challenge of trying to understand how these R gene products work; how do they recognize avr-dependent signals, and how is that recognition signaled to activate a defense response.

Pam Ronald (pp 294–298) addresses the intriguing issue of R gene evolution. If pathogens are constantly selected to lose avirulence genes to evade recognition, as has been demonstrated in several cases, how does the plant keep up? It is widely believed that the plant must be able to generate novel recognition specificities. There are examples, particularly in the maize Rp1 rust resistance locus, of novel alleles arising correlated with local meiotic recombination. Pam discusses the significance of the observation that many R genes reside as members of local gene families, and that depending on haplotype combination, these loci can be at sites of unequal crossing over. An important point to note is that apparent gene conversion frequencies are very high in R genes and that positive selection appears to act on portions of the R protein.

The question of how to address R gene function is picked up by Roger Innes in his review (pp 299–304) of what has been learned from genetic analysis. One recent insight comes from the observation that some R genes in Arabidopsis, such as RPS2 and RPM1, are suppressed by the mutations at the NDR1 (non-race-specific disease resistance 1) gene, whereas others such as RPP5 and RPS4 are not. Instead, the latter are suppressed by mutations at a different gene, EDS1 (enhanced disease susceptibility 1). EDS1 is not required for RPS2 and RPM1 function. This genetic result is the first to subdivide R protein structural subclasses functionally. Caution is urged, however, as it is now apparent that the signal pathway bifurcation indicated by these results is over-simplified. Roger puts these analyses in the context of other approaches to the question of how plants resist disease.

Genetic analysis is not the only tool that has been productively deployed to study plant recognition and defense. A sustained effort has been made to use biochemistry to understand the events that ensue upon recognition of pathogen-derived elicitors. The most comprehensive information has emerged from work on the parsley cell culture/Phytophthora sojae PEP13 system. Two leading participants in this endeavor review the early events...
of the defense response (Dierk Scheel, pp 305–310) and transcriptional events in the defense response (Paul Rushton and Imre Somssich pp 311–315). Dierk provides a masterful synopsis of the use of suspension culture systems to discover the earliest events correlated with defense activation. In particular, he provides insight into the role of active oxygen species, calcium influx and protein kinases, as well some very recent work that implicates nitric oxide in the defense response and cell death. Paul and Imre pull together a variety of work on different promoters of different defense-related genes, and the approaches that have been used to clone the corresponding transcription factors. The convergence of top down signal transduction and bottom up approaches working from promoters backwards into the transduction chain is upon us.

The historically well studied systemic acquired resistance (SAR) response is also grist for the genetics mill, and Xinnian Dong (pp 316–323) reviews recent progress in analysis of what could be termed the effector arm of the plant innate immune system. Isolation of a key player, the NPR1/NIM1 gene provides Xinnian with a starting point for discussion of mutants in Arabidopsis which illustrate the concept that response pathways are more appropriately modeled by a spider’s web than a circuit diagram. She uses a series of mutants isolated as being ‘more susceptible’ to pathogens to argue that mechanisms of resistance overlap those utilized to limit infection in a nominally susceptible host. How the loci discussed in Xinnian’s review interplay with the R genes and genes defined by virtue of their requirement for R gene function will doubtless be a central theme in next year’s issue of this Current Opinion in Plant Biology topic area.

So much for the defense team. All good invasion forces recognize the need for a broad arsenal, and also for stealth. Plant pathogens are no exception and it is becoming clear that many catapult a broad variety of weaponry into the fray. Wolfgang Knogge (pp 324–328) reviews recent advances in the very earliest events of fungal pathogenesis with an eye toward understanding how individual fungal spores get a grip and start their life cycles. Obviously, if a spore cannot adhere to and recognize its new substrate on the plant and start its life cycle, it is dead. Wolfgang suggests that the evolution from saprophyte to pathogen included the acquisition of key signaling components to take measure of the new environment and pass that information along to the weaponry controls. This advanced scouting by the fungus is relayed by perception of positive colonization signals from the plant and a variety of attachment processes. Clearly, interdiction strategies based on disruption of the initial attachment mechanisms used by fungi would be useful and will follow on what is described in this chapter.

Alan Collmer (pp 329–335) reviews a key mechanistic paradigm for pathogens accessing host nutrient sources during both plant and animal pathogenesis. Alan’s discussion of how plant (and animal) bacterial pathogens secrete and/or inject their weapons into host cells using an evolutionarily conserved system should leave the reader pondering why the bacteria bother to inject proteins into the host that limit pathogen host range—the mechanism of avirulence proteins. The answer that is emerging is logical, namely that these proteins are also virulence factors and that the bacteria deploy a bazaarful of arms into the cell hoping to maximize the return in nutrient access. Sometimes the bug loses, if the appropriate R gene product is present. If not, the so-called avr protein is free to provide a positive function for the bacteria. What the cellular targets of these pathogen proteins are, and how many each bacterial isolate deploys, will be a fascinating chapter in future issues and promises to baffle arms control experts for some time to come.

Viruses survive by stealth and speed. They require host replication factors for multiplication; yet they must have mechanisms to suppress or avoid host responses and to move through the host. Jim Carrington and Steve Whitham (pp 336–341) eloquently describe recent advances in our understanding of these issues. They stress the need to understand host response from the viral perspective and our emerging knowledge regarding the viruses’ ability to manipulate the host cell cycle and replication machinery. Their review should be followed by a refresher course in animal virology, as Jim and Steve make clear that this is one area where convergence with key elements of that field is in sight. The study of plant viral replication will inevitably lead to clarification of plant cell cycle issues, and will add significantly to understanding pathogen avoidance of host response. When plant hosts respond to viruses, they often do so using a very intriguing set of controls which operate by measurement of mRNA or nascent viral transcript levels. Jim and Steve segue neatly into a discussion of what is colloquially known as gene silencing and describe recent experimental evidence which solidifies the notion that silencing plays a functional role in viral protection.

As viruses seek to influence plant cell cycle to their replicative advantage, plant pathogenic nematodes often enslave cell cycle control to build specialized giant, endoreduplicated feeding cells. Pathogenic nematodes are collectively horrific agronomic pests, yet their biology has made them unsatisfactory models (compared to bacteria and viruses) in plant–microbe studies. Charlie Opperman and David Bird (pp 342–346) discuss strategies to remedy this using the nearly completed Caenorhabditis elegans genome sequence as a tool. In addition, they describe their own breakthrough work in mapping soybean cyst nematode virulence loci. This article highlights the need for plant–microbe diplomats to think laterally across species boundaries and also points to a field where comparative genomics will soon have major impact.
John Salmeron and Bernard Vernooij (pp 347–352) describe how the genes isolated thus far are being deployed as a means of controlling disease-related crop losses. They draw attention to the difficulties and limitations of merely using ‘effector arm’ targets such as chitinases, glucanases and other PR proteins. These results illustrate the need to utilize regulatory control points in the defense response for the engineering of broad and durable resistance.

Many important plant–microbe interactions are controlled by biological treaty, and these, of course, are symbiotic. Marco Bladergroen and Herman Spaink (pp 353–359) update the well studied field of nitrogen fixing nodule formation by rhizobia, and are keen to point out that the time has come to integrate the understanding of plant cell biology and hormone responses, thereby creating a detailed understanding of plant processes that translate the action of the nod factor into production of the specialized plant organ, namely the nodule. This is an intriguing question of cell and developmental biology and Marco and Herman integrate recent advances in the biochemistry of Nod factors with emerging knowledge of how their action is ultimately perceived by root cortical cells.

Finally, development of the most common subterranean mutualistic relationship, that between plants and arbuscular mycorrhizal fungi, is detailed by Maria Harrison (pp 360–365). An impressive array of similarities between mycorrhizal colonization and nodulation are illustrated, including the probable role of plant-derived flavonoids in both processes, and the induction of common plant genes in response to both arbuscular mycorrhizal fungi and Rhizobium. Maria is quick to illustrate the importance of genetics, physiology and cell biology as tools to dissect the colonization process. As this sort of colonization is common and critical to sustained agriculture, the development of this new sub-discipline deserves attention.

In sum, these authors are eavesdroppers, snoopers and intelligence experts whose investigations give us insight into different interactions between the Great Powers: the photosynthesizers and the non-photosynthesizers. They have not only outlined today’s knowledge but attempted to help the reader think about the future. The Balkanization of research in plant–microbe interactions that has been observed in the past is behind us, as neatly illustrated by Maria Harrison’s chapter. We can expect more convergence between the types of plant–microbe interaction covered here. And we will surely use plant–microbe interactions not only to inform us of how plants recognize and respond to pathogens, but also to begin to understand how microbes manipulate host cell biology. This manipulation can be crude in the case of necrotrophic pathogens, stealthy in the case of biotrophs and viruses, and nuptially intimate in the cases of rhizobia and mycorrhizal fungi. Investigations into how plants manage their various foreign relations will continue to add to our overall knowledge of plant biology. Isolationism is impossible for sessile organisms, and U. S. Senator Jesse Helms might be well advised to learn from plants that too strong a hypersensitive response can cut you off from vital nutrients.