ANNUAL REVIEWS

Annual Review of Phytopathology

Molecular Dialog Between Parasitic Plants and Their Hosts

Christopher R. Clarke,[1] Michael P. Timko,[2] John I. Yoder,[3] Michael J. Axtell,[4] and James H. Westwood[5]

[1]Genetic Improvement for Fruits and Vegetables Laboratory, Beltsville Agricultural Research Center, United States Department of Agriculture, Agricultural Research Service, Beltsville, Maryland 20705, USA

[2]Department of Biology, University of Virginia, Charlottesville, Virginia 22904, USA

[3]Department of Plant Sciences, University of California, Davis, California 95616, USA

[4]Department of Biology and Huck Institutes of the Life Sciences, The Pennsylvania State University, University Park, Pennsylvania 16802, USA

[5]School of Plant and Environmental Sciences, Virginia Tech, Blacksburg, Virginia 24061, USA; email: westwood@vt.edu

Annu. Rev. Phytopathol. 2019. 57:279–99

First published as a Review in Advance on June 21, 2019

The *Annual Review of Phytopathology* is online at phyto.annualreviews.org

https://doi.org/10.1146/annurev-phyto-082718-100043

Keywords

parasitic plants, *Orobanche*, *Striga*, *Cuscuta*, RNA exchange, haustorium

Abstract

Parasitic plants steal sugars, water, and other nutrients from host plants through a haustorial connection. Several species of parasitic plants such as witchweeds (*Striga* spp.) and broomrapes (*Orobanche* and *Phelipanche* spp.) are major biotic constraints to agricultural production. Parasitic plants are understudied compared with other major classes of plant pathogens, but the recent availability of genomic and transcriptomic data has accelerated the rate of discovery of the molecular mechanisms underpinning plant parasitism. Here, we review the current body of knowledge of how parasitic plants sense host plants, germinate, form parasitic haustorial connections, and suppress host plant immune responses. Additionally, we assess whether parasitic plants fit within the current paradigms used to understand the molecular mechanisms of microbial plant–pathogen interactions. Finally, we discuss challenges facing parasitic plant research and propose the most urgent questions that need to be answered to advance our understanding of plant parasitism.

INTRODUCTION

Parasitic plant: plant that receives all or part of its nutrients through a haustorial connection to another plant

Host plant: plant that provides resources to parasitic plants, likely at a fitness cost

Hemiparasite: parasitic plant that can photosynthesize and thus is not completely dependent on the host plant for sugars

Holoparasite: parasitic plant that lacks the capacity for photosynthesis and thus relies entirely on uptake of sugars from host plants

Facultative parasite: a parasitic plant that can complete its lifecycle as an autotrophic plant but retains the capacity to opportunistically parasitize host plants. Facultative parasitic plants must be hemiparasitic

Haustorium: organ that invades the host and forms a physical and physiological bridge between parasite and host

Parasitic plants live by obtaining part or all of their nutritional needs from another plant. The evolution of parasitism as a life strategy in angiosperms represents the transition of species from autotrophy to an association with—and, in many cases, obligate dependence on—other plant species. This dependence is associated with parasitic plants evolving mechanisms that enable them to identify host plants, grow invasively into the hosts, and establish connections to withdraw water and resources from the hosts. As such, parasitic plants illustrate some of the most exciting examples of plant–plant interactions.

Parasitism as a life strategy is widespread among living species, and plants are no exception (99). Indeed, the ability to form parasitic associations has evolved independently at least 12 times in different plant families (136), and parasitic plants account for roughly 1% of angiosperm species. Parasitism, therefore, can demonstrably provide a fitness benefit to plants in multiple ecological contexts. One trait common among parasites is the need to obtain water and mineral nutrients from their hosts, suggesting that access to water and nitrogen is a driving factor in this evolution (52). In terms of dependency on host photosynthesis and metabolism, large differences exist among parasitic plant species, spanning the spectrum from hemiparasite to holoparasite and facultative to obligate parasites. Nevertheless, it is important to note that all parasitic plants evolved from autotrophic ancestors, so at the cellular level, they retain the distinctive features of plants such as plastids and cell walls.

At the whole-plant level, parasitic plants exhibit a wide range of different morphologies and structurally different haustorial connections. The haustorium connects parasites to their hosts and provides a conduit for extracting host resources. As a result, parasite species are freed from many of the normal constraints of autotrophic plants; they may also have reduced or absent organs such as roots or leaves and may lack chlorophyll. Some species exist primarily underground, attached to host roots, emerging above the soil only to flower (e.g., *Phelipanche* and *Orobanche*). Other species may pass substantial portions of their vegetative life cycle growing inside their hosts (e.g., *Rafflesia*, mistletoes). For the latter, the shelter provided by living underground or within the host could provide another advantage for parasitism. But given the diverse origins of parasitic plants, it is difficult to generalize among the various types.

The Orobanchaceae and Cuscutaceae families have received the most research attention to date (**Figure 1**) because they include some of the most economically important parasitic weed species. Orobanchaceae are root parasites and include the genera *Striga* (witchweed) and *Alectra*, which pose a great threat to cereal production in sub-Saharan Africa, India, and Asia (94). The related species in *Phelipanche* and *Orobanche* (together commonly called broomrapes) destroy yields of broad-leaved crops throughout North Africa, the Mediterranean, Europe, and the Middle East. Although the hemiparasites *Triphysaria versicolor* and *Phtheirospermum japonicum* are not weedy, they have been developed into model parasite species that can be readily cultured in the laboratory and genetically transformed (54, 121). Besides the Orobanchaceae, *Cuscuta* species are important stem parasites that cause substantial economic impact (94) and have increasingly become the subject of parasitic plant research. All these species have contributed to exciting developments in our understanding of the mechanisms of parasite–host interactions.

The study of parasitic plants spans the disciplines of weed science and plant pathology. On the one hand, these parasites are angiosperms and have the same basic biochemistry, genetics, and physiology as do fully autotrophic plants. From an agronomic perspective, parasitic weeds may be controlled in part by mechanical or chemical methods similar to those used on autotrophic weeds, thus falling under the purview of weed science (4, 30, 37). On the other hand, parasitic plants may deviate substantially from fully autotrophic plants in terms of physiology and morphology because they behave like pathogens, with specific adaptations for acquiring resources from their

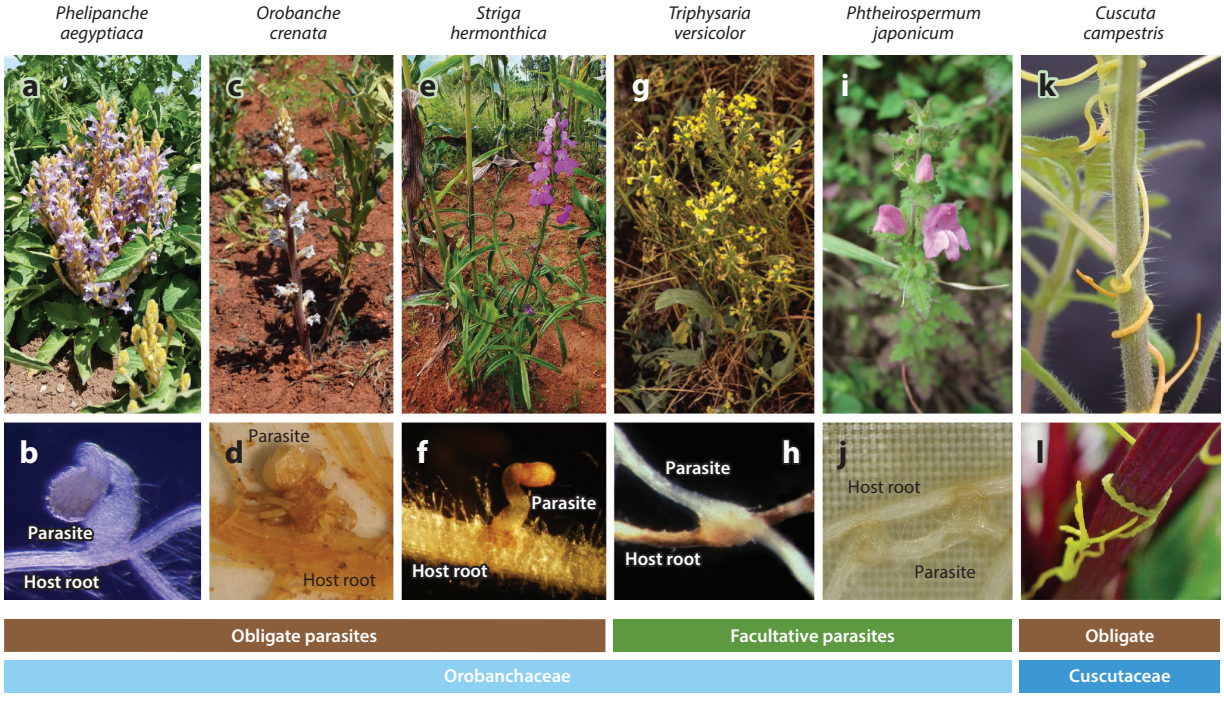

Figure 1

Some of the most widely studied parasitic plant species. The top row shows a typical view of the parasite, and the bottom row shows examples of parasite–host connections. *Phelipanche aegyptiaca* is shown (*a*) tomato and (*b*) *Arabidopsis*, *Orobanche crenata* on (*c*) faba bean and (*d*) lentil root, *Striga hermonthica* on (*e*,*f*) sorghum, *Triphysaria versicolor* (*g*) in the field and (*h*) connected to the root of the unknown host. *Phtheirospermum japonicum* is shown (*i*) in the field and (*j*) connected to rice root via two haustoria. *Cuscuta campestris* is shown (*k*) on tomato and (*l*) as a close-up of the haustorial region on beet. Photos provided by (*a*,*b*,*c*,*e*,*k*,*l*) James Westwood, (*d*) Rashid Mentag, (*f*) Michael Timko, (*g*,*h*) John Yoder, and (*i*,*j*) Satoko Yoshida.

hosts. Researchers have described multiple levels of parasitic plant signaling, including the specific triggering of germination of parasite seeds by host root exudates, tropic growth of the parasite radicle/stem toward the host, and initiation of the haustorium as well as myriad chemical and physical interactions that mediate the haustorial interaction with host tissues (**Figure 2**). This review focuses on recent advances in parasitic plant biology that relate to these interactions and support the consideration of parasitic plants as plant pathogens.

PARASITE IDENTIFICATION OF HOSTS

Germination

One of the most intriguing aspects of plant–plant communication is the ability of seeds of certain obligate parasites of the Orobanchaceae family to coordinate their germination with the presence of a nearby host root. This feature is important because these seeds are extremely small and limited to only a few millimeters of radicle growth before they must contact and attach to a host root for continued survival. Germination without a host, or in the presence of a nonhost, results in death. The mechanisms underlying host-specific germination posed a long-standing challenge to researchers, but recent breakthroughs have reshaped our knowledge of germination stimulants. We briefly describe the process of host-specific germination, but several recent reviews provide excellent coverage of this area (27, 83, 85, 87, 107, 109, 132, 148).

Obligate parasite: parasitic plant that relies on host plant parasitism to complete its life cycle

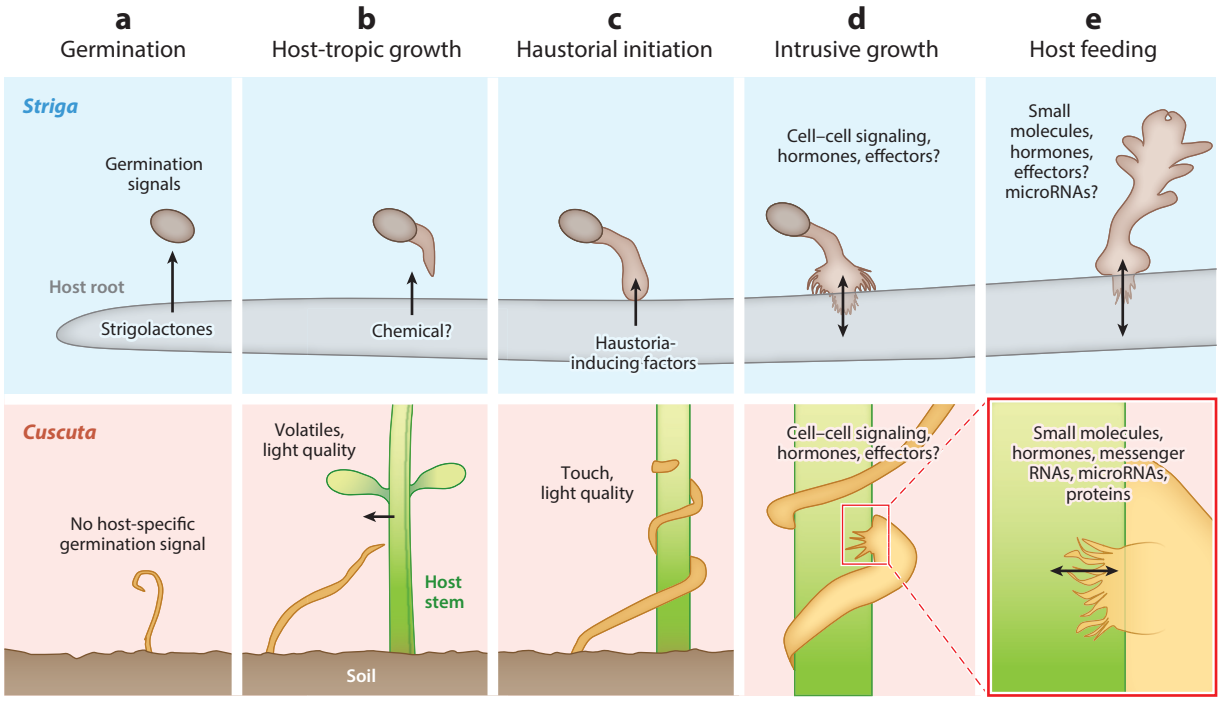

Figure 2

Schematic of key aspects of parasite–host communication contrasting between the Orobanchaceae root parasite *Striga* (*top*) and the stem parasite *Cuscuta* (*bottom*). Communication is divided into five points in the life cycle in which prominent aspects of plant interactions are recognized.

Seeds of obligate parasites such as *Orobanche*, *Phelipanche*, and *Striga* may lie dormant in the soil for years until they detect the chemical signature of an appropriate host root (**Figure 2*a***). Strigolactones (SLs) are the best-characterized class of germination stimulants for members of the Orobanchaceae. SLs are hormones produced by all green plants higher than algae (29). They help regulate plant architecture through control of branching and recruit soil mycorrhizal fungi via exudation into the rhizosphere (1, 38, 126). At least 20 different SL molecules have been identified in plants (138), and plant families produce varying forms such that parasite seeds are able to differentiate among hosts on the basis of the identity of exuded SLs.

Strigolactones (SLs): class of plant hormones exuded from plant roots to foster interactions with mycorrhizal fungi

The structure of SLs is key to their function. Canonical SLs are carotenoid-derived compounds (79) that have a four-ring structure consisting of fused ABC rings linked to a D ring by an enol-ether bond. Critical to SL function, the D ring is cleaved and subsequently bound by a receptor in SL signaling (148). Different SLs are distinguished by the presence or absence of methyl or hydroxyl groups on the A and B rings and by the stereochemistry of the BC ring conformation. Like SLs, other compounds such as methyl carlactonoate can stimulate germination (132). A common feature of such compounds is a butenolide ring similar to the D ring of SLs.

Recent advances in SL signaling have partially solved the long-standing mystery of how parasitic plants detect germination stimulants. Plants are able to perceive SLs through a family of receptors related to DWARF 14 (D14), an α/β-hydrolase superfamily protein. For signaling of

endogenous SLs, binding to D14 results in hydrolytic cleavage of the D ring from the ABC rings and covalent binding of the D ring to D14, leading to a conformational change that promotes physical association between D14 and MORE AXILLARY GROWTH2 (148). The latter is an F-box protein that functions in signaling as part of a Skp1/Cullin/F-box E3 ubiquitin ligase complex. Parasitic plants use a parallel and more ancient (16) mechanism that involves a protein related to D14 termed KARRIKIN INSENSITIVE 2 (KAI2; also known as HYPOSENSITIVE TO LIGHT) (82). The KAI2 genes in parasitic Orobanchaceae appear to have undergone expansion and specialization to become a rapidly evolving family termed diverged KAI2 (22) that provides parasites with a mechanism to recognize specific host SLs, adapt to changes in host SL profiles, and shift to recognize new hosts. For example, *Striga hermonthica* has at least 11 KAI2 genes, although only *ShTHL7* encodes a protein that is especially responsive to SLs (120), leaving the remaining genes free to evolve to encode proteins that recognize different SLs or related compounds.

Locating the Host

Chemotropic growth of the parasite radicle in Orobanchaceae has been postulated for years (**Figure 2*b***) (58). Without such a mechanism, the emerging radicle, which is already constrained in terms of available resources for growth, would have a relatively low chance of contacting the host root. Although no specific chemical signal has been identified, time-lapse video suggests that the *S. hermonthica* radicle bends toward the host root as it elongates (143).

Growth toward the host is more easily observed in *Cuscuta*. The seedling of this plant germinates without the need for specific signals and emerges from the soil in spring along with other plant seedlings. The challenge for *Cuscuta* is that the thread-like shoot must make contact with a host plant so that it can coil and form haustorial attachments. The stems of *Cuscuta* seedlings rotate in a counterclockwise rotation that is not entirely random as they explore their aerial environment for host plants (67, 68, 74, 125). One factor guiding the shoot toward a host is the detection of volatile chemicals emitted from the host plant (105). Under carefully controlled conditions, *Cuscuta pentagona* seedlings preferentially grew toward tomato plants or tomato volatiles (an acceptable host) rather than to wheat (which will not sustain *Cuscuta*) or wheat volatiles. *Cuscuta* is also sensitive to light spectrum for coiling and haustoria formation (43, 61), and the tropism of the *Cuscuta* shoot apex is a response to far-red light, which indicates nearby vegetation (49, 93). Exposure to red light (660 nm) inhibits the induction of haustoria, suggesting the involvement of phytochromes in haustorium initiation (36).

Cuscuta and Orobanchaceae use tactile signals in addition to chemical cues and light quality to identify appropriate hosts. Although light quality is critical for coiling, haustorium development is initiated only after coils come in contact with the host, indicating a further requirement for a tactile signal (118). A true host is not essential for the tactile response because any solid surface (e.g., glass rod or plastic stick) is sufficient for coiling and haustorium initiation. Tactile cues are also stimulatory for haustorium development in Orobanchaceae, and haustoria form and attach to inanimate surfaces such as rocks or petri dishes (75, 101, 137).

THE HAUSTORIUM AND ITS DEVELOPMENT

Haustorium Initiation

Haustoria are parasite-encoded, multifunctional organs that attach the parasite to the host, penetrate into host tissues, and ultimately establish the physiological conduit through which resources

flow between parasite and host (9, 57, 142). There are two general types of haustoria: lateral haustoria, which develop on the sides of roots or stems, and terminal haustoria, which develop on the tips of newly emerging radicles. Lateral haustoria are more common and are characteristic of haustoria in *Cuscuta* and most Orobanchaceae (**Figure 1**). Terminal haustoria are more specialized. They are restricted to obligate parasites such as *Striga*, *Orobanche*, and *Phelipanche*, to which they provide a mechanism for rapidly invading a host root within days of germination. Terminal haustoria alter the tip meristem so no additional root growth occurs until the haustorium has successfully invaded the host. In contrast, lateral haustoria develop on functioning roots without altering the tip meristem so multiple haustoria can develop on a single root. Phylogenetic distribution of haustorial types suggests lateral haustoria predate terminal haustoria (133). We focus primarily on Orobanchaceae parasites, which dominate the literature.

Haustoria-inducing factors (HIFs): chemical cues released by host plants stimulating production of haustorial connections in parasitic plants

Haustoria typically develop only in the presence of host plants, suggesting that parasitic plants have mechanisms to locate and distinguish potential host plants (**Figure 2*c***). Orobanchaceae recognize potential host roots through a combination of chemical and tactile cues. Five general classes of molecules have been identified as active haustoria-inducing factors (HIFs): flavonoids, phenolic acids, quinones, cytokinins, and cyclohexene oxides (3, 20, 32, 101, 114). The redundancy in HIFs may account for the broad range of host plants parasitized by Orobanchaceae species.

The first HIF identified from host root extracts was 2,6-dimethoxy-1,4-benzoquinone (DMBQ). It is formed from the oxidation of syringic acid, which can be an oxidative degradation product of lignin (20). This finding led to the model that parasite-encoded peroxidases, together with hydrogen peroxide present at the parasite radical tip, convert host cell wall lignins into active HIFs (69). Consistent with this model, the effects of lignin composition on haustorium induction differ among parasites: *P. japonicum* and *S. hermonthica* develop haustoria in response to S- and G-type lignins, respectively (24). However, nearly eliminating both of these lignins in the *Arabidopsis med5/5b* mutant did not eliminate HIF activity, suggesting that additional HIF molecules are present in *Arabidopsis* root exudates (24).

Because phenolic acids become active HIFs only after oxidization to their sister quinones, haustorium induction may depend on the redox state of the inducer (84, 111). Subsequent experiments indicated that semiquinone intermediates formed during redox cycling between quinone and hydroquinone states of the HIF initiate a redox-sensitive signaling pathway leading to haustorium development (70, 145). Redox cycling is catalyzed by quinone oxidoreductases, and transforming *T. versicolor* or *P. japonicum* roots with inhibitory RNA molecules targeting specific quinone oxidoreductases significantly reduces haustorium development (8, 55). Interestingly, different quinone oxidoreductase enzymes are recruited in *Triphysaria* and *P. japonicum*, so multiple enzymes may be involved in redox changes to the HIFs.

Not all Orobanchaceae respond to the same HIFs. For example, *T. versicolor* forms haustoria in response to DMBQ, but *Triphysaria eriantha* does not (56). Similarly, *Orobanche* and *Phelipanche* do not form obvious haustorial structures in response to DMBQ (60), but they form haustoria when treated with root exudates of *Brassica napus* (39). In the latter case, the HIF may be a host-exuded cytokinin signal. Sphaeropsidone and episphaeropsidone, two phytotoxic cyclohexene oxides isolated from the fungus *Diplodia cupressi*, induce haustorium development in *S. hermonthica*, *Orobanche crenata*, and *Orobanche cumana* (32). Thus, there is redundancy among the types of molecules capable of inducing haustorium development in Orobanchaceae.

Attachment via Haustorial Hairs

The first contact between certain Orobanchaceae parasites and hosts is made by haustorial hairs, which cement the parasite to the host. Both haustorial and root hairs are of epidermal origin, and

their development entails rapid tip growth (101, 108). However, haustorial hairs are also distinct from root hairs. Whereas root hairs develop at multiple locations on the root, haustorial hair proliferation is highly localized to the area of the root that develops into a haustorium. Screening of a mutagenized population of *P. japonicum* yielded three recessive mutations defective in haustorial hair formation, two of which are allelic (25). These lines formed haustoria but neither root nor haustorial hairs, suggesting a genetic connection between the two hair types. When host and parasite roots were forced to grow closely together, haustorial hair mutants produced similar numbers of haustoria as do wild-type *P. japonicum*. Thus, haustorial hairs may play a role in host–parasite associations but not in haustorium initiation.

Hormone Action

As growth regulators of plant organogenesis, hormones play multiple roles in the development of haustoria. Whereas exogenous auxin stimulated haustorium development in *T. versicolor* seedlings, both auxin efflux inhibitors (2,3,5-triiodobenzoic acid) and auxin activity inhibitors (*p*-chlorophenoxyisobutyric acid) did the opposite (122). Transgenic *Triphysaria* roots bearing reporter constructs driven by auxin or ethylene-responsive promoters reacted positively when exposed to DMBQ, suggesting these two hormones are differentially abundant at the sites of haustorium development.

The YUCCA (YUC) family of flavin monooxygenases catalyzes the rate-limiting step of indole-3-acetic acid formation (86). In *P. japonicum*, YUC3 is highly upregulated specifically in the parasite epidermal cells around the host contact site (53). Transgenic *P. japonicum* roots expressing YUC3-silencing constructs formed significantly fewer haustoria than did nontransgenic controls. Furthermore, transgenic *P. japonicum* roots expressing YUC3 ectopically induced the formation of haustorium-like structures at the root epidermal cells. Thus, YUC3 expression in epidermal cells near the site of host contact may play a role in haustorium development.

In addition to Orobanchaceae, auxin is associated with haustorium development in *Cuscuta* and *Thesium chinense*, a facultative root hemiparasite in Santalaceae. In both *Cuscuta* and *T. chinense*, auxin-related genes are upregulated in haustoria (100, 146). Metabolite analysis of *T. chinense* haustoria indicated the presence of very-long-chain fatty acids that are often associated with cell proliferation, tissue patterning, and roles in polar auxin transport that determine cell polarity in *Arabidopsis* lateral root development (104).

Cytokinins also contribute to haustorium function. Parasite-synthesized cytokinins that are transported from *P. japonicum* into *Arabidopsis* cause hypertrophic swelling near the site of infection (113). *P. japonicum*–induced host swelling required the host cytokinin signaling genes *AHK3* and *AHK4* but not the cytokinin biosynthesis genes *IPT1*, *IPT3*, *IPT5*, or *IPT7*. Host plants with hypertrophic swelling were smaller than were *AHK3* mutants, which did not swell, suggesting that the movement of cytokinin from parasite into hosts is a factor in successful parasitism (113).

Evolutionary Origins of Haustoria

There are two general theories for the evolutionary origins of the haustorium. The first model proposes that genes involved in haustorium development have an exogenous origin and were introduced into parasitic lineages through pathogen infection or horizontal gene transfer (7). Recently, 52 high-confidence horizontally transferred genes were identified in the transcriptomes of *T. versicolor*, *S. hermonthica*, and *Phelipanche aegyptiaca* (140), and 64 such transfers were detected in *Cuscuta campestris* (130). Most of these genes in the Orobanchaceae family are preferentially

expressed during haustorial formation, suggesting that horizontal gene transfer contributed to the evolution of their haustoria.

RNA interference: process by which RNA molecules inhibit gene expression

microRNAs: small noncoding RNA molecules that function in RNA silencing and post-transcriptional regulation of gene expression

The second model indicates that genes for haustorium development are endogenous to parasitic lineages where they fulfill functions unrelated to parasitism. Subsequent mutations in these genes or their regulation machinery resulted in altered functions or ectopic expression used for parasitic purposes. Orobanchaceae haustoria are formed on roots, and gene expression in haustoria is most similar to that of roots (139). Development of both haustoria and lateral roots requires localized auxin accumulation, and inhibiting auxin flux reduces haustorium development (53, 122, 142). Thus, the molecular developmental machinery used for lateral root formation in nonparasitic plants may have been co-opted into the developmental reprogramming of haustorial formation in parasitic plant lineages.

In *Cuscuta*, the KNOTTED-like homeobox transcription factor SHOOT MERISTEMLESS is upregulated during haustorium formation, and inhibition of its RNA through host-induced gene silencing reduced the ability of haustoria to connect and establish (2). SHOOT MERISTEMLESS is also associated with stem phenotypes in other plants, and its elimination has no effect on *Arabidopsis* roots. These results suggest the evolutionary origin of haustoria in *Cuscuta* contains elements of both root and shoot developmental programs (2).

PARASITE–HOST EXCHANGE OF RNAs

Parasites exchange proteins and RNAs with their hosts that could contribute to shaping interactions between species (**Figure 2*e***). Host mRNAs can move into *Cuscuta*, often traveling long distances (30 cm) from a haustorium (26, 103). There appears to be selectivity for the uptake of certain mRNAs (73), and different host-derived mRNAs have different decay rates once inside *Cuscuta* (77). Thus, delivery or uptake of host mRNAs by *Cuscuta* is at least partially directed, although random bulk acquisition may also occur (19). *Cuscuta* mRNAs can also be found in the host, and large numbers of mRNAs are transferred (72). To date, it has not been conclusively shown that mobile mRNAs are functional after transfer across *Cuscuta* haustoria. Mobile mRNAs could be functional in recipient plants as templates for translation, thus delivering foreign proteins. Alternatively, mobile mRNAs could serve as templates for the production of small silencing RNAs once they move into the recipient species.

Functional RNA-interference signals also move across parasitic plant haustoria. Hairpin transgenes in host plants trigger effective RNA interference in both *T. versicolor* (10, 123) and *C. pentagona* (2). This host-induced gene silencing can be used to target parasite genes critical for parasite growth and is therefore a potential strategy for crop improvement. It also implies that small RNAs, which are the effectors of RNA interference, may be able to enter the parasite through haustoria. Furthermore, naturally occurring small RNAs, which are very numerous in plants, might also be transiting haustoria.

In *C. campestris*, there is clear evidence that naturally occurring small RNAs transit haustoria and are functional in recipient host plants (110). A large number of *C. campestris* microRNAs are strongly expressed specifically at haustorial junctions. Molecular evidence shows that several of these microRNAs are active against host mRNAs. Confirmed host targets of *C. campestris* microRNAs include mRNAs encoding auxin receptors, developmental regulators, pathogen defense signaling, and phloem function (**Table 1**). Host plants mutated in two different targets (*AFB3* encoding an auxin receptor and *SEOR1* encoding a phloem protein) support increased growth of *C. campestris* (110). This is consistent with the hypothesis that *C. campestris* microRNAs silence host genes to increase parasite fitness. Therefore, *C. campestris* microRNAs act as virulence factors. *C. campestris* has a very broad host range, so parasite-derived microRNAs should be active in multiple

Table 1 Host mRNAs targeted by *Cuscuta campestris* microRNAs and their functions

Target mRNA family	microRNA family	Confirmed species	Target functions
SEOR1	ccm-miR12480	*Arabidopsis thaliana*	Major phloem protein that reduces loss of sugar upon wounding
TIR/AFB	ccm-miR12497	*A. thaliana*, *Nicotiana benthamiana*	Auxin receptors
BIK1	ccm-miR12495, ccm-miR12463	*A. thaliana*, *N. benthamiana*	Kinase involved in signaling during defense responses to multiple pathogens
SCZ/HSFB4	ccm-miR12494	*A. thaliana*	Transcription factor controlling stem-cell identity in roots
Ran BP2/NZF	ccm-miR12486	*N. benthamiana*	Unknown

hosts. Indeed, similar sets of *C. campestris* microRNAs are induced when either *Arabidopsis thaliana* (Brassicaceae) or *Nicotiana benthamiana* (Solanaceae) is used as a host. Additionally, homologs of the same mRNAs in different host species are targeted by *C. campestris* microRNAs, and bioinformatic predictions suggest that targeting of these gene families is feasible across the host range of *C. campestris* (110).

PARASITIC PLANTS AND THE ACTIVATION AND SUBVERSION OF HOST PLANT IMMUNITY

Haustoria and Host Defense Interactions

Parasitic plants affect a wide range of hosts, with some species showing high levels of host selection and specialization. We think of host range as the total number of different species that can be parasitized by a given parasite species, whereas host preference refers to which host the parasite may be best adapted to parasitize. The battle between host innate immunity (resistance) and parasite virulence occurs across a number of developmental stages.

Host resistance to attack by parasitic Orobanchaceae (and to some extent *Cuscuta*) can be categorized as occurring at two different time points: either pre- or post-attachment resistance (**Figure 2**) (52, 119). Pre-attachment resistance includes all mechanisms that allow a potential host plant to avoid or prevent parasite attachment, including (*a*) no or reduced production of germination stimulant(s); (*b*) production of germination inhibitors; (*c*) delay, reduction, or complete inhibition of haustorium formation leading to attachment incompetence; and (*d*) development of preformed mechanical or structural barriers on the host surface to impede attachment. The latter could include evolved enhanced cell wall lignification, suberinization, or other modifications and structures (hairs or other outgrowths) that retard attachment to the host.

Post-attachment resistance operates when the attached parasite haustorium at the host surface attempts to penetrate host tissues to make connections with the vascular system. During these developmental stages, cells within a palisade layer at the leading tip of the haustorium differentiate to form a penetration peg and begin the process of traversing the host cortex. Little is known about the exchange of molecular information that cues the transition to the penetration peg, although studies currently underway looking at the host–parasite interface could shed light on this process (50, 139). Because successful penetration and vascular linkup are crucial to parasite reproductive success, parasite post-attachment strategies may have been fine-tuned through evolution.

In fact, substantial experimental evidence demonstrates that parasitic plants activate expression of genes encoding various cell wall degrading/softening enzymes (e.g., pectate lyases, pectin

methylesterase, polygalacturonase, endocellulase, β-glucosidase or β-xylanase, expansins) to help breach the host epidermis and cortex and allow penetration through and past the endodermis (11, 71, 81, 92, 96, 100, 128). In addition to breaking down the pectin-rich apoplastic layers between the cells and fabric of host cell walls, mechanical pressure exerted by the parasite penetration peg assists in creating a path for parasite invasion (95). Comparative transcriptomic analysis of pre- and post-attachment haustoria from three Orobanchaceae species (*S. hermonthica*, *P. aegyptiaca*, and *T. versicolor*) has also shown that gene duplication during haustorial evolution and subsequent repurposing may have led to the recruitment of genes from other invasive structures such as pollen tubes (139) to be expressed in the haustorium, allowing the parasite haustorium to function as an intrusive organ.

Resistance genes: host plant genes that confer strong immunity against a pathogen, often through direct or indirect detection of effector proteins

During this intrusive process, the host can succumb passively, rely on constitutively expressed general defense responses, or activate specific innate immune response cascades to fend off parasitic ingress (45, 47, 88, 91, 117, 119). Innate immunity can present as (*a*) abiosis, the synthesis and release of cytotoxic compounds (e.g., phenolic acids, phytoalexins), by the challenged host root cells; (*b*) rapid formation of physical barriers to prevent possible pathogen ingress and growth (e.g., lignification and other forms of cell wall modification at the host–parasite interface); (*c*) release of reactive oxygen species and activation of programmed cell death in the form of a hypersensitive response at the point of parasite attachment to limit parasite development and retard its penetration; and (*d*) prevention of the parasite establishing the essential functional vascular continuity (i.e., xylem-to-xylem and/or phloem-to-phloem connections) with the host, delaying parasite growth followed by parasite developmental arrest and eventual death (15, 91).

At each stage of parasite ingress, the challenged host has an opportunity to mount a resistance response such that measures of parasite success (e.g., rate of parasite establishment or tubercle development, final number of emerged shoots) more properly reflect the ability of the parasite to overcome the various resistance mechanisms activated. We often think of susceptibility (compatible host–parasite interactions) as a null situation where the apparent lack of host response belies the inability of hosts to recognize the invading haustorial penetration peg and activate the cascade of defense-related signaling events. Host susceptibility to parasite attack could also result from active suppression of the host defense mechanism by parasite-derived/-secreted molecules (6). Indeed, the parasite could have evolved mechanisms to bypass surveillance or limit host defense activation (discussed below).

The exchange of potential defense signaling molecules across the host–parasite haustorial interface is well established. For example, in *Cuscuta*, defense signals mediated by small molecules clearly pass through haustoria. Glucosinolates, which are typical secondary metabolites made by Brassicales plants, move from host to *Cuscuta* and affect aphid feeding (112). Insect feeding triggers jasmonic acid–dependent transcriptome responses in hosts that are bridged by a *Cuscuta* vine (46), thereby affecting subsequent insect feeding (147). Thus, jasmonic acid or a related compound likely moves through *Cuscuta* haustoria in response to herbivory.

Most interactions between *Striga* species (e.g., *S. hermonthica*, *Striga asiatica*, *Striga aspera*) and members of the Poaceae appear to elicit resistance responses controlled in a polygenic fashion; each quantitative trait locus contributes a small effect, and hosts often exhibit high phenotypic variations when challenged by different parasite populations (5, 40, 41, 44, 102, 117). In contrast, resistance to *Striga gesnerioides* in legumes and other dicots appears to be monogenically inherited, and, in some cases, the resistance genes are highly parasite-race specific. The one reported exception is resistance to the biotype found in Mali and Niger (SG2/SG3) in cultivar IT82D-849 that is conferred by a single recessive gene (124). Resistance to *Orobanche* and *Phelipanche* spp. in legumes is polygenic (33, 34), whereas it is monogenically conditioned with a number of epistatic environmentally conditioned modifiers in sunflower (*Helianthus* spp.) (35, 97).

A Place for Parasitic Plants in the Current Model of Plant–Pathogen Interactions?

The above discussion suggests that parasitic plants are more similar to microbial plant pathogens than to weeds. Parasitic plants, like microbial plant pathogens, must interact closely with host plants to extract nutrients and suppress defense responses. For example, parasitic plants form haustorial connections with host plants that are functionally similar to haustoria formed by oomycete and fungal plant pathogens (98, 141). It is likely that knowledge of host immune responses against other plant pathogens can be used to develop testable hypotheses about the roles of the host immune system in interactions with parasitic plants. Although the mechanisms of host immunity against microbial plant pathogens are being elucidated at an increasing rate, these insights have not been systematically applied toward understanding interactions between host plants and parasitic plants.

The leading paradigm for host plant–microbial pathogen interactions is known as the zigzag model (62). In this model, plants and pathogens are locked in a perpetual arms race. The plant immune system surveils for conserved microbe-associated molecular patterns (MAMPs) of microbial invaders using pattern-recognition receptors and, upon detection, elicits an immune response known as pattern-triggered immunity (66), which is sufficient to fend off the vast majority of would-be microbial pathogens. Successful pathogens rely on their own deployment of immunity-suppressing effector proteins leading to effector-triggered susceptibility—a compatible interaction resulting in disease.

The immune response of host plants to parasitic plants is also likely multifaceted. To date, layered immunity against parasitic plants is best demonstrated in the resistance of diverse monocot and dicot species against *Striga* species (143) and the resistance of sunflower to *O. cumana* (89). In both of these parasitic plant–host plant interactions, the host plant activates immune responses at multiple stages of parasite development, limiting the severity of parasitization. The specific molecular actions that underpin most parasitic plant–host plant immune interactions, however, remain largely unknown. We hypothesize that many aspects of the models used to describe host plant–microbial pathogen interactions provide a valuable framework for understanding host plant–parasitic plant interactions, specifically post-attachment.

Because parasitic plants and host plants are much closer relatives than are microbial pathogens and host plants, detection of nonself molecular patterns (such as MAMPs) is likely less common in interactions between the former than the latter. However, a parasite-associated molecular pattern (ParAMP) was recently identified in *Cuscuta reflexa*, and heterologous expression of its cognate pattern-recognition receptor, *CuRe1*, in host tomato increases resistance to parasitism (45). This discovery demonstrated that pattern-triggered immunity can be an important component of the host plant immune response against parasitic plants. ParAMPs must be present in only the parasitic plant. Therefore, we hypothesize that many ParAMPs are related to structures specifically associated with the development of the haustorium, the primary parasite-specific structure. A fundamental concept in plant pathology is that, owing to a robust innate immune system, most plants are actively resistant to most pathogenic organisms. If most host–parasite interactions are likewise incompatible, detection of ParAMPs may play a critical role, similar to detection of MAMPs in host plant–microbial pathogen interactions.

Not included in the original formulation of the zigzag model is the important role of the host plant immune system in detecting so-called damage-associated molecular patterns (DAMPs). These endogenous components are present only following the effects of pathogens (42). Many DAMPs are likely released during haustorial formation, when large-scale remodeling of host cells occurs (discussed above), and may be critical determinants of the compatibility of parasite

Microbe-associated molecular patterns (MAMPs): oft-conserved feature of microbes for which the plant immune system surveils as an elicitor of pattern-triggered immunity

Pattern-recognition receptors: receptors that perceive pathogen-associated danger signals to elicit an innate immune response

Pattern-triggered immunity: innate host plant immune response activated following detection of pathogen-derived components

Effector: pathogen-derived protein that acts in the cytoplasm or apoplast of host plants to suppress immunity or otherwise increase virulence

ParAMP: parasite-associated molecular pattern

Damage-associated molecular patterns (DAMPs): endogenous host plant structures present following pathogen-induced or other damage to the host plant

interactions. To our knowledge, DAMPs in host–parasite interactions have not yet been identified or functionally characterized for any parasitic plants.

Effector-triggered immunity: host plant immune response resulting in an incompatible interaction following detection of pathogen-secreted effector proteins by host plant resistance proteins

Susceptibility factor: host plant–derived product or feature critical for parasitic plants to complete successful parasitism

Microbial plant pathogens subvert immunity elicited following the detection of MAMPs and DAMPs through either the diversification of MAMPs to evade detection (129) or the secretion of immunity-suppressing effector proteins (13). Parasitic plants likely employ a number of distinct strategies to suppress host plant immunity, but the deployment of effectors is a reasonable hypothesis considering that bacteria, oomycetes, fungi, nematodes, and aphids all independently evolved this strategy to deceive host plant immune systems (14, 28, 31, 48, 64, 90). Fungi and oomycetes both rely on their haustorial connections to secrete effectors into host plants (80). However, this mechanism is likely distinct in parasitic plants given the stark structural differences in the corresponding haustoria.

Owing to the increased availability of genomic and transcriptomic data sets, the potential role of effector proteins in mediating plant–plant parasitism is beginning to be elucidated. Transcriptome analyses of *S. hermonthica*, *S. gesnerioides*, *T. versicolor*, and *P. aegyptiaca* haustoria before and after host interaction allowed for the identification of novel developmentally expressed transcripts associated with compatible and incompatible interactions as well as of transcripts encoding putative secreted effector proteins that may serve to manipulate host immunity (51, 115, 135, 139, 143, 144). Functional characterization of candidate effector proteins was recently performed for race-specific putative effectors from *S. gesnerioides* and many haustorial upregulated putative *P. aegyptiaca* effectors (115) (C.R. Clarke, S.-Y. Park, Z. Yang, X. Jia, E. Wafula, L. Honaas, C. Yang, C.W. dePamphilis, J.H. Westwood, unpublished results). Surprisingly, from both of these independent screens, short proteins with three leucine-rich repeat motifs emerged as top candidate effectors. The candidate effectors are homologous to the leucine-rich repeat domain of SERK family proteins involved in plant immunity and development (21), suggesting that translocation of these parasite-derived proteins into host cells may perturb host development or immunity through molecular mimicry. Additionally, analysis of the *S. asiatica* genome has identified several-hundred putative uncharacterized secreted proteins whose functional characterization as effectors requires further demonstration (142).

An additional zag in the zigzag model is effector-triggered immunity (23). In this phase, host plants evolved *Resistance* (*R*) genes capable of detecting pathogen-derived effector proteins and initiating a robust immune response. Effector-triggered immunity has yet to be directly demonstrated in parasitic plants. However, a nucleotide-binding site–leucine-rich repeat immune sensor protein, typical of classical *R* genes, confers resistance against the parasite in cowpea–*S. gesnerioides* interactions (78). Identification of a cognate *S. gesnerioides* effector for this *R* gene would provide an unambiguous demonstration of effector-triggered immunity in parasitic plants. Whether such immunity is mediated by *R* genes per any of the current leading models (63) or through novel mechanisms is currently unknown.

Also not expressed in the zigzag model are the host-derived susceptibility factors that are required for the pathogen to complete its life cycle (127). Although they have not been robustly characterized in parasitic plant–host plant interactions, potential susceptibility factors include (*a*) host-derived germination stimulants and HIFs, (*b*) components of the cell wall and cell membrane that must be modified to form successful haustoria, (*c*) regulators of the plant immune system that are affected by parasitic plants, and (*d*) metabolic or nutrient transport genes that are hijacked by parasitic plants to meet their nutritional needs. Recently, screening for the ability of *P. aegyptiaca* to parasitize a large collection of *Arabidopsis* mutants, researchers found that perturbations to the jasmonic acid biosynthetic or signaling pathway led to less compatible interactions, suggesting that components of these pathways may represent potential susceptibility factors (C.R. Clarke, S. Park, R. Tuosto, X. Jia, A. McGough, J.H. Van Mullekom, J.H. Westwood, unpublished results).

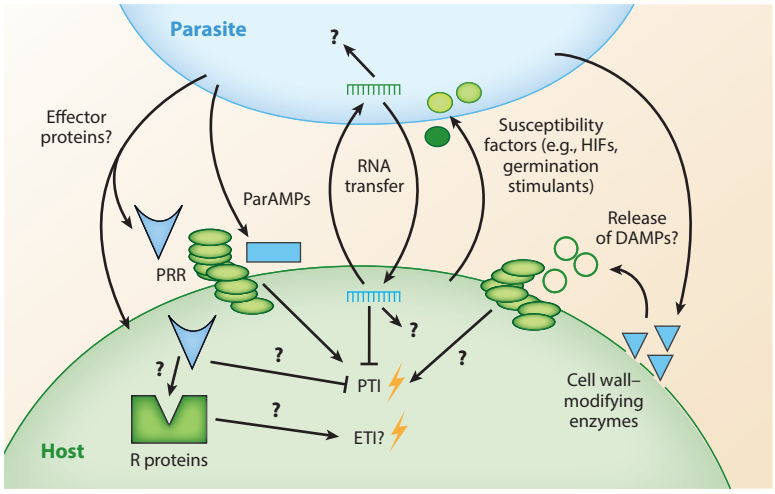

Figure 3

Parasitic plant interactions with host defenses. Parasite-derived components are shaded light blue, and host plant–derived components are shaded green. Components of the model that have not been validated in parasitic plants are indicated with question marks. Abbreviations: DAMPs, damage-associated molecular patterns; ETI, effector-triggered immunity; HIFs, haustoria-inducing factors; ParAMPs, parasite-associated molecular patterns; PRR, pattern-recognition receptor; PTI, pattern-triggered immunity.

An additional virulence strategy employed by some lineages of parasitic plants is the translocation of small interfering RNA molecules to alter host gene expression (see the section titled Parasite–Host Exchange of RNAs). For example, *C. campestris* transfers microRNA with sequence homology to *BIK1* (110), a conserved component of the plant immune system central to pattern-triggered immunity responses (76). Transfer of this microRNA is expected to reduce expression of *BIK1* in host plants and thereby attenuate the immune response of host plants. Although this virulence strategy is not broadly part of the molecular plant–microbe interactions paradigm, it is employed by the fungal pathogen *Botrytis cinerea*, which translocates several microRNA molecules into host plants to silence host immunity–related genes (131, 134).

Whether a host plant–parasitic plant interaction ultimately results in either a susceptible or resistant interaction likely depends on a balance of the various factors discussed above (65). Some paradigmatic host plant–microbial pathogen interaction mechanisms have already been elucidated for parasitic plant–host plant interactions, but others are in early stages of research or still unknown (**Figure 3**). A successful parasitic plant likely must evade or suppress pattern-triggered immunity induced by ParAMPs and DAMPs released during haustorial penetration, possibly through the secretion of immunity-interfering effector proteins. Additionally, if a parasitic plant secretes effectors into host plants that encode cognate *R* genes, then the interaction may become incompatible owing to effector-triggered immunity. A successful parasitic plant also needs to encounter a host that is actively producing the correct combination of susceptibility factors to induce and enable a successful haustorial connection and metabolic rewiring to meet its nutritional needs.

The extent to which these immunity-subversion strategies are deployed across the phylogeny of parasitic plants remains to be determined. Because parasitism independently evolved at least 12 times in parasitic plants, we hypothesize that different clades of parasitic plants likely employ divergent strategies to suppress or otherwise evade host plant immunity. Elucidation of the conserved and lineage-specific molecular tools employed by parasitic plants to subvert host plant immunity may enable the development of novel agricultural control strategies.

CHALLENGES TO AND FUTURE OUTLOOK FOR PARASITIC PLANT RESEARCH

The study of parasitic weeds and the underlying genetic factors that govern post-attachment resistance responses lags behind progress in understanding other plant–pathogen interactions. The slower rate of research progress with parasitic plants is partially due to (*a*) the inherent problems that exist with growing and manipulating parasites free of hosts, (*b*) the fact that both the host and parasite are angiosperms, (*c*) the relatively limited and only recently available high-quality parasitic plant genomes and transcriptomes, and (*d*) the difficulty or impossibility of transforming most species of parasitic plants. As more omic data sets and functional genomics tools, such as methods for transformation, become available for parasitic plants, research progress will accelerate.

Despite these challenges, the outlook for research on parasitic plant–host plant interactions is promising. Parasitic plants are likely to provide examples of novel variations on familiar pathogenesis mechanisms such as the discovery of parasitic plant–derived microRNAs that target host genes. Knowing how widespread this *trans*-species microRNA delivery is in *Cuscuta* and other parasitic plant lineages will be interesting. Because the functionally relevant exchange of small RNAs between fungal pathogens and plant hosts has been described in several systems (17), this delivery system could also be more widespread. The available genome assemblies from *C. campestris* (110, 130) and *Cuscuta australis* (116) will facilitate further studies.

Another pressing issue is the molecular mechanism by which *C. campestris* microRNAs are delivered to host cells. Parasitic plants may have direct connections to host cells, including joint plasmodesmata (12, 128), but other mechanisms are possible. One attractive hypothesis is that they are delivered via extracellular vesicles, which are enriched in proteins involved in pathogen defense and stimulated by pathogen invasion (106), and have been shown to carry small RNAs that can be delivered to the fungal pathogen *B. cinerea* and target *Botrytis* mRNAs (18). Considering these pathways for exchange and the documented transfer of mRNA and proteins (12, 72, 73), we hypothesize that effectors may move as gene transcripts or as small soluble proteins that do not need to fit profiles of canonical secreted effectors. Thus, given current knowledge, parasitic plants may have solved the same essential problems solved by other pathogens. Understanding the precise mechanisms by which host defenses have been defeated and host resources redirected will add additional perspectives to the diversity of plant pathology.

SUMMARY POINTS

1. Research on parasitic plants has surged in recent years, describing how different lineages of parasitic plants have evolved to detect and parasitize their hosts.
2. Strigolactone receptors and their signaling mechanisms have been identified, and their role in parasite identification of hosts is being elucidated.
3. Development of the haustorium and its interactions with the host plant are becoming exciting areas of research, and new insights have emerged into its role in the exchange of hormones, nutrients, and macromolecules, including RNAs.
4. Transfer of mRNA and microRNA between host and parasite appears to be an important virulence and host adaptation strategy in *Cuscuta*, although the mechanism regulating mobility is currently unknown.
5. Parallels are emerging between the molecular mechanisms mediating parasitic plant–host interactions and other plant–pathogen interactions, including the elicitation of host innate immunity.

FUTURE ISSUES

1. Sequencing of additional parasitic plant transcriptomes and genomes will lead to better characterization of the evolution and mechanisms of parasitism.
2. The processes by which germination factors and haustoria-inducing factors (HIFs) are secreted from host roots as well as their fate in the microbial-rich rhizosphere need to be better understood.
3. To clarify the importance of this novel virulence and adaptation strategy, researchers need to determine whether the transfer of microRNA and mRNA enhances parasite fitness and whether it is restricted to *Cuscuta* or widespread in parasitic species.
4. It is important to determine which damage- and parasite-associated molecular patterns as well as other elicitors of host plant immunity are present during host–parasite interactions and during which stages of parasitism the elicitors are present.
5. The presence and function of putative parasitic plant effector proteins should be studied across a wide range of parasitic plants to elucidate their host targets and the pathways they disrupt.
6. Identification of additional host plant signaling pathways on which parasitic plants rely for successful perturbation of host plant development (susceptibility factors) may be a valuable tool for increasing resistance in host plants. Host plant–derived germination stimulants and HIFs may also function as susceptibility factors altering control of parasites.
7. A critical issue—as yet unrealized—is to translate our gains in fundamental knowledge of parasitism toward deployment of effective parasitic weed control strategies.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

The authors acknowledge support from the US Department of Agriculture National Institute of Food and Agriculture awards 2015-67012-22821 (C.R.C.), 2018-05102 (M.J.A. and J.H.W.), and 135997 (J.H.W.), as well as from US National Science Foundation award 1238057 (J.H.W., M.P.T., and J.I.Y.).

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