Parasitic plant responses to host plant signals: a model for subterranean plant–plant interactions

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The ability of plants to fulfill nutritional needs by parasitizing neighboring plants has originated several times in angiosperm evolution. Molecular tools are now being exploited to investigate the evolutionary origins of plant parasitism and to dissect the genetic mechanisms governing parasitic plant–host plant interactions. Investigating the nature of signal exchanges between parasitic plants and their hosts serves as a tractable system for understanding how plants in general communicate in the environment. This work should also lead to the development of novel strategies for minimizing the devastation caused by parasitic weeds in international agriculture.

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Introduction

Parasitic plants have intrigued plant scientists since the first description over 175 years ago of *Rafflesia arnoldii*, a bizarre, almost leafless tropical plant comprised of little more than the world's biggest flower. About 4000 species in 22 dicot families are currently recognized as parasitic [1•]. Parasitic plants can be placed into eleven independent phylogenetic clades, indicating that parasitism originated several times during the evolution of angiosperms [1•].

Parasitic species take different forms and invade host plants via alternative routes. Some parasites, like mistletoes and dodders, invade aerial parts of the host while other parasites invade underground roots. Parasitic plants also vary in the degree to which they rely on host resources, with some being completely dependent on host plant resources and others completing their life cycles autotrophically. In all cases, the parasite obtains at least some nutritional benefit by robbing water, carbohydrates, and minerals from the host. The effect on the host plant can be dramatic and lead to the weakening, deformation, and eventual death of the host. Indeed, parasitic plants are noxious agricultural pests in diverse agricultural settings. Dwarf mistletoe (*Arceuthobium* ssp.), for example, is the most damaging disease in conifer forests throughout the western United States and causes lumber losses of over 50% in infested forests [2]. Even more significant are the root parasites *Striga* and *Orobanche*. These parasitic weeds are particularly insidious in developing countries, notably Africa, where they cause devastating yield losses in maize, sorghum, grain legumes and other staple crops. Much of the current parasitic plant research is focused on developing control procedures and resistant germplasm against these agricultural pests.

This review is focused on root parasites in the Scrophulariaceae and closely related Orobanchaceae. These parasites directly invade host roots via haustoria — specialized invasive organs unique to parasitic plants. The classic reference to these and other parasitic plants is the beautifully illustrated book by Job Kuijt [3]. More recent reviews cover the taxonomy of parasitic plants [1•], the interactions between parasitic plants and their hosts [4–7] and the role of parasitic weeds in agriculture [2]. This review will discuss three areas of plant parasitism: the molecular phylogeny of parasitic plants and evolutionary fate of chloroplast genomes in non-photosynthetic plants; parasitic plant recognition and response to host plant signals; and the response of host plants to invading parasitic plants.

Origin and evolution of parasitic Scrophulariaceae

Scrophulariaceae is a large family of plants that includes the well studied genera *Antirrhinum* and *Mimulus*. Although typical Scrophulariaceae are not parasitic, about a third of the genera obtain at least some of their nutritional resources by parasitizing the roots of neighboring plants [1•]. Scrophulariaceae is an interesting family for evolutionary studies because extant genera represent all nutritional modes of plant parasitism including achlorophyllous holoparasites (*Orobanche*), photosynthetic, obligate hemiparasites (*Striga*), photosynthetic, facultative parasites (*Triphysaria* and *Agalinis*), and full autotrophs (*Antirrhinum* and *Mimulus*). Phylogenetic reconstructions based on sequence comparisons of the *rps2*, *matK* and *rbcL* plastid genes place all parasitic Scrophulariaceae and Orobanchaceae on a single, monophyletic clade [8••,9•]. This indicates a single evolutionary origin of parasitism prior to the divergence of these families.

The first parasitic plants were photosynthetically competent, facultative parasites. Photosynthesis was subsequently lost in several lineages, resulting in at least five distinct clades of parasitic Scrophulariaceae are achlorophyllous [8••]. The remnant plastid genomes in these species are marked by large deletions. For example, *Epifagus virginiana* has a plastid genome just one third the size of that in tobacco [10]. This dramatic reduction is a consequence of hundreds or thousands of small, independent deletions. The order of remnant genes is, however, the same as in intact chloroplasts.

The deletions in *E. virginiana* are heavily biased for regions containing photosynthetic and chlororespiratory genes [10].

The intact plastid genes function in gene expression and encode ribosomal RNAs, ribosomal proteins, and transfer RNAs; however, even genes in these classes are deleted — only 15 of the 21 ribosomal protein genes, and 17 of the 30 tRNA genes present in tobacco plastid DNA are intact in *E. virginiana*. Also, all four plastid encoded RNA polymerase subunits are missing in *E. virginiana*. Because *E. virginiana* plastid genomes are transcribed and translated, these functions must be fulfilled by nuclear components [11,12].

Some plastid genes in achlorophyllous parasites serve functions distinct from photosynthesis. The *rbcL* gene, encoding the large subunit of RuBisCO, is found either intact or as a pseudogene in most parasitic Scrophulariaceae [13]. RuBisCO is weakly functional in *Lathraea clandestina*; the weak expression resulting from both a low level transcription and an accumulation of point mutations [9•,14]. Also, nine of the intact tRNA genes in *Epifagus* are more conserved in *Orobanche minor* than are intervening sequences, suggesting that these genes are functional and maintained by natural selection [12].

In conclusion, the origin and subsequent evolution of parasitism in the Scrophulariaceae was accompanied by rapid and significant losses in chloroplast genomes. It seems likely that at least some nuclear encoded functions have been lost in these species as well. The origin of plant parasitism, however, was also marked by the acquisition of new genetic traits and developmental programs not found in autotrophic species. Some of these acquired traits are discussed below.

Parasitic plant responses to host plant signals

Germination

The identification of appropriate host roots is most critical for obligate parasites like *Striga* and *Orobanche* and host recognition systems are most advanced in these genera. Because these plants must attach to a host within days after germination in order to survive, the critical decision point is seed germination. Consequently, these parasites recognize specific molecules released from host roots as germination cues [15–17].

The first germination stimulant identified for *Striga* seeds was the tetracyclic sesquiterpene called strigol [18]. Several structurally related synthetic analogues of strigol, commonly referred to as GR compounds because of their growth regulating properties, were later identified as germination stimulants for both *Striga* and *Orobanche* [19]. Ironically, strigol was first isolated from cotton, itself a non-host for *Striga*. Later, similarly structured molecules were isolated from sorghum, a true host for *Striga*, where they were recovered from minor fractions [15].

Strigol and its structural relatives are considerably more stable than natural germination stimulants, raising the question as to the relevance of these molecules in nature [16]. The major *Striga* germination stimulants released from sorghum are 2-hydroxy-5-methoxy-3-[(8',11'Z)-8'Z,11',14'-pentadecatriene]-*p*-hydroquinone and three other structurally related but less abundant dihydroquinones [17,20]. The hydroquinones, which stimulate germination, are readily oxidized to their more stable and abundant benzoquinone analogs that do not stimulate germination [21]. A related compound, resorcinol, retards the oxidation process and reduces the effective concentration of root exudate needed for *Striga* seed germination [22]. *Striga*'s strategy of using unstable molecules as germination stimulants provides a means of ensuring that appropriate host roots are within reach before germinating.

Haustorium induction and early development

Another class of signal molecule(s) released by host roots initiate the development of haustoria, novel root structures that invade the host and subsequently act as the conduit through which host resources are redirected to the parasite. Investigations into haustorium development have been greatly facilitated by the early observations that these structures can be induced *in vitro* by applying host root exudates to the roots of aseptically grown parasites [23]. Although there are some differences in detail, the overall ontogeny of early haustorium development in response to host factors is similar for all Scrophulariaceae [24]. Within a few hours after exposure to host exudates, a localized swelling near the parasite root tips can be observed. The swelling is initially caused by a rounding and isodiametric expansion of cortical cells, later new divisions also contribute to the swollen phenotype. Concomitantly, there is a proliferation of haustorial hairs overlying the swollen zone. These hairs are physically distinct from typical root hairs in that papillae rich in hemicellulose coat their surfaces. The biological significance of these papillae is that they bond the hairs to cells on the host surface, thereby functioning to attach the haustoria to the host roots [25,26]. The globular haustorium is competent to attach to host tissue within 24 hours.

Haustoria are classically distinguished as either primary or secondary, depending on whether they are terminally or laterally localized on the roots respectively [3]. In all parasitic Scrophulariaceae, the cells that are most sensitive and initially responsive to host factors are near the root meristem ([27]; J Yoder, unpublished data). When *Striga* radicles are incubated *in vitro* in the continued presence of haustorial inducing factors — a condition met in nature when the radicle contacts a host root — haustorium development is determinant and the resulting haustorium is terminally localized (primary). In contrast, when *Agalinis* or *Triphysaria* are incubated under these conditions, root tip cells initially respond but soon revert to normal root development; the resultant haustoria become laterally positioned behind the root tip (secondary) [27,28]. Interestingly, *Striga* haustoria become similarly positioned by washing the inducer from the roots [29]. The distinction between primary and secondary haustoria, therefore, rests on whether sensitivity to the continued presence of haustoria inducers is transitory or determinant. Elucidation

of these mechanisms may be important for designing novel host resistances.

Although previous 2-D PAGE studies showed that protein profiles change during early stages of haustorium development [30,31], changes in transcriptional levels are only now being explored. My lab has used subtractive hybridization to recover hundreds of cDNAs that are differentially abundant in *Triphysaria* root tips soon after haustorium induction (M Matvienko and JI Yoder, unpublished data). Most of these transcripts increase about 2–10 fold upon exposure to host root exudates, but a few increase transcription levels by several orders of magnitude. Although the role of these genes in mediated plant parasitism is not yet clear, the structure and expression pattern of some suggest roles in host signal recognition and transduction. In any case, these represent a unique class of plant genes whose transcription is controlled by the presence of neighbouring plants. The promoters driving these genes may be useful for engineering novel weed management strategies.

Parasite recognition of haustorial inducing factors

A diverse array of natural and synthetic quinones, hydroxy acids, and flavonoids have been identified as haustorial inducing factors [28,32–34]. Several experiments suggest that hydroxy acids are inactive until enzymatically converted to the analogous quinones [35]. *Striga* seedlings need to be exposed longer, and at higher concentrations, to hydroxy acids than do similarly substituted quinones. Furthermore, HPLC analysis shows that syringic acid is oxidized to benzoquinone in the presence of root cells with kinetics that mirror that of haustorium development. Apoplastic oxidases that convert syringic acid to the haustoria inducer 2,6-dimethoxybenzoquinone (DMBQ) have been extracted from *Striga* roots [36••]. Because similar peroxidases were also identified in host plant roots, it was not possible to conclude which plant partner encodes the responsible enzymes. The authors suggested that *Striga* supplies the hydrogen peroxide required for the reaction [36••]. As hydrogen peroxide is also released from most plants under various stress conditions, however, it is unlikely to be a parasite specific component [37]. Interestingly, anthocyanidins that are active haustoria inducers exist in multiple tautomeric forms, some of which contain quinone structures which might be the active components [28]. Enzymatic conversion to active quinones, therefore, may not always be essential.

Structural considerations alone have been insufficient to identify critical determinants of haustoria inducers. On the basis of the observation that active quinones have redox potentials that fall within a narrow window whereas inactive quinones have redox potentials outside this window, it is likely that activity is a redox function [38]. The importance of oxidation–reduction reactions for haustoria induction was further supported by developing specific inhibitors based on proposed semiquinone intermediates [39].

The molecules that induce haustoria are common components of plant cell walls and generally abundant in plant roots. This is consistent with haustorium development being rather promiscuous, even in parasites with narrowly defined host ranges. In this light it is interesting that parasitic plants do not typically form haustoria on their own roots, or on those of closely related parasites [40••]. Although the evolutionary advantages of not invading related parasites are easy to rationalize, the mechanisms of vegetative self-recognition are not known. The elucidation of these mechanisms may afford insights into host resistance design.

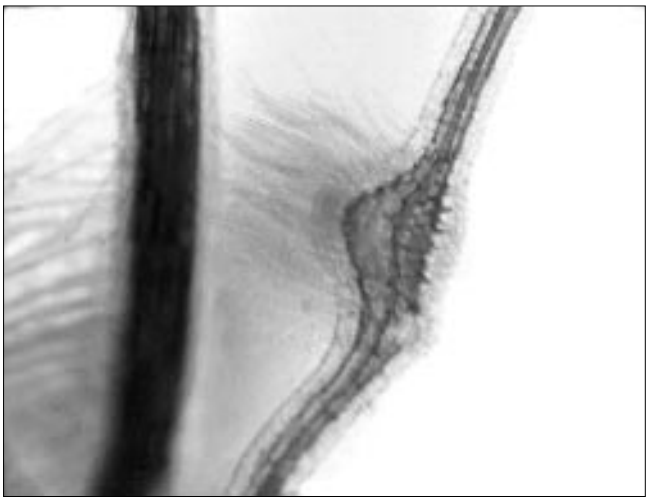
Host invasion and haustorium maturation

Haustoria attachment is non-specific and parasites will attach onto non-host plants as well as inert substrates. Host penetration occurs when haustorial cells at the parasite–host interface elongate and divide, pushing through the epidermis and cortex of the host root. The lack of cell wall disruption at the invasion site suggests the penetration peg pushes between rather than through the host cells [41,42].

It is generally assumed that cell wall degrading enzymes, either parasite or host encoded, assist in the penetration process, though there is little direct evidence of their role. Pectin methyl esterases have been detected cytologically in endophytic *Orobanche* haustoria [43•]. These authors also show changes to host cell wall pectins at the site of haustorium invasion. This is consistent with the earlier detection of pectin methyl esterase activity in growth media of *in vitro* cultured *Orobanche* [44]. Further such studies are needed to distinguish the relative roles of enzymatic digestion and mechanical force in haustorium penetration.

Advancement of the penetration peg into the host cortex is remarkably responsive to host-specific factors. When *Striga* seedlings are placed on sorghum roots, the host cortex is transversed within 48 to 72 hours. In contrast, penetration of the cortex of a non-host plant like marigold is prematurely terminated and rarely reaches the endodermis [45••]. Haustorial cells at the interface are necrotic and have degraded cell walls, suggesting that nonhost plants produce factors cytotoxic to the invading haustorium. Host cells adjacent to the endophyte are also necrotic with increased intracellular wall appositions. Host necrosis is also observed in a resistant line of cowpea (*Vigna unguiculata*) near the site of *Striga gesnerioides* penetration [46]. Several genes differentially expressed in Marigold during aborted *Striga* invasions have been recently cloned and at least one of these has homology to the Toll/interleukin receptor portion of the disease resistance genes *RPP5*, *N*, *L6* and *Mi* (BS Gowda, JL Riopel, MP Timko, personal communication). If this gene proves to be a causative agent of nonhost resistance, it will further extend the broad spectrum of plant pathogens controlled by these genes.

Figure 1



Haustorium development in response to *Bromus* root. Single *Bromus carinatus* and *Triphysaria pusilla* seedlings were grown together in a single well of a 24 well plate in agar media. A secondary haustorium is developing on the root of *T. pusilla* in response to molecular signals released from the *Bromus* root. The polar development of haustorial hairs towards the host, and the asymmetric swelling of the root, can be easily seen on the *T. pusilla* root on the right.

Once the haustorium has reached the host stele, haustorial cells at the interface penetrate host vessel members through their pits. These cells then open at their tips and loose their cytoplasm [47•]. Adjacent cortical cells progressively differentiate into xylem elements until a continuous water conducting system is established linking the host and parasite vascular systems [24,26]. The development of the xylem bridge is absolutely dependent upon direct contact of the haustorium with the host stele [23,40••]. The nature of the host signal(s) that triggers xylem differentiation is currently uncharacterized but the phytohormones auxin and cytokinin are good candidates since these are known to trigger vascular regeneration in wounded tissues [48].

Host responses to parasite invasion

Because of the agricultural devastation caused by parasitic weeds worldwide, there is considerable motivation to identify and exploit host resistances. The most common resistance mechanisms are those in which the hosts lack factors needed by the parasite, particularly germination stimulants [2]. The majority of resistance factors are not simply inherited nor are they particularly robust [49]. With a few exceptions, the incorporation of host resistance against parasitic weeds into acceptable cultivars has been disappointing.

Transgenic strategies for engineering dominant resistance, such as driving lethal gene functions by host promoters specifically induced by parasites, are being attempted. Some potentially useful promoters have been identified using GUS reporter genes. The promoter driving transcription of 3-hydroxy-3-methylglutaryl CoA reductase (*hmg2*) is activated in transgenic tobacco within one day of penetration of *Orobanche* [50••]. Expression of the *hmg2* promoter is centralized around the penetration point and extends into the host cortical and vascular tissues. Similarly, the promoter driving transcription of the pathogenesis related protein PR-1 is induced by *Orobanche* infection [51]. This demonstrates that hosts initiate different defense genes, but without apparent benefit.

Conclusions

Parasitic plants trigger novel developmental processes in response to host plant signals. The robustness and synchrony of these responses *in vitro* affords an excellent system for investigating plant–plant interactions. Several lines of evidence suggest that oxidation–reduction plays a critical role in triggering different developmental processes critical to the parasitic life style. It will be interesting to learn how general these mechanisms are in plant development.

Parasitic plants debilitate and kill neighboring plants. Because parasitic plants have independently originated from non-parasites, multiple times, it might be argued that relatively few genetic changes are required to confer parasitism. Identifying the genes controlling plant parasitism should help answer these questions and support the development of control strategies. In addition, it might be possible to incorporate the parasite genes themselves into crop plants; this would enable crops to biologically reduce the growth of unwanted vegetation in their vicinity. Using modern approaches, it should be possible to turn the tables and exploit the genes that make parasitic weeds so destructive for the benefit of agriculture.

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