Host-plant recognition by parasitic *Scrophulariaceae*

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Parasitic plants in the *Scrophulariaceae* invade the roots of neighboring plants in order to rob them of water and nutrients. A distinctive feature of these parasites is their ability to cue their development to small molecules released by host-plant roots. Evidence is continuing to emerge that parasite perception of host factors occurs via a redox-associated mechanism. Genes predicted to function during the early stages of parasite–host interactions have been cloned from both plant partners, and their characterization is providing a genetic framework on which to model subterranean plant–plant interactions.

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Abbreviations
CPBQ cyclopropyl-*p*-benzoquinone
DMBQ 2,6-dimethoxybenzoquinone
SXSg sorghum xenognosin for *Striga* germination

Introduction

Parasitic plants fill at least some of their nutritional requirements by robbing other plants. The effects of such parasitism on host plants can be debilitating, and some of the world's most pernicious agricultural pests are parasitic weeds [1]. Job Kuijt beautifully illustrates the unique biology of parasitic plants in his now classic monograph '*The Biology of Parasitic Flowering Plants*' [2], whereas more recent information is well covered in the book '*Parasitic Plants*' [3]. An electronic gateway to the diversity of parasitic plants, their habits, and the people who study them, can be found on the '*Parasitic Plant Connection*' website, which is maintained by Dan Nickrent [4].

The parasitism of plants by other plants provides an exceptional opportunity for investigating chemical cross-talk between neighboring plants, a phenomenon traditionally termed allelopathy [5]. Parasitic genera in the *Scrophulariaceae* use secondary metabolites released by host-plant roots to signal developmental programs that are critical for heterotrophic growth. The release of recognition molecules from host roots and their identification by parasitic plants has recently been reviewed [6••]. This review is focused on recent progress in defining the molecular genetic mechanisms associated with host recognition by parasitic *Scrophulariaceae*.

Parasitic *Scrophulariaceae*

It is estimated that over 3000 species of angiosperms can parasitize other plants [4]. Parasitic plants are found throughout the world and encompass a huge diversity of ecological niches, growth habits, and host associations. The degree to which parasites rely on host resources varies from non-photosynthetic holoparasites, which are completely dependent upon host resources, to photosynthetically competent facultative hemiparasites. The one commonality among parasitic angiosperms is their ability to penetrate neighboring plant tissues and to acquire water, carbohydrates, and mineral nutrients from their hosts through invasive structures called haustoria. Because angiosperm haustoria function in host attachment, penetration, and nutrient translocation, they fulfill functions attributed to both appressoria and haustoria in fungal pathogens.

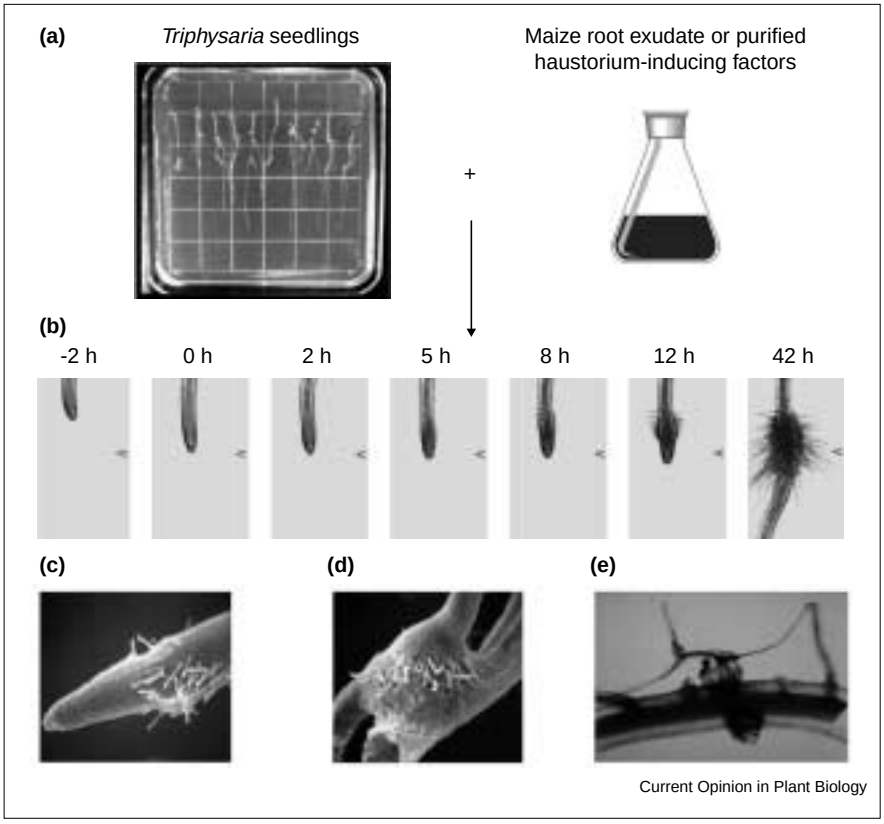
Phylogenetic analyses of parasitic and non-parasitic genera have established two important facts about the evolutionary origin of parasitism in angiosperms. First, parasitic plants are derived from non-parasitic ancestors, and second, parasitism has originated multiple times in the evolution of angiosperms [2,7]. These conclusions lead to the hypothesis that the developmental mechanisms that distinguish parasitic from non-parasitic plants are readily evolved. Because the haustorium is 'the organ that embodies the very idea of parasitism' [2], its developmental control is an obvious candidate for involving genetic mechanisms that defines plant parasitism.

The *Scrophulariaceae* is a large family of plants, the vast majority of which are non-parasitic autotrophs. About thirty genera of *Scrophulariaceae* are, however, competent to develop haustoria and parasitize neighboring plant roots. Included among the parasitic *Scrophulariaceae* are the devastating agricultural weeds *Striga* and *Orobanche* [8]. Phylogenetic analyses of chloroplast gene sequences place all of the parasitic members of the *Scrophulariaceae* and *Orobanchaceae* in a single clade that is distinct from the non-parasites, indicating that the genetic pathway for haustorium development originated once in the evolutionary history of these families [9]. The loss of photosynthetic competence and associated purging of chloroplast genome sequences occurred in subsequent independent events [10,11].

Host preference and selection

The total host range of all parasitic plants is large and most plants are susceptible to one or more parasites. Nevertheless, the host range of any given parasitic species can be quite specific. Host specificity as measured in natural populations is an overall consequence of the ability of a parasite to recognize and attack a particular host plant, the defense responses of the host plant, and the suitability of host resources for optimal parasite growth.

Figure 1



Haustorium development in response to host root factors. **(a)** The monitoring of haustorium development *in vitro* by exposing aseptically grown *Triphysaria* seedlings to maize root exudates or purified haustorium-inducing molecules. **(b)** Haustorium ontogeny in a single *Triphysaria* root tip that has been exposed to exudate. The exudate was added at 0 h, and the arrows to the right of each photograph mark the position of the meristem at this time. Manifestations of haustorium development include an almost immediate cessation of root-tip elongation (compare –2 h, 0 h and 2 h), haustorial hair elongation, cortical swelling, and reversion to normal root-tip growth, all within 12 h of exposure. A time-lapse animation of these events can be found at URL http://veghome.ucdavis.edu/Faculty/Yoder/Lab/index.html. **(c)** The initiation of haustorial hairs from epidermal cells. **(d)** The attachment of a *Triphysaria* root (top) to a host root (bottom). **(e)** A mature *Triphysaria* haustorium attached to a maize root. The roots were cleared in NaOH to visualize vascular connections [50]. (e) is copyrighted by the American Society of Plant Physiologists and reprinted with permission.

The recognition of host tissues by parasitic plants will be discussed in the next section. Host defense against plant parasitism has received considerable attention given the agricultural significance of parasitic weeds. Histological comparisons of *Striga* parasitizing host, *Sorghum*, and non-host, marigold, roots show that the penetration of non-host tissues terminates in the outer cortex [12]. Subsequent degeneration of the distal-most *Striga* cells suggests the action of active necrosis factors. A transcript encoding a protein similar to the tobacco *N* gene product is upregulated in marigold roots during *Striga* invasion [13••]. This gene is predicted to encode a protein sharing six regions of sequence homology with the cytoplasmic domains of the *Drosophila* Toll and mammalian Interleukin-1 receptor genes. The protein does not, however, have homology to the nucleotide-binding site (NBS), leucine-rich repeat (LRR) motifs, or the serine/threonine kinase domains that are typical of other resistance-related genes. Plant responses to parasitic invasion are also being investigated using reporter genes to monitor transcription from pathogenesis-related promoters [14,15].

Field and pot studies have repeatedly demonstrated the differential performance of parasites on certain hosts [16]. The uptake of host metabolites by parasitic plants is apparently non-discriminatory, and most types of molecules are translocated from host to parasite. Biologically active secondary metabolites that are acquired by the parasite will affect its ecological interactions in natural environments. For example, when the hemiparasite *Castilleja* is grown on near-isogenic lines of *Lupinus albus* that differ primarily in quinolizidine alkaloid content, parasites grown with high-alkaloid lines had decreased herbivory damage, increased visitations by pollinators, and increased lifetime seed production relative to parasites grown with low-alkaloid lines [17•]. The incorporation of host resources into parasitic plants broadens their ecological adaptability beyond that encoded by their own genomes.

Xenognostic molecules

Parasitic *Scrophulariaceae* sense their prey through the recognition of secondary metabolites released by host roots. Host recognition factors, termed xenognosins, activate developmental programs in parasitic plants [18]. Xenognostic molecules are a subset of allelopathic molecules that are released by one plant and which modify the growth and development of a second [5].

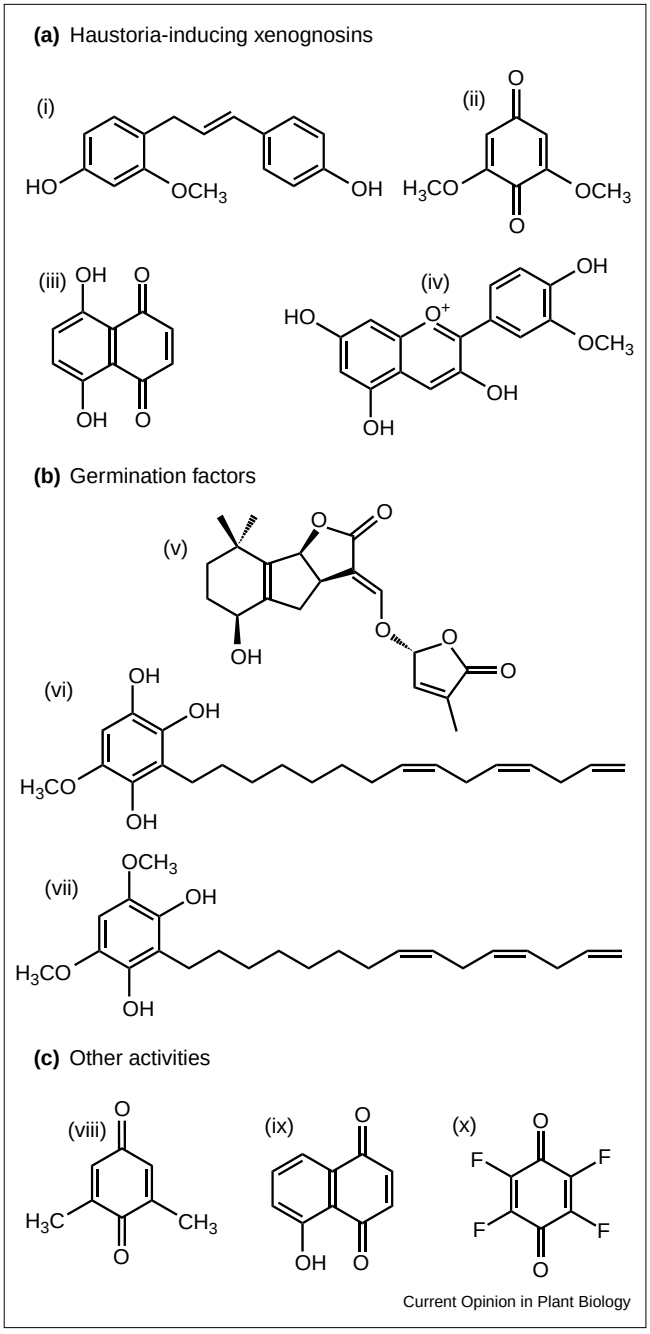
Xenognostic molecules trigger the transition from autotrophic to heterotrophic growth by signaling haustorium development. It has been known for several years that host-root factors stimulate haustorium development when applied to parasite roots *in vitro* [19,20]. Figure 1 illustrates haustorium development in roots of the hemiparasitic plant *Triphysaria* that have been exposed to root exudates

recovered from hydroponically grown maize. The morphological changes that take place are rapid and highly synchronous. The first observation is an almost immediate cessation of root tip elongation. In *Striga*, this period is hallmarked by a cessation of DNA synthesis [6••]. Within four to five hours of exposure, epidermal cells begin to elongate and form hairs that proliferate just behind the root tip; these hairs function to attach the developing haustorium to host tissues [21,22] (Figure 1d). Also during this period, cortical cells underlying the proliferating epidermal hairs enlarge, producing a swollen region just behind the parasite root tip. This swelling and hair proliferation continues for about 20 h during which time the root tip reverts to its typical growth program, resulting in a normal root growing distal to the haustorium. In contrast, the primary haustoria of *Striga* and *Orobanche* are terminal differentiations that typically do not revert to normal growth unless the inducer is removed.

The first natural inducers of haustorium development that were identified were xenognosin A (Figure 2i), xenognosin B, and 2,6-dimethoxybenzoquinone (DMBQ) (Figure 2ii) [18,23,24]. These results prompted the *in vitro* assay of a variety of related molecules and the subsequent findings that various phenolic acids, quinones, and flavonoids are active inducers (Figure 2) [25–27]. There is a redundancy of haustoria-inducing factors in host root exudates. Genetically defined lines of *Triphysaria pusilla* have been isolated that have differential responses to DMBQ, some make haustoria whereas others do not [28•]. Both DMBQ-responding and non-responding lines make haustoria in response to total host root exudates, however, indicating that multiple factors can be recognized in host-root exudates by different parasites.

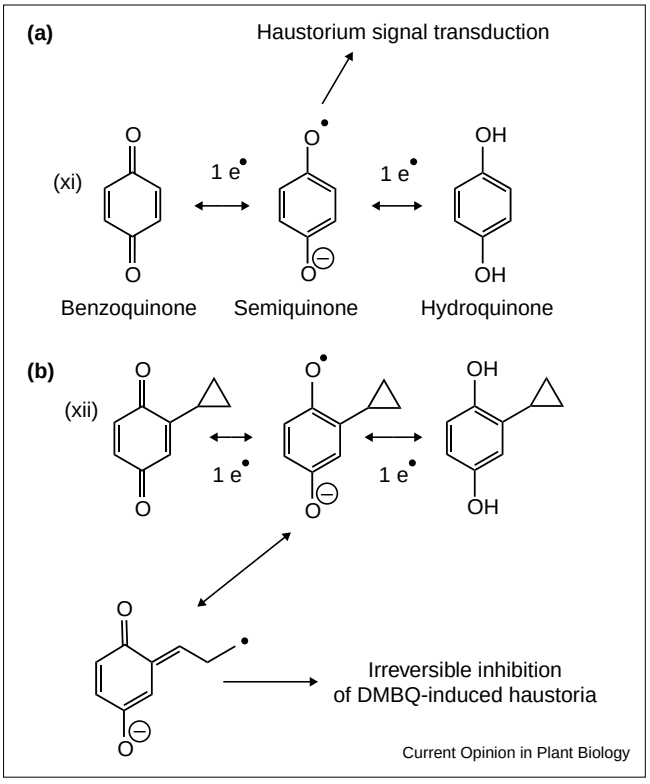
A mechanistic model to account for the xenognostic activity of diverse phenols and quinones has been developed over the years in David Lynn's group [6••]. The core molecule that induces haustoria is 1,4-benzoquinone (Figure 3xi). Certain structural alterations are tolerated; for example, dimethoxy (Figure 2ii) but not dialkyl (Figure 2viii) substitutions are tolerated. A more striking feature of haustoria-inducing quinones, however, is that their electrical potentials fall within a relatively narrow redox window [27]. Molecules with redox potential near the ends of the range (Figure 2iii) are partially active, whereas molecules outside this redox window are inactive (Figure 2ix,x). The inactivity of reduced quinones (hydroquinones) points to the mechanism of reduction as critical. The observation that tetrafluorobenzoquinone (Figure 2x), a molecule that is easily reduced but not reoxidized within the requisite window, is a reversible inhibitor of DMBQ led to the proposal that xenognosin perception requires repetitive redox cycling. The significance of the radical intermediates generated during redox cycling was supported by the irreversible inhibition of DMBQ by cyclopropyl-*p*-benzoquinone (CPBQ) (Figure 3xii). The electron-rich semiquinone formed by univalent reduction of CPBQ opens the cyclopropyl moiety, which then irreversibly binds to or inactivates the parasite xenognosin receptor [29] (Figure 3b). These experiments make a

Figure 2



Xenognostic and phytotoxic allelopathic quinines. **(a)** Some of the haustoria-inducing xenognosins discussed in the review are (i) xenognosin A [18], (ii) DMBQ [24], (iii) 5,7-dihydroxynapthoquinone [27], and (iv) peonidin [26]. **(b)** Host germination factors include (v) strigol [51], (vi) SXSg [32], and (vii) an antioxidant enhancer of SXSg [33]. **(c)** Other molecules discussed in the text are (viii) dialkyl benzoquinone, a quinone structurally related to DMBQ but inactive in haustorium formation [27], (ix) juglone, a phytotoxic allelochemical [35], and (x) tetrafluorobenzoquinone, a reversible DMBQ inhibitor [27].

Figure 3



Quinone reductions and radical intermediates. (a) The one-electron reduction of xenognostic quinones results in radical semiquinone intermediates that are predicted to trigger haustorium development [27]. (b) Evidence for the requisite semiquinone includes the irreversible inhibition of DMBQ by CPBQ [12], presumably through the opening of the semiquinone cyclopropyl ring [29].

strong case for the initiation of the haustorium signal transduction pathway through the recurring one-electron oxidoreduction of the xenognostic quinone to the semiquinone state (Figure 3a) [27]. There is also a potential for the use of the reactive CPBQ semiquinone intermediate to specifically bind and label the quinone receptor.

Obligate parasites that require host resources within days after germination have evolved astute host identification mechanisms that rely on host factors for germination [30]. Xenognostic molecules that function as germination stimulants include sesquiterpenes related to strigol (Figure 2v) and hydroquinones related to the sorghum xenognosin for *Striga* germination (SXSg) (Figure 2vi) [31,32]. The bioactivity of xenognostic hydroquinones is modulated by their oxidoreduction states, as evidenced by the identification of an antioxidant analog (Figure 2vii) that enhances SXSg activity [33]. Therefore, although host germination factors are structurally distinct from haustorium-inducing factors, the bioactivities of both are related to their oxidation potentials.

The importance of oxidoreduction mechanisms in ecological interactions in the rhizosphere has been discussed [34]. A relevant example in plant–plant signaling relates to the phytotoxicity of allelopathic quinones that are released by certain plants. It has been known for centuries that walnut trees secrete phytotoxic substances that poison the soil for other plants. The phytotoxic molecule was identified in the 1940s as the hydroxy-naphthoquinone juglone (Figure 2ix) [35]. Walnut trees do not synthesize juglone but rather the non-toxic reduced hydrojuglone, which is rapidly oxidized to the phytotoxin upon exposure to oxygen in the soil [36]. On the basis of numerous mechanistic studies of pharmacological quinones, it is likely that juglone toxicity is associated with reactive intermediates formed through redox transformations [37].

In conclusion, parasitic plants are able to use a variety of molecules as host recognition factors provided that they fulfill certain structural and electrochemical requirements. The redundancy of host molecules that can serve as xenognosins likely contributes to the overall broad host range of parasitic plants, as well as to difficulties in the identification of resistant germplasm. Reactive intermediates generated during univalent redox cycling are associated with both developmental signaling and phytotoxic allelopathic responses. It was proposed 25 years ago that parasitic plants use host defense chemicals, such as those that make plants unattractive to insect herbivores, as cues to stimulate germination and haustorium formation [38]. It would be an ironic twist if the xenognostic molecules used by parasites to identify host roots originally evolved in the hosts to deter the growth of neighboring plants.

Quinone signaling and leaky capacitors

The past year has seen the use of cloned genes to further investigate xenognosin recognition. A unigene set of approximately 140 early DMBQ-induced transcripts (EDIT) was assembled from a subtractive cDNA library enriched for *Triphysaria* root transcripts that were upregulated within five hours of exposure to DMBQ [39]. The assembly and clone sequences are cataloged on the website URL http://veghome.ucdavis.edu/Faculty/Yoder/Lab/index.html. Early stages in haustorium formation involve changes in cell size and shape, and several genes encoding proteins associated with cell-wall structure are differentially regulated during this period [39]. Transcripts predicted to encode expansin proteins have been cloned from *Striga*, and two of these are upregulated in DMBQ-treated radicals with a kinetics that mirrors that of haustorium ontogeny [40••]. Transcript accumulation is also correlated with xenognosin specificity, so these sequences are useful as molecular markers to monitor early stages in xenognosin recognition and processing.

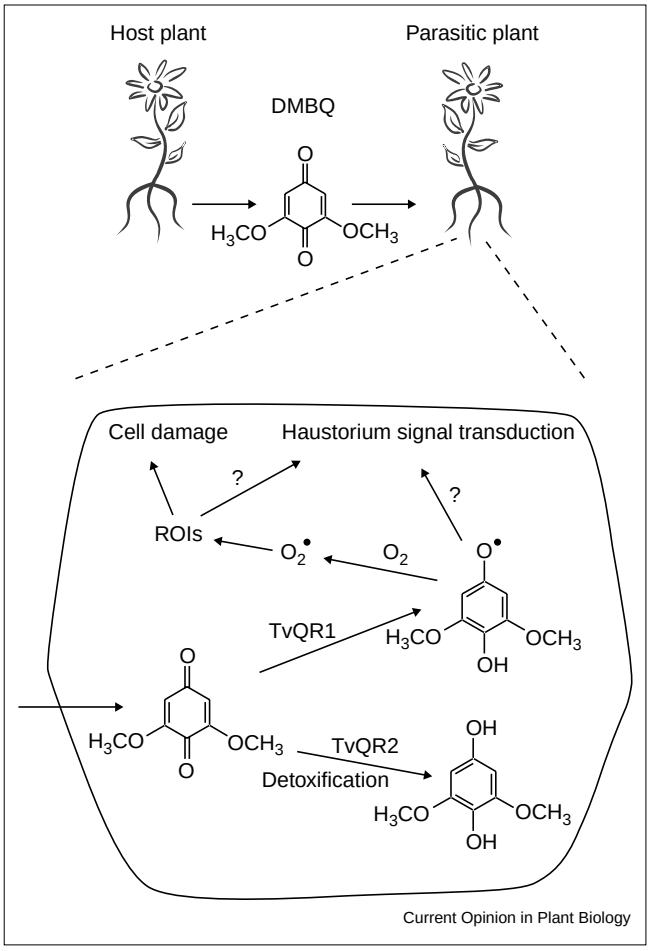
These experiments follow up earlier studies demonstrating that xenognosin exposure times of several hours are required for irreversible commitment to haustorium development [24,41]. The tips of *Striga* radicals enlarge linearly over time when exposed to DMBQ, but if DMBQ is removed prior to the required exposure time, further

development ceases. The requisite exposure period is dependent upon xenognosin concentration with greater DMBQ concentrations requiring shorter exposure times. These results are interpreted to mean that unstable factor(s) produced in response to DMBQ accumulate over time and must reach a threshold level before the parasite irreversibly commits to haustorium development. The mechanism of haustorium induction is likened to that of a leaky capacitor, with the 'charge' being some component that accumulates in the parasite upon continued exposure to xenognosin [40••]. These studies provide an excellent working model for xenognosin recognition, and also evoke a more generalized regulatory mechanism that may function in other signaling systems in which spatial parameters are important.

Parasite genes associated with the oxidoreduction of allelopathic quinones

Lynn's model [6••] predicts that an unstable factor accumulates to a threshold level before developmental commitment occurs. Reactive semiquinones generated during xenognosin reduction are likely candidates for the signaling charge, and so recent investigative efforts are directed towards characterizing quinone reductases in parasitic plants. Two transcripts encoding distinct quinone reductases are regulated by xenognosins in *Triphysaria* root tips [42••]. One transcript, TvQR1, encodes a protein that is related to a family of NAD(P)H-dependent quinone reductases. The best-characterized members of this family are the zeta-crystallins, which catalyze single-electron reductions [43]. An *Arabidopsis* zeta-crystallin, P1-ZCr, catalyzes ferricytochrome c reduction and confers enhanced tolerance to diamide, a thiol-oxidizing agent in transgenic yeast [44,45]. A second transcript, TvQR2, belongs to a family of quinone oxidoreductases, exemplified by the carcinogen detoxification enzyme DT-diaphorase, that protect cells from radical damage by reducing xenobiotics via a two-electron mechanism that avoids radical intermediates [46]. The TvQR2 protein expressed in yeast is an NAD(P)H-dependent quinone oxidoreductase that reduces a variety quinones, including juglone (Figure 2ix) and DMBQ (Figure 2ii) (R Wrobel, JI Yoder, unpublished data). These substrates also regulate TvQR2 message levels so that the parasite evokes the same biochemical response to phytotoxic and xenognostic allelopathic quinones. The TvQR2 enzyme has similar substrate and kinetic properties to those of an intracellular quinone reductase characterized from the wood-rotting basidiomycete *Phanerochaete chrysosporium*, which serves to reduce small molecules generated during lignin degradation [47]. We propose that TvQR2-related enzymes function in many plants to detoxify small allelopathic xenobiotics, whereas TvQR1 fulfills functions that are specific to haustorium signaling in parasitic lineages (Figure 4). TvQR1 and TvQR2 enzymes therefore act towards opposing ends in parasitic plants, TvQR1 generates reactive semiquinones that are needed for haustorium initiation whereas TvQR2 eliminates them. Therefore, the relative levels of each protein are predicted to determine the cell's commitment to haustorium development.

Figure 4



Model of xenognostic quinone perception and processing. Host-plant roots (of maize, *Arabidopsis*, tobacco and so on) release xenognostic quinones (e.g. DMBQ) as a component of root exudates or as lignin degradation products. The quinones enter the parasite root (of *Triphysaria*, *Striga*, *Orobanche* and so on) where they are processed by two distinct cytoplasmic quinone reductases [42••]. TvQR1 catalyzes univalent reductions, resulting in radical-bearing semiquinones that are proposed to activate the haustorium-development signal transduction pathway [6••]. Semiquinones also donate electrons to molecular oxygen, resulting in the production of phytotoxic reactive oxygen intermediates (ROIs). A second quinone reductase, TvQR2, catalyzes two electron quinone reductions that detoxify xenognostic and other allelopathic quinones by avoiding the radical intermediates.

Both TvQR1 and TvQR2 transcripts are rapidly upregulated in *Triphysaria* roots that are exposed to DMBQ, even in the presence of cycloheximide [42••]. However, their regulation in parasitic and non-parasitic plants is distinct: TvQR2 homologs are upregulated in both parasitic and non-parasitic plants, whereas TvQR1 homologs are upregulated only in parasites. Recent work in my laboratory has shown that transcriptional regulation of TvQR1 is tightly

coupled to haustorium development in *Triphysaria* families that differ in their responsiveness to DMBQ [28•] (D Jamison, JI Yoder, unpublished data). The evolutionary history of zeta-crystallins in primates provides a provoking precedent for the differential regulation of this enzyme in different taxa. Zeta crystallins are highly abundant lens proteins in hystricomorph rodents and camelids but not other animals. One-electron reducing quinone reductase was independently recruited as a lens crystallin in these taxa as a result of the insertion of a lens-specific enhancer into the existing promoter [48]. It seems that both plants and animals have recruited this enzyme, which produces highly reactive molecules, to function in distinct taxon-specific developmental programs.

Conclusions

The transition from autotrophic to heterotrophic growth in parasitic plants is cued by host-plant recognition factors that are perceived via redox-associated mechanisms. Similar mechanisms probably function in other allelopathic interactions, though in many cases the phenotypes may not be as obvious as haustorium formation. Future experiments will exploit phylogenetic relationships to dissect parasite-specific events and to investigate the evolutionary origins of plant parasitism. One important outstanding question is the degree to which haustorium development in different parasitic families shares common genetic pathways. Work to identify host resistances against parasitic weeds will certainly continue, and advanced appreciations of developmental mechanisms may suggest novel approaches for engineering resistance [49]. Knowledge about parasitic-plant recognition of host plants may also facilitate the exploitation of allelopathic interactions to improve the subterranean performance of crop plants in the presence of competing weeds.

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