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Plant Genomics: Evolution and Development of a Major Crop Parasite

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https://doi.org/10.1016/j.cub.2019.07.067

Parasitic plants in the genus *Striga* bedevil crop production throughout Africa and Asia. A new genome assembly reveals how repurposing of developmental pathways, gene gains and losses, and horizonal gene transfer all contributed to the evolution of these destructive pathogens.

Plants are often admired for their energetic self-sufficiency or for fulfilling nutritional needs through mutually beneficial associations with microbes. However, a seedy underbelly of thievery in the plant world is less broadly appreciated. About 1% of flowering plant species parasitize other plants. Using specialized structures called haustoria, parasitic plants tap into the roots or shoots of hosts and siphon off water, nutrients, or photosynthate. Doing so allows parasites to boost their own reproduction at the host's expense. Some degree of parasitic habit has evolved at least 12 independent times across the plant kingdom [1], and since many crop species are susceptible to parasitic plants, understanding how these evolutionary transitions have occurred and how parasitic plants develop to invade hosts is of great agronomic importance. To this end, a new study by Yoshida and colleagues [2], published in this issue of *Current Biology*, reports the assembly and analysis of a genome from a species in the genus *Striga*, a burdensome group of crop parasites also known as witchweeds. The authors' findings develop a rich picture of how haustoria may have evolved and how parasites then became ever more reliant and specialized on the hosts they devastate.

Like their over 2000 relatives in the Orobanchaceae, witchweeds are persistent root pathogens. *Striga* infestations ravage over 100 million hectares of cereal and legume crops annually, causing severe losses throughout Africa and parts of Asia [3]. Eradicating *Striga* is very difficult because one individual can produce hundreds of thousands of dust-sized seeds. These seeds can stay dormant for decades, lying in wait until they sense molecules belonging to a group of structurally related chemicals known as strigolactones (SLs) that are exuded into the soil by potential hosts. Post-germination, host-released cues also induce development of the haustorium from cells in the embryonic root. The haustorium has hairs that cement it to the host and intrusive cells that invade host tissues to establish

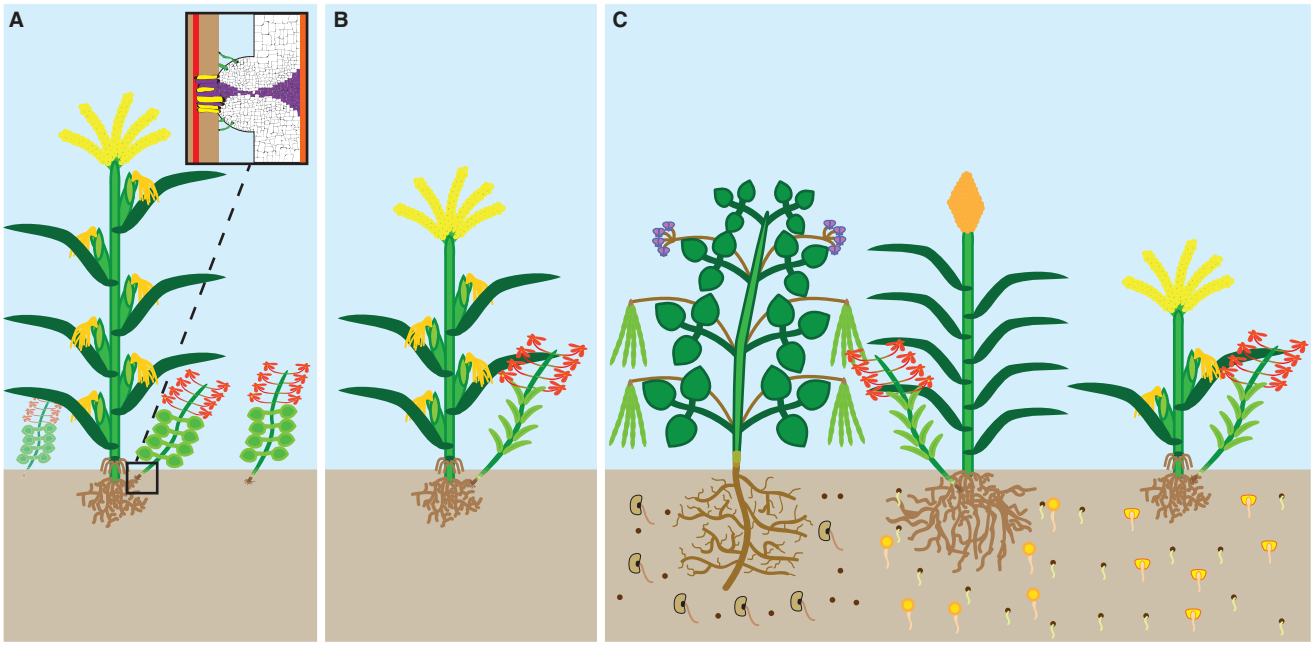

Figure 1. Three-phase model of parasitic plant evolution.
(A) Phase I: Facultative parasitism evolves through gain of haustorium development. Inset (adapted from [1]): Haustorial hairs (green) establish contact with host root. Intrusive cells (yellow) penetrate host tissue until they reach host vasculature (red), where some will differentiate into xylem cells (purple) and carry resources back to the parasitic vasculature (orange). (B) Phase II: Evolution toward greater, possibly obligate reliance on host plants through genetic degradation of mechanisms necessary for independent acquisition of resources (e.g., by reducing allocation to leaves). (C) Phase III: Evolution of greater host specificity, for instance through increased selectivity of host-induced germination responses.

vascular connections and divert resources (Figure 1A).

To uncover the evolutionary causes and consequences of these parasitic adaptations, Yoshida and colleagues assembled the genome of *Striga asiatica* and also sequenced transcriptomes from that species and *Striga hermonthica* sampled over a developmental time series of host infection [2]. The transcriptome data reveal striking gene expression dynamics through time, highlighting which batteries of genes contribute critical functions during specific phases of infection. For instance, enzymes that degrade cell wall carbohydrates are upregulated during host penetration, and transport-related genes are enriched among genes expressed as *Striga* begins looting nutrients.

The authors' gene expression time series also indicates that the essential first innovation in parasitic plant evolution, the origin of haustoria (Figure 1A), likely proceeded by evolutionary rewiring of existing genetic pathways. Haustorial gene expression patterns of parasitic Orobanchaceae share greatest similarity with root expression patterns in non-parasites [4], and haustoria must form new vascular elements after burrowing their way through overlying tissue layers [5], like the lateral roots that emerge from tissues beneath the root epidermis. Thus, it has been hypothesized that regulators of lateral root formation may have been evolutionarily repurposed, or co-opted, to regulate haustorium formation [1]. Consistent with this proposal, the authors found that many genes expressed during early haustorial development are homologous to a key signaling module regulated by the plant hormone auxin that directs the initiation and proliferation of lateral root founder cells [6]. These genes also promote auxin influx into overlying tissues to induce cell wall remodeling, reducing biophysical resistance to penetration by the developing lateral root [7]. Intriguingly, the authors observed expression of genes homologous to downstream targets of this module from the genome of the host plant soon after *Striga* infection, suggesting haustoria may also deploy auxin signaling to assert control over host tissues by reprogramming cellular identity and proliferation patterns.

As parasitic plants evolve the capacity to steal host resources, producing the machinery required to meet those same needs independent of a host becomes unnecessary and may come at the cost of extra seed production. Consequently, parasitic plants are expected to become more reliant on their hosts through the evolutionary loss of genes involved in resource acquisition (Figure 1B). Consistent with this prediction, many gene families — groups of related genes that have multiplied by duplication from a common ancestral sequence — have lost copies or been lost entirely since the divergence of *Striga* and *Mimulus guttatus* (yellow monkeyflower), the most closely-related, non-parasitic plant with a sequenced genome [2]. Moreover, as observed in the genome of the independently derived shoot parasite *Cuscuta australis* (Australian dodder) [8], these reductions preferentially affected genes involved in photosynthesis, leaf development, and hormonal responses to drought and other stresses. Thus, gene losses that strengthen dependence on host plants for carbon and water appear to be a

generalizable feature of parasitic plant evolution.

The authors also show that gene family expansion has likely facilitated the evolution of parasitism. Many *Striga* gene families have gained copies, and these gains appear to derive equally from recent duplications of individual genes as from a whole genome duplication event that occurred since the divergence of *Striga* and *Mimulus*. Because several of these new genes have haustorium-specific expression, they may have been important contributors to the origin of haustoria. However, other expansions may be more relevant to the final phase of parasitic plant evolution, when parasites further specialize to their hosts or expand their sphere of menace to infect yet additional victims (Figure 1C).

For instance, the number of SL receptors has expanded substantially within the Orobanchaceae. Initially identified as stimulants of *Striga* germination [9], SLs also play fundamental roles in stem and root development and in the recruitment of beneficial microbes [10,11]. Thus, *Striga* breaks seed dormancy only when its SL receptor arsenal binds these essential molecules that host roots cannot help but exude. Notably, the SL receptors that induce germination by *Striga* and its relatives gained this function following duplication from an ancestral receptor for karrikins, smoke-derived compounds that trigger germination in many non-parasitic plants [12]. Since that innovation, this receptor type has been amplified to a whopping 17 copies in *S. asiatica* [2]. Previous work has shown that sequence differences among SL receptors adjust their sensitivities for different SLs [13]. Thus, analogous to the large families of olfactory receptors found in animals, the expansion and specialization of SL receptors may enable *Striga* and its relatives to respond selectively to the diverse SL bouquets exuded by host and non-host species.

The *S. asiatica* genome assembly also reveals how over evolutionary time this parasite has stolen more than nutrients from its hosts. In 34 instances, *Striga* has acquired large DNA fragments, often tens of kilobases in length and containing multiple genes, from the very distantly related grasses it parasitizes [2]. Several transposable elements derived from grasses have also invaded the *S. asiatica* genome by this process known as horizontal gene transfer (HGT), including a recent case from sorghum. Although genes leaping between lineages that have been separated for tens of millions of years is well appreciated in microbes, the occurrence and extent of HGT of nuclear genes between multicellular eukaryotes is only now becoming apparent. Parasitic plants have been a central focus for investigating this surprising phenomenon, as the signal of HGT from hosts is readily discernible and vascular connections established by parasites have been viewed as potential conduits for DNA transfer. Other parasitic plants have similar numbers of HGT events as *Striga*, and the extent of HGT seems associated with how obligately dependent parasites are on their hosts [14–16]. However, work published this year documented comparable numbers of HGT events into the genomes of several non-parasitic grasses [17], raising the possibility that the focus on parasitic plants may be a case of 'looking under the lamppost' and HGT is a more general phenomenon. Going forward, the *S. asiatica* genome will aid investigations into whether the transition to parasitism leads to higher HGT and what mechanistic connections may be shared with non-parasitic plants that also acquire genes by HGT [18].

Yoshida and colleagues [2] have established a valuable genomic foundation for future research into parasitic plant evolution and agronomic control strategies. The preliminary connection between a well-studied auxin regulatory pathway and haustorial development presents a particularly promising new entry point for learning how novel structures evolve by co-opting existing regulatory mechanisms. Investigations of auxin signaling dynamics in *Striga* and its hosts are sure to reveal important similarities and differences between the cellular progression of haustorial and lateral root development [19]. Comparative studies across diverse root and stem parasites can also assess whether the lateral root pathway has been repeatedly co-opted or whether alternative mechanisms have produced convergent outcomes. Finally, the genome will help advance the identification of targets for engineering *Striga* resistance, and knowledge of the many *Striga* SL receptor sequences may now potentiate development of crops that grow and interact with symbionts normally using SLs that avoid triggering parasite germination [20].

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Neocortical Cell Classes: Essential Contributions from Electrophysiology

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Neocortical cells form classes that likely take on unique functional roles in the cortical microcircuit. A new study finds that, with sufficient sampling, the electrophysiological signature of cells distinguishes four cell classes across primate frontal and parietal cortex.

Understanding how cortical circuits operate requires understanding how individual cells in that circuit contribute to the transformation of inputs into outputs. These contributions fall into generic classes, such as feedforward inhibition for normalizing input to the circuit, fast feedback inhibition for gain modulating the output from a circuit, or the slow rhythmic pulsing of activity for sustaining activity levels even in the absence of synaptic inputs [1–3]. One assumption from studies tagging cells with molecular markers is that these generic circuit functions are realized by different groups, or classes, of cells that share the same molecular make-up [3,4]. For example, cells expressing selectively the protein parvalbumin (PV+ cells) have been associated on average with fast feedback inhibition in cortical circuits [4].

However, such a *molecular based* strategy is limited. For one thing, cells expressing the same molecular marker are not a homogenous group with respect to other criteria, such as their morphology, electrophysiological response profiles, or location in the circuit [5,6]. PV+ cells, for example, consist of disparate forms of basket cells and chandelier cells with varying laminar locations, connectivity and physiological responses to electrical stimulation [7]. Moreover, observing cells in a circuit that are tagged for specific proteins imposes a positive bias to assigning functions to the observable, tagged cells without revealing how the same function may map onto subpopulations of unidentified, non-tagged cells. These limitations of a molecular based approach call for complementary strategies to obtain a more complete picture for understanding cell class-specific circuit functions [2,5].

A new study by Trainito *et al.* [8] reported in this issue of *Current Biology* pinpoints the versatility of such a strategy by emphasizing the cell's physiological signature. Their *electrophysiological based* approach used the cells' action potential waveform to robustly distinguish four classes of cells in extracellular recordings from the primate prefrontal and parietal cortex that were made while rhesus monkeys prepared for goal-directed choices. The action potential of a cell reflects a cascade of ion channel openings and closings that occur with dynamics determined by the cell's particular state of differentiation as defined by the genes that it expresses [9]. The authors used the trough-to-peak duration and repolarization speed of the cell's average action potential for data-driven two-dimensional clustering. This clustering revealed one large class with 69.1% of all cells having broad and slow action potentials, while three classes had narrower action potentials. The discovery