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Review Article

A Review on the Biology and Resource Acquisition of Striga, and the Defense Responses of Host Plants

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Abstract

Striga is an herbaceous hemiparasitic angiosperm that belongs to the Orobanchaceae family of root parasites. It is an annual and very rarely perennial parasite. It is one of the most destructive crop pests in Africa, India, and Southeast Asia. Particularly in the Sahel region, it is the most significant invasive species of sorghum, maize, pearl millet, upland rice, and sugarcane. In sub-Saharan Africa, more than 50 million hectares of arable land covered with cereals and legumes are infested with one or more of these species. Consequently, annual crop losses are estimated at $75 million, $87 million, and $1.2 billion, in Ethiopia, Mali and Nigeria, respectively. In general, it causes over US $10 billion annual yield losses across the world.

From the total 42 Striga species, 11 of them are identified to infest crops. Except Striga gesnerioides that can infect some dicotyledonous crops all the others are parasites of monocotyledons. Among the parasitic members, *Striga hermonthica* and *Striga asiatica* are the most economically important species. Soon after the germination, Striga uses chemical exudates from the host's root to develop a special organ called the haustorium. The haustorium dissolves and invades the host plant's roots in a matter of hours, forming vascular connections that allow it to successfully siphon all the resources required for growth. Following the parasite's attachment, the host plant recognizes it as an alien and tries to defend itself by deploying a variety of defensive mechanisms against the parasite's attacks at various locations along the root.

Striga's life cycle is extremely complex. Through a variety of mechanisms, it can easily hijack the defensive responses of a compatible host and could result in yield losses ranging from a few percent to total crop failure. Because of this and other reasons, this study concluded that integrated management approaches have a higher potential of reducing *Striga* infestation than do single control measures. Therefore, care should be taken to test and identify effective control methods by integrating cultivars resistant to *Striga*.

INTRODUCTION

Background of the Study

Parasitic plants are plants that are partially or completely reliant on their hosts. They obtain their resources partially or completely from the other plants [1]. Globally there are about 4500 species of parasite plants belonging to 275 genera and 28 dicotyledonous families [2,3]. With the exception of the aquatic environment, they are widely distributed throughout the world along with angiosperms [4].

Based on the presence or absence of chlorophyll, parasite plants are broadly classified into holoparasites and hemiparasites [2]. Holoparasites are achlorophyllous parasites that are entirely dependent on their host for the supply of assimilates whereas hemiparasites do have some chlorophyll and can perform photosynthesis to some extent but not necessarily self-sufficient with carbon [2]. Based on the site of attachment to the host plant, they are also broadly classified as root parasites, which attach to the root of the plants, and stem or aerial parasites, which attach to the shoot system of the host plant [4]. According to Heide-Jorgensen [4], about 90% and 60% of the parasites belong to the hemiparasite and the root parasite groups, respectively. Different sources [1,2,5] revealed that members of Orobanchaceae, particularly *Striga* species (witch weeds) are among the most agriculturally destructive parasite plants in the world.

The genus Striga is one of the most important crop pests in the Africa, India and Southeast Asia Ejeta [6] and Scholes and Press [7] particularly in the Sahel region, it is the main invasive pathogen of cereal crops [8,9]. According to Rodenburg, et al [10]. it has been observed in at least 44 countries in Sub-Saharan Africa. It is one of Africa's most destructive pathogens, affecting economically important crops [11]. Based on the findings of Gressel, et al [12]. About 64% of cultivated land in West Africa and 23% of it in East and Central Africa are infested with it. According to Ejeta [6] and Scholes and Press [7] reports, over

50 million hectares of the arable farmlands covered with cereals and legumes in sub-Saharan Africa are infested with one or more of its species. This is the cause of over US $10 billion annual losses of yield in the world. According to African Agricultural Technology Foundation [13], annual losses for Ethiopia, Mali and Nigeria are estimated at US $75 million, US $87million, and US $1.2 billion, respectively. In particular, *Striga hermonthica* is a serious pathogen of crops grown by subsistence farmers in the Sahel region [14]. It accounts for about 25% of the annual sorghum losses in Ethiopia [13] and it also affects about 20 to 30% of the country's maize cultivated areas [15].

So far there are about 42 Striga species, of which at least 11 are known to infest crops [16]. Except Striga gesnerioides that can infect some dicotyledonous crops all the others are parasites of monocotyledons [17,18]. From the total species of it, Striga gesnerioides, Striga asiatica, and Striga hermonthica have severe devastating impacts in agriculture almost in the worldwide range [19]. According to Runo and Kuria [9], Striga gesnerioides attacks dicotyledonous species like cowpea (*Vigna unguiculata* (L) *Walp*), tobacco (*Nicotiana tabacum* L), and sweet potato (*Ipomoea batatas* L) while Striga hermonthica and Striga asiatica infest staple cereal crops such as sorghum (*Sorghum bicolor* (L) *Moench*), maize (*Zea mays* L), pearl millet (*Pennisetum glaucum* (L) R Br), upland rice (*Oryza sativa* L), and sugarcane (*Saccharum officinarum* L).

Although *Striga* species are diverse worldwide, *Striga hermonthica* and *Striga asiatica* are the most economically important species [20,21]. Of these, *Striga hermonthica* is probably the most dangerous parasitic weed in the world [22]. *Striga hermonthica* is believed to have originated in the Nuba Mountains of Sudan and Ethiopia [23]. It is presently widespread throughout northern tropical Africa and reaches as far west as West Africa from Ethiopia and Sudan. Moreover, it extends southward into Namibia and Angola from the western Arabian Peninsula. *Striga asiatica* is relatively widespread and is found in semiarid regions of tropical and subtropical Africa, Asia, and Australia. All of this ensured that it is the most prevalent parasite in soils that are barren or where plants are cultivated under moisture stress. Under such conditions, it causes devastating agricultural losses [24].

Striga species are able to develop green leaves and possess intact chloroplast genomes [11,25], but they only have poor rates of photosynthesis [26], so they must rob the majority of organic matter from their hosts [11,27]. Moreover, they have high transpiration rates which allow them to absorb a lot of water and dissolved minerals from their hosts [14]. All of these facts assured that they can lead to yield losses ranging from a few percent to complete crop failure (Figure 1) depending on crop species, crop variety, and intensity of the infestation [11,22].

In Africa, almost two-thirds of the 70 million hectares used for cereal production are infested by Striga. It is therefore thought by many experts to be the biggest challenge to food production in Africa, especially in the Sahel region, as it adversely affects the lives of about 300 million people [6,31-33]. Plants infested by it continue to be stunted, wilt, and turn yellowish; if they are severely parasitized, they will eventually die [11] and (Figure 2). Many of their haustoria that are attached to the infected root and feed on it are present in considerable numbers (Figure 2). As a result, many of them may therefore be grown close to the infected plants (Figure 3).

In general, the primary goals of this review were to explain the *Striga's* reproductive, seed dispersal and germination mechanisms, to describe the ways of haustorium formation and their nutrient acquisition, and finally, to demonstrate host plant defenses against it.

Description of the Genus Striga

Striga plants are annual, rarely perennial herbaceous parasites. They are small pretty plants with a bright green,

Figure 1 Complete and partial impacts of Striga on crops [28-30], respective.

Figure 2 The stunting effect of Striga on maize [34].

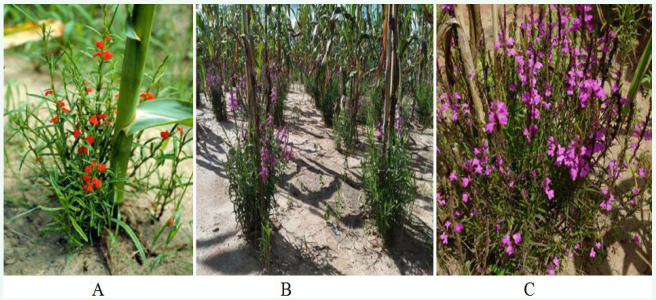

Figure 3 The concentrated growth of Striga around the host plant [35,28,36], respectively.

slightly hairy stems and leaves [11]. They lack roots and are instead attached to the hosts by haustoria. The haustoria are white and rounded in cross section. They have no root hairs [11]. Stems are erect, usually quadrangular and often ridged. Leaves are opposite or nearly so, sessile or subsessile; in most species reduced to small linear lanceolate scales, with entire margin [18,22,37-40].

Inflorescences are terminal, racemose, congested or lax, occasionally capitate, frondobracteate or frondose, and bracteoles present. Flowers are bright, irregular, sessile or subsessile. Calyx is tubular, five lobed or five (rarely four) toothed. Corolla is white, rose, lilac (pink), red or cream to orange (yellow), bilabiate, strongly 2-lipped, upper lip bifid or ernarginate, erect, lower lip tripartite, spreading, tube narrow, curved in right angle, orifice of tube small, less than 1 mm in diameter, usually densely pubescent. Stamens are 4, didynamous, included, inserted in tube below orifice, anthers monothecous, basifixed on short filaments. Ovary is tubular, with basal disc, with terete, elongate style. Stigma is clavate [18,22,37-40]. The spike has occasionally more than 10 open flowers and the corolla normally drops a few days after fertilization. The number of capsules per plant may be on the average 42 to 110. Fruit a capsule with loculicidal dehiscence. Seeds are numerous, minute (dust seeds), and testa with prominent encircling ridges (Figure 4). For instance, in *Striga hermonthica* seeds are extremely small, about 0.15 mm wide, 0.3 mm long, and weighing about 0.7 µg [40]. The number of seeds per capsule varies from about 700 in *Striga hermonthica* to 800 in *Striga asiatica* and 1800 in *Striga* gesnerioides. The embryo is highly reduced, lacking cotyledons and root cap. It is enclosed in an endosperm, which function as storage tissue [18,22,37-40]. Seeds can germinate in a relatively short period of time.

Reproduction, Seed Dispersal and Germination Mechanisms

Striga plants are small pretty plants with a bright green, slightly hairy stems and leaves [11]. They have attractive pink, white, red, purple, or yellowish flowers in their flourishing periods [14,40]. Their flowers are mostly pollinated by insects [4]. A single *Striga* plant can produce between 50,000 and 500,000 black, tiny, and fertile seeds following successful pollination [11,19]. Such high fertility increases the epidemiological potential of the pathogens. To increase their likelihood of finding the host plant, mature seeds are dispersed by a variety of means, including wind, water, insects, livestock, people, contaminated tools and equipment, or contaminated soil carried on farm machinery [11,40]. Although some of their seeds can germinate immediately, the majority need a dormancy period of 15 to 18 months before germination [11].

According to Musselman [14] and Agrios [11], *Striga* seeds germinate only in response to the chemical signals released by the host plant. These life cycle-initiating chemical stimuli are called germination stimulants. They are secreted by host roots and promote the germination of the parasite's seeds [27,42]. However, the parasite seeds need a pre-treatment known as preconditioning [43] or warm stratification before they can respond to these stimuli and germinate [44]. According to Joel, et al [43] and Ejeta [45], preconditioning activates the seed's metabolic pathways, including respiration and the synthesis of DNA, proteins, and hormones. After the conditioning phase, the parasite seeds will only begin to germinate if they are exposed to sufficient concentrations of germination stimulants, ensuring that germination only takes place near the host roots (Figure 3). However, if the preconditioning is not favorable for germination, seeds remain dormant for several months [46]. Their seeds remain viable in the soil from 15 to 20 years [47]. However, Scholes and Press [7] and Berner, et al [48] assured that they can remain viable for more than 20 years.

Zhongkui [1] claims that a variety of secondary metabolite classes have been identified as germinating stimulants. For instance dihydrosorgoleone, the strigolactones and the sesquiterpene lactones are the main classes of germination stimulants. Among the various types, strigolactones are the best explored so far [49]. Seven natural and one synthetic strigolactone germination stimulants have so far been identified and studied in plants (Figure 5). From these, Synthetic germination stimulant (GR24) is widely used in parasitic weed research to stimulate parasitic weed seed germination at the global level [50].

Figure 4 Mature *Striga* capsules shedding seed [41].

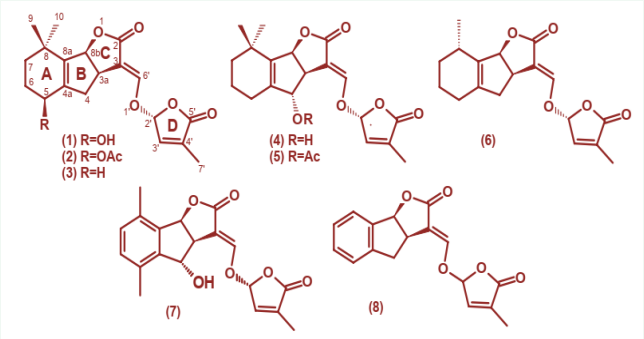

Figure 5 Structures of strigolactones [1].

Since the parasites' tiny seeds have limited reserves and the seedlings will die after 3 to 7 days of germination unless a host root is invaded, the parasites' evolutionary adaption to respond to the germination stimulants is significant [19,40,51]. Because of this, their seeds are only briefly responsive to germination stimulants before going into secondary dormancy, where they can be hidden until the occurrence of suitable conditions. From the moment a seed germinates until the growing plant releases its first seeds, it takes 90 to 120 days [11].

According to Joel [52], seedling growth occurs in stages that are sequential, starting with the seedling developing alone for a brief time before attaching to a host. This is a short independent stage of the parasite development. The next stage of development is called intrusive development, during which a terminal haustorium forms at the tip of the radicle, the haustorium invades the host tissues, and the principal conductive connections with the host are formed. Finally, during the compatible phase, the host and parasite development are synchronized (Figure 6).

Striga Haustorium Formation and Its Way of Resource Acquisition

Striga species produce a large number of seeds to increase their chances of finding a host, but this comes at the expense of seed size. The number of seeds per capsule varies depending on the species from 700 to 1800 [40]. Their tiny seeds contain only small amounts of nutrients and cannot continue their life cycle without directly connecting to a compatible host for nutrient supply. This is accomplished via the growth of the haustorium, which ensures the availability of nutrients following germination. According to Joel [52] definition, haustorium is a specialized organ of parasitic plant that invades host tissues and serves as the structural and physiological bridge that enables the parasite to extract water and nutrients from the conductive systems of the host plant.

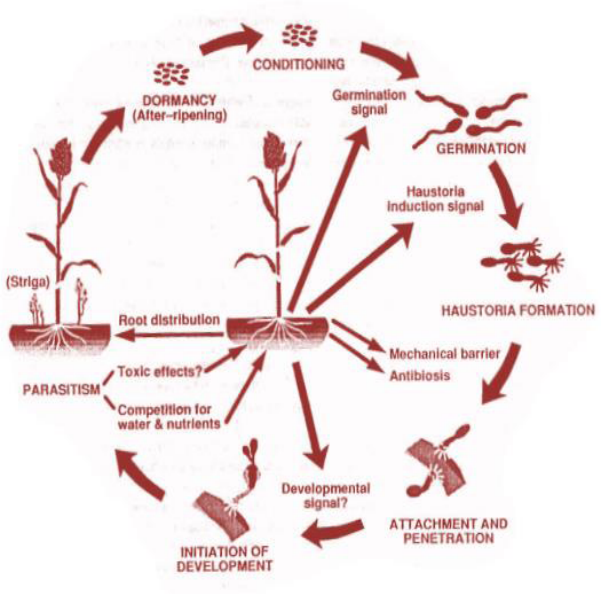

Figure 6 General life cycle of Striga species and potential mechanisms for host-plant resistance [56].

During the germination process of parasite seeds, the radical must grow towards the host root possibly directed by the concentration gradients of germination stimulants [17]. The development of haustorium initiates upon the parasite's recognition of a nearby host triggered by host root exudates such as strigolactones, flavonoids, quinones and cytokinins [52,53]. These haustorial-inducing factors (HIFs) initiate signaling cascades that lead to the accumulation of reactive oxygen species (ROS) in parasitic roots and the formation of haustoria [54].

The attachment and penetration of the host root are accomplished by the haustorium once the radicle has reached the host root. Within 8 to 24 hours, the haustorium dissolves and penetrates the host root [11]. The haustorium enters the host root and connects the host's vascular system to that of the parasite. Another host-derived chemical signal, 2, 6-dimethoxy-p-benzoquinone (DMBQ), is needed to initiate and guide this developmental transition during the formation of this organ [55]. After vascular connection, the parasite will develop a so-called tubercle that helps to accumulate nutrients. The lifecycle can restart once it develops a shoot, emerges above ground, blooms, and produces seeds (Figure 6). The development of the haustorium depends on its ability to overcome host resistance mechanisms and to compete with host organs on available host resources. In general, the parasite's success ultimately depends on its capacity to establish and maintain a union with the host [5].

Obligate hemiparasaites, such as *Striga species*, initially develop the terminal haustorium and then produce lateral haustoria after the development of their shoots [57]. The terminal haustorium develops directly from the radicle's apex, while the lateral haustoria emerge laterally on young lateral or adventitious roots.

The cell wall-degrading enzymes that loosen host cells and allow the penetration peg to move between cells may be the mechanism by which the intrusive cells of most Orobanchaceae, including *Striga*, invade the host cortical parenchyma without apparent damage to the host's cells [58,59]. In *Striga hermonthica*, the haustorium penetrates the host's endodermis by dissolving the casparian strip and advancing between the endodermal cells without harming them [59,60]. After cell wall degradation, it is thought that the intrusive cells enter the host cells using the mechanical force exerted by cell enlargement and cell division of the haustorial inner cortex and pericycle [4,61]. Hence, it appears that parasitic plants use enzymes to weaken the cell walls and the pectin layers connecting adjacent cells, and this loosening is crucial for haustorium penetration. Once the haustorium passes through the endodermal layer, the parasite starts the process of establishing vascular connections with the host. The host xylem vessel is penetrated by some of the invading cells. For instance, in *Striga hermonthica* some of the intrusive cells invade host xylem vessel elements and develop into ocular, which are tube-like vessel elements [62]. After that, cells in the middle

of the haustorium differentiate into vessel elements to form a continuous file of xylem cells between the parasite and its host, the xylem bridge, which enables the parasite to absorb water and nutrients [63]. However, it is not yet clearly understood how *Striga* shares the C-assimilates from the host plant.

The nature of hustarium is very complex. Initially, it acts as an attachment organ, but later it transforms into invasive structure that enters the host tissue. Eventually, by forming vascular connections it becomes an organ that can absorb all nutrients and water from the host [11]. This connection, commonly called the xylem bridge, connects the parasite xylem to the host xylem for successful invasion [2]. Severe *Striga* infection suggests that there may be a phloem connection to siphon out photosynthate, though this is not yet obvious. Anyhow, after successfully forming a vascular connection, the parasite readily absorbs all the necessary materials from its host. However, research suggests that transport between the host and the parasite is bidirectional, carrying substances like RNA and plant hormones between the parasite and the host [64].

In general, the *Striga* life cycle is highly synchronized with its host and includes the following three general phases: germination, haustorium formation, and penetration [9]. Eventually, the haustorium penetrates the host until it reaches the endodermis, where it encounters haustorial cells that elongate and divide to form vascular connections with the host. Once the vascular systems of the host and parasite are connected, it uses this connection to effectively siphon out all the necessary nutrients.

Host Defense against Striga Species

Striga is commonly called Witchweed. This is a logical name because the parasite "bewitches" its host before it emerges and is visible above ground. This indicates that it causes stunted growth and early discoloration of crop leaves before it emerges [65]. It must first attach itself to the host's root in order to penetrate and share all the nutrients of the host plant. The parasite plant uses its haustorium to attach and penetrate host tissue, form vascular connection, and obtain resources from the host [55]. Infection requires a precise and compatible interaction with the host plant, including signaling and the regulation of host defenses.

According to Joel and Portnoy [66], a susceptible host can recognize the parasite as an alien and attempts to defend itself against the parasite attacks using various types of defensive responses. However, the parasite hampers a compatible host's defense responses through a variety of mechanisms. Mayer [67] suggested two possibilities, the first possibility is related to camouflage because the parasite and the host are both higher plants and share many biochemical and physiological characters, and the second possibility is that the parasite plant actively suppresses the host's defense responses. The logical response might be that there are active parasite mechanisms that impede host responses. He pointed out that phenolic compounds might serve as deterrents of host defense responses. Lack of intracellular reactive oxygen species (ROS), either from the host or the parasite, may indicate that the compatible host is unable to defend itself from parasitic attack [68].

However, the host plants attempt to defend themselves against the devastating *Striga* species by utilizing a variety of physical and biochemical weapons. Host resistance is multidimensional, involving both general and specific defense mechanisms that are constitutively deployed or activated to specifically interfere with critical steps throughout the parasite life cycle. Observed resistance mechanisms to parasitic weeds can be broadly classified as pre- or post-attachment resistance [7,8].

Pre-Attachment Host Defense

The timing of germination near the host root is critical for *Striga* because its small seeds cannot support a long growing season without attachment and utilization [46]. They recognize potential hosts through the sufficient secretion of Stregtolactins from the host roots [1,42,49]. This promotes ethylene production in the seed, which in turn stimulates parasite seed germination [69].

Germination is an early stage of the parasite lifecycle where host resistance can occur. Potential hosts can alter parasite success prior to attachment via allelochemicals [70]. Plants used allelochemicals to inhibit the germination and growth of neighboring competitors [71], as well as the growth of parasite plants [70]. For instance, the flavonoid isoschaftoside isolated from the root exudates of Desmodium uncinatum acts as an allelochemical to prevent radical development in *Striga hermonthica* [72]. Based on Kim, et al. [73] report, host plant catalase enzymatic activity can also inhibit its haustorium induction by scavenging H_2O_2, thereby preventing the oxidation of syringic acid to 2, 6-Dimethoxy-P-Benzoquinone (DMBQ).

Pre-attachment resistance, in its broadest sense, refers to all mechanisms that enable a potential host to avoid or prevent parasite attachment. These mechanisms include the absence or minimal production of germination stimulant, germination inhibition, inhibition or reduction of haustorium formation, partial inhibition of haustorium development and formation of mechanical barriers to infection like thickened host root cell walls [2].

However, the pre-attachment processes of *Striga* infection leading up to root attachment can happen in a matter of hours in the absence or presence of weak host resistance [7]. The host plant attempted to implement a different defense mechanism, post-attachment defense, when *Striga* species resisted the host's pre-attachment defense response and proceed to grow.

Post-Attachment Defense

Several host resistance reactions can occur during the attachment and penetration stage of the *Striga* lifecycle [74]. One sign of active identification and the onset of a hypersensitive response (HR), for instance, is the occurrence of necrosis around the attachment site in more resistant hosts (HR). For example,

Striga gesnerioides infection of the resistant cowpea cultivar B301 leads to HR, which is mediated by a defense gene that encodes a type of resistance (R) sensor protein (coiled-coil nuclear-binding site leucine-rich repeat (CC-NBS-LRR)) known to induce resistance against many plant pathogens in other plant species [75]. The presence of host R sensor proteins also suggests the presence of parasite effectors.

As the penetration peg gets closer to the host vascular tissues, parasite development can be prevented in several regions of the root. In the cortex, prevention of parasite development is associated with the deposition of physical cell wall barriers such as lignin, and poisonous phenolic compounds resulting from phenylpropanoid metabolism [76]. It has also been noted that the endodermis is protected against penetration. For instance, *Striga hermonthica* can invade the cortex of the resistant rice cultivar Nipponbare but is unable to pass through the endodermis. Instead, it develops around the vascular cylinder [77]. Based on their findings, lignin deposition was not observed so it is thought that this response involves a lesion in the signaling pathways that penetrate the endodermis. The endodermis may provide a significant physical barrier to parasite progression.

Due to the importance of physical barriers like lignin and the related accumulation of phytoalexins during resistant interactions with parasite plants, the biosynthetic pathways that lead to their production are a significant component of a defense response. The production of phytoalexins, as well as lignin, suberin, and callose, which are employed for physical defense against penetration, is dependent on phenylpropanoid metabolism in plants [78].

In general, post-attachment resistance occurs once the haustorium has formed and the parasite attempts to penetrate the host root tissues and connect to the vascular system. Several constitutive or induced incompatibility or host resistance mechanisms may be triggered during these developmental phases. These include the synthesis and release of cytotoxic substances (phenolic acids, phytoalexins, etc) by the attacked host root cells, a process called abiosis, the development of physical barriers to stop potential parasite or pathogen ingress and growth (such as the lignification and suberization of cell walls) and programmed cell death (PCD) in the form of a hypersensitive response (HR) at the point of parasite attachment to restrict parasite development and retard its penetration and prevention of the parasite establishing the essential functional vascular continuity (that is, xylem-to-xylem and/or phloem-to-phloem connections) with the host [2].

General Mechanisms of Host Resistance

Jones and Dangl [79] claim that plants use a two-level innate immune response mechanism (the 'zigzag' model) to react to pathogen challenges. Although this is derived from the interactions between pathogenic microbes and plants, it can be easily applied to the discussion of resistance to root parasitic angiosperms [2].

The first level responds to molecules that are slowly evolving and are referred to as pathogen-associated molecular patterns (PAMPs) [80]. Pattern recognition receptors (PRRs), a group of receptor-like kinases, serves as host sensors that detect PAMPs and trigger host defensive mechanisms known as pathogen-triggered immunity (PTI) [81,82]. The parasites or phytopathogens have evolved specific effectors and virulence factors that are capable of entering the host cell and inhibiting PTI to circumvent this host monitoring mechanism [2]. According to Go¨hre and Robatzek [83] and Tyler [84], the effectors of the parasite or phytopathogen mostly penetrate plant cells by specific secretion machinery.

The host plant develops a second line of defense termed effector-triggered immunity (ETI) to detect and attempt to disable the parasite effectors for further restricting the parasite growth. ETI involves a second class of receptor proteins that often comprise a nucleotide binding site (NBS) and a leucine-rich repeat (LRR) domain [85,86].

Resistance (R) proteins, also known as NBS-LRR proteins, are often expressed by genes that plant breeders have identified as the main R genes that defend against particular strains (or races) of a pathogen or parasite [87,88]. Historically, pathogen or parasite genes encoding effectors detected by plant R genes have been called avirulence genes (Avr) because they prevent infection of host plants carrying these R genes [2]. Once Avr proteins or effectors enter host plant cells, they are either directly or indirectly detected by R proteins [88-91]. When recognition takes place, a complicated cascade of signaling processes is activated, triggering defense reactions that restrict the growth of pathogens or parasites.

In general, the parasite challenge is often mitigated by a variety of mechanisms in both host and non-host plants. The rate of parasite establishment, tubercle development, and the total number of emerging shoots, however, more accurately represent the parasite's capacity to circumvent the various resistance mechanisms engaged to prevent parasitism.

CONCLUSIONS AND IMPLICATIONS FOR PRACTICE AND RESEARCH

Striga is an annual and rarely perennial herbaceous root parasite belonging to the family Orobanchaceae. From the 42 identified species, 11 of them have been identified as crop pests. Among the pests, *striga gesnerioides*, *striga asiatica*, and *striga hermonthica* have devastating impacts on agriculture across the world. A number of studies have been conducted in this area across the world to reduce or halt their economic impact. Despite extensive research, effective methods of managing parasitic plants are still elusive, posing a threat to agricultural crops across the globe.

Although the life cycle of *Striga* is highly complicated, researchers have made an effort to comprehend the biology and physiology of the parasite, including the characteristics of its seeds, dispersal and germination mechanisms, haustorial

initiation and development, attachment, penetration, and establishment. To a certain extent, they also attempted to determine the host plant reactions. However, the mechanisms through which they recognize a host, initiate haustoria formation, penetrate the host and form vascular connections especially phloem connection remain poorly understood and require more research. Moreover, resistance and tolerance are two highly complementary defense strategies against the parasite; therefore future studies should focus on understanding the host defensive mechanism and knowing how the parasite hijacks host developmental programs to successfully infect. Changing these host development initiatives could provide a novel approach for enhancing resistance.

When paired with fertilizers, myco-herbicides, crop rotation, intercropping with nonhost plants that induce suicidal germination and/or are allelopathic to root parasites, herbicide-based seed coating, or synthetic germination stimulants, *Striga*-resistant cultivars can be incorporated into integrated management approaches, which have a greater potential to reduce infestation than single control methods until effective remedies are obtained. In particular, the production of *Striga*-resistant maize genotypes may greatly benefit from the application of biotechnological technologies such as marker assisted breeding, targeted gene editing or mutation breeding, and RNA interference (RNAi). For farmers with limited resources, all of this contributes to securing food supplies, income, and yield levels.

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