PLANT SIGNALING & BEHAVIOR
2017, VOL. 12, NO. 2, e1274481 (19 pages)
http://dx.doi.org/10.1080/15592324.2016.1274481

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REVIEW

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Components of the SNARE-containing regulon are co-regulated in root cells undergoing defense

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ABSTRACT

The term regulon has been coined in the genetic model plant *Arabidopsis thaliana*, denoting a structural and physiological defense apparatus defined genetically through the identification of the *penetration* (*pen*) mutants. The regulon is composed partially by the soluble N-ethylmaleimide-sensitive fusion protein attachment protein receptor (SNARE) syntaxin PEN1. PEN1 has homology to a *Saccharomyces cerevisae* gene that regulates a Secretion (Sec) protein, Suppressor of Sec 1 (Sso1p). The regulon is also composed of the β-glucosidase (PEN2) and an ATP binding cassette (ABC) transporter (PEN3). While important in inhibiting pathogen infection, limited observations have been made regarding the transcriptional regulation of regulon genes until now. Experiments made using the model agricultural *Glycine max* (soybean) have identified co-regulated gene expression of regulon components. The results explain the observation of hundreds of genes expressed specifically in the root cells undergoing the natural process of defense. Data regarding additional *G. max* genes functioning within the context of the regulon are presented here, including Sec 14, Sec 4 and Sec 23. Other examined *G. max* homologs of membrane fusion genes include an endosomal bromo domain-containing protein1 (Bro1), syntaxin6 (SYP6), SYP131, SYP71, SYP8, Bet1, coatomer epsilon (ε-COP), a coatomer zeta (ζ-COP) paralog and an ER to Golgi component (ERGIC) protein. Furthermore, the effectiveness of biochemical pathways that would function within the context of the regulon ave been examined, including xyloglucan xylosyltransferase (XXT), reticuline oxidase (RO) and galactinol synthase (GS). The experiments have unveiled the importance of the regulon during defense in the root and show how the deposition of callose relates to the process.

Abbreviations: SNARE, soluble N-ethylmaleimide-sensitive fusion protein attachment protein receptor; ERGIC, ER to Golgi component

ARTICLE HISTORY
Received 27 October 2016
Revised 14 December 2016
Accepted 15 December 2016

KEYWORDS
α-SNAP; β-glucosidase; ABC transporter; callose; pathogen; regulon; SNARE

Introduction

The ability of eukaryotic membranes to fuse is important to many biological processes, including secretion.[1] Owing to its importance, the genetically regulated process is ancient with gene homologs found in all eukaryotes.[2] Subsequent biochemical experiments have determined the affinity that these proteins have for each other under various conditions.[3,4] These experiments have provided foundational knowledge while also revealing new roles for some of the proteins. In *A. thaliana*, the secretory apparatus functions in defense responses.[5] Furthermore, this secretion system has been expanded to include structural, biochemical and physiological components and is referred to as a regulon, a binary system composed of two parallel pathways converging on defense.[6,7,8] Subsequent experiments have shown that the regulon components have the ability to coordinately regulate their expression (co-regulate) other regulon genes.[7,9] In the review presented here, details are provided that describe the components of the regulon. The review examines how observations first made over a century ago regarding ecological plant variants capable of warding off pathogens have provided clues as to how the plant defense process functions.[10] The review subsequently intercalates more recent genetic evidence that puts those original ecological observations into the context of the regulon.[5,6,8-12] The review describes co-regulation of the regulon genes. The review then describes the identification of other genes expressed in the cells undergoing the process of defense that would be expected to function within the regulon platform.

Early observations of a genetic basis for the regulon

Observations made over a century ago identified the capability of certain ecological variants of the legume *Trifolium repens* to engage a successful defense response in their shoot that acts against various herbivores.[10] This genetic system is defined by two loci.[10-12] One diploid parent provides the dominant allele (*Ac*) driving α-hydroxynitrile glycoside production and one parent provides a dominant allele (*Li*) encoding for its hydrolyzing α-hydroxynitrile glycosidase.[10-12] These observations implicate two different modes of secretion would converge during the process of defense because

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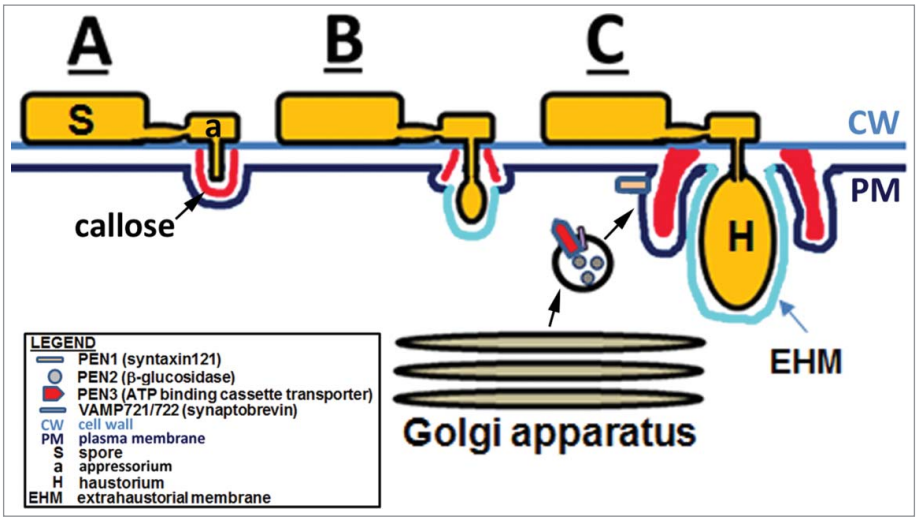

Figure 1. The regulon. In experiments involving *Blumeria graminis* f. sp. *hordei, Erysiphe cichoracearum, Golovinomyces orontii*, a structural, biochemical and physiological apparatus referred to as the regulon has been designated to refer to a defense apparatus involving the PENETRATION1-3 proteins. (A) The spore has germinated producing a neck that is attached to the appressorium which begins to push through the cell wall resulting in a plant cell response that leads to callose deposition. (B) The haustorium develops and pushes through the callose during a susceptible response. (C) Golgi-drived vesicles deliver the PEN1-3 proteins to the infection site, leading to a successful defense response that includes increased deposition of callose.[5,6,8,15,16,254-266]

α-hydroxynitrile glycosidases have a signal peptide allowing it to enter the secretion system while glycosides can be mobilized by ABC transporters.[13,14] Subsequent experiments performed in the plant genetic model *A. thaliana* have led to the identification of this genetic framework, referred to as a regulon.[6] The framework of the regulon is defined by three genes including the syntaxin *PENETRATION1* (*PEN1*), β-glucosidase (*PEN2*) and an ATP binding cassette (ABC) transporter (*PEN3*) (Fig. 1).[5,6,15,16]

Knowledge of how regulon components function clearly implicated other genes would have roles within the context of the regulon. Using the *Glycine max-Heterodera glycines* pathosystem as an experimental model, Klink et al.[17] performed gene expression studies examining RNA isolated from *G. max* root cells parasitized by *H. glycines*. The experiments led to the determination of gene expression patterns in cells undergoing the process of defense. In those and related studies, Klink et al.[17-21] identified that the defense apparatus may be quite extensive, possibly composed of hundreds to thousands of genes. Experiments then examined gene expression patterns occurring at the major *H. glycines* resistance locus, *resistance to heterodera glycines 1* (*rhg1*).[22,23] Transcriptional mapping studies of the *rhg1* locus have identified α-SNAP/Sec 17 as a highly expressed component.[23,24] α-SNAP is homologous to the *Saccharomyces cerevisae Secretion* (*Sec*) gene, *Sec 17* (α-SNAP/*Sec 17*) that functions in secretion.[1] In *S. cerevisiae*, mutants of *Sec 17* (*sec 17*), accumulate 50 nm vesicles that cannot fuse with a target membrane leading to the failure of the cells to transport the cargo protein carboxypeptidase Y.[1,25] This result demonstrates that α-SNAP/Sec 17p functions in membrane fusion during anterograde transport. Invaluable to the study of the *rhg1* locus in *G. max* has been the availability of the genetic mapping data and its sequenced genome.[22,26-29] Subsequent work has shown the *rhg1* locus contains multiple copies of α-SNAP/*Sec 17*, but no clear understanding of a defense role had been obtained.[30,31] However, Matsye et al.[23,24] and Sharma et al.[9] demonstrated a clear role for Gm-α-SNAP in defense of *G. max* to *H. glycines* parasitism. The identification of α-SNAP/*Sec 17* as a resistance gene in *G. max* strengthens the observation that identified SNARE functioning in defense in the plant genetic model *A. thaliana*.[5,9,24]

Genetic identification of the importance of secretion

Secretion is an orderly stepwise process that has been demonstrated through the genetic identification of the *Sec* and related genes in *S. cerevisiae* (Fig. 2).[1,32-35] Functional equivalents (homologs) subsequently have been identified in all eukaryotes.[2,36-43] A core set of proteins, known as Soluble NSF Attachment Protein REceptor (SNARE) is one macromolecular part of the membrane fusion apparatus (Fig. 2). The components of SNARE include syntaxin (SYP)/*Suppressor of sec 1* (*SSO1*), a gene homologous to *A. thaliana PEN1*. 5 Other SNARE components include synaptobrevin (SYB)/YKT6/*SEC22* and SNAP-25/*SEC9*.[44-49] The SNARE proteins tether the vesicle to the target membrane. Mammalian uncoordinated-18 (MUNC18/*SEC1*), also known as SM, may inhibit or facilitate fusion.[50,51] Synaptotagmin (SYT)/*Tricalbin-3* (*TCB3*) is believed to serve as a calcium sensor. SNARE metabolism, including its disassembly is mediated by two additional proteins including α-SNAP/Sec 17p and the ATPase N-ethylmaleimide-sensitive factor (NSF)/Sec 18p.[1,3] The entire SNARE complex can be biochemically isolated as part a larger 20 S particle, including α-SNAP/Sec 17p and NSF/Sec 18p, that mediates secretion.[52,53] Complimentary studies in animal systems investigating pathogenesis have identified botulinum and tetanus microbial neurotoxin effectors that target SNARE components and thus inhibit secretion.[54-61] The effect of the neurotoxins is paralysis. Similar types of effectors are also being identified in plants leading to impaired functionality of 20 S components during defense, confirming its importance in the process of defense.[62,63]

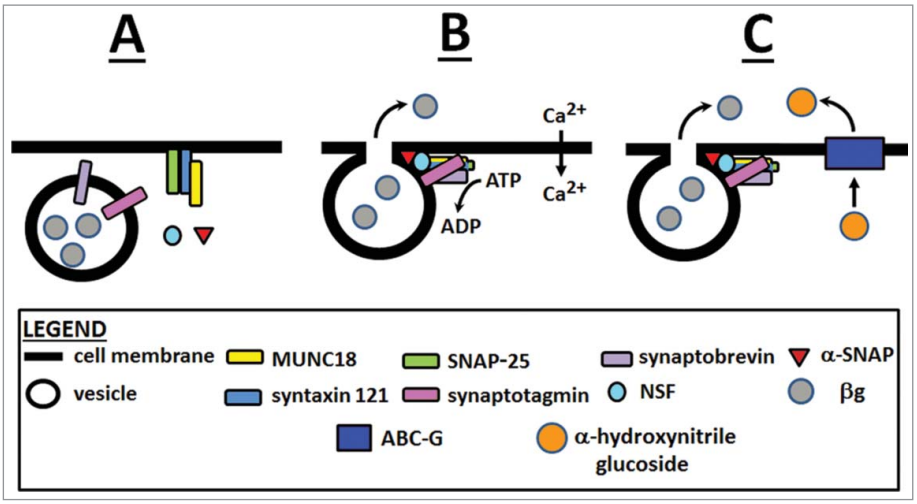

Figure 2. The process of membrane fusion involving the regulon. The involved proteins include SYP121 (PEN1/Sso1p), MUNC18/Sec 1p, SNAP-25/Sec 9p, SYB/VAMP/Ykt6p/Sec 22p), SYT/Tcb3p, NSF/Sec 18p and α-SNAP/Sec 17p, composing the 20 S particle. (A) the secretory vesicle, containing membrane fusion proteins comes into close association with target membrane proteins. (B) membrane fusion and release of cargo. (C) conjugated glucosides are transported through an ABC transporter in the vicinity of its secreted glucosidase resulting in activation of the glucosidase.[3]

α-SNAP/Sec 17 relates to plant defense by its association with SNARE during membrane fusion

The observation that Gm-α-SNAP functions in *G. max* defense to *H. glycines* has introduced questions regarding its activity. The observations that have been made regarding α-SNAP/Sec 17p is that it possesses diverse roles based on the cellular milieu. Through biochemical experiments Söllner et al.[52] have identified a number of SNARE-related proteins that are specific for a certain vesicle or target membrane, but requires α-SNAP/Sec 17p for fusion. This work has indicated α-SNAP/Sec 17p is a universal component of constitutive and regulated membrane fusion with variations in SNARE composition dictating specificity.[52] In examining neuronal exocytosis, Barszczewski et al.[62] have identified clusters of α-SNAP/Sec 17p at the plasma membrane revealing the addition of NSF/Sec 18p facilitates exocytosis. Schwartz and Merz[64] have shown α-SNAP/Sec 17p can rescue a SNARE complex that is stalled in its ability to complete fusion, identifying the central role of α-SNAP/Sec 17p in membrane fusion. Lobinger et al.[65] have examined α-SNAP/Sec 17p further. The experiments have demonstrated α-SNAP/Sec 17p, along with Sec 1p (Munc18) (SM), actually accelerates fusion that is engaged by SNARE while also protecting it from disassembly by NSF/Sec 18p. Yu et al.[66] have determined that the amount of SNARE-SM components in the cell are important for faithful fusion to occur. Yu et al.[66] have also determined that for secretion to occur efficiently, the components require macromolecular crowding which is a condition known to influence the thermodynamic and kinetic behaviors of macromolecules.[66] Experiments presented by Zick et al.[67-69] have revealed the high concentration of purified components can overcome the requirement of lipids for progression to fusion under physiological conditions. This point is important because even in other systems including *Drosophila*, the balance of α-SNAP/Sec 17p and NSF/Sec 18p concentrations are important for secretion even when overexpressed.[42] In other experimental systems, α-SNAP/Sec 17p works with NSF/Sec 18p to facilitate and disassemble all types of SNARE complexes after membrane fusion.[70-73] Therefore, the experimental evidence demonstrates α-SNAP/Sec 17p has multiple functions prior to, during and after the process of membrane fusion and that it works at all sites of fusion of membrane-bound structures that utilize SNARE.

Regulon components homologous to the SNARE component PEN1 exhibit co-regulation

A number membrane-bound structures exist within the vesicle transport system.[4] These structures include the endoplasmic reticulum (ER), conserved oligomeric Golgi (COG) complex, *trans*-Golgi network/early endosome (TGN/EE), homotypic fusion and protein sorting (HOPS) complex, the class C core vacuole/endosome tethering (CORVET), exocyst, trafficking protein particle (TRAPP) I–III complexes, Golgi-associated retrograde protein (GARP) complex, endosome-associated retrograde protein (EARP) complex, depends on SLY1-20 (Dsl1) complex and plasma membrane (PM). For details, please refer to Vukašinoví and Žárský.[4] These structures utilize different macromolecular protein complexes to facilitate their interactions and fusion events.

SNARE, benefitting from having the longest experimental history, has had its core components identified decades ago (Fig. 2).[3] While the proteins function effectively to mediate fusion, comparatively little is understood regarding whether the genes influence each other's expression (co-regulation). An early attempt made in *S. cerevisiae* attempted to understand whether the SNARE genes exhibited co-regulation.[74] In this study, it has been shown that *sec* mutants did not exhibit coordinated suppressed transcriptional activity of the other *Sec* genes.[74] However, technical limits of the time may have complicated such observation. In contrast, engineering SNARE components for constitutive induced expression has been shown to lead to induced expression of other SNARE components.[75] Furthermore, experiments in other biological systems have shown ε-COP overexpression can overcome a

temperature-dependent suppression of α-COP expression.[76] Similar results have been shown for the ER to Golgi component (ERGIC).[77] The observation of co-regulated gene expression of vesicle fusion components has held up to genomics-level scrutiny.[78] These observations are consistent with those made in the *G. max-H. glycines* pathosystem.[7,9] The question in relation to SNARE became how extensive is co-regulated gene expression during plant defense?

Regulon components homologous to β-glucoside PEN2 exhibit co-regulation

Experiments have been presented showing that the co-regulation of defense-related genes is not limited to the vesicle transport system components. Work done in *Lotus japonicus* has shown that LjBGD7, a root expressed PEN2 β-glycosidase homolog, is related to α-hydroxynitrile glucosidase.[79] LjBGD7 acts to produce hydrogen cyanide (HCN), functioning effectively in defense.[79] Furthermore, LjBGD7 is co-regulated with the cytochrome P450 protein LjCYP79D4. 79. In *L. japonicus*, Lj*CYP79D4* has increased relative levels of expression exclusively in the roots where LjBGD7 occurs.[79] In related studies, Morant et al.[80] demonstrated the co-expression of α-hydroxynitrile glucoside and their cognate hydrolyzing α-hydroxynitrile glucosidase. Furthermore, the heterologous expression of a *Manihot esculenta* (cassava) *CYP79D2* in *L. japonicus*, results in cyanogenic α-hydroxynitrile glucoside accumulation.[79] The experiments have clearly identified that genes functioning in α-hydroxynitrile glucoside production and metabolism are co-regulated.

Regulon components homologous to the ABC transporter PEN3 exhibit co-regulation

The ATP binding cassette (ABC) transporter regulon component PEN3 delivers metabolites through various membranes. While comparatively little is known about ABC transporters in plants, work in animal systems have demonstrated they are capable of co-regulating the expression of other genes relating to their own function.[81,82] However, while evidence exists for the ability of the regulon components to effect co-regulation the scope and breadth had not been examined in detail. The co-regulation of pathway components leading to the production of secondary metabolites is not limited to PEN2. For example, The *Nicotiana benthamiana* terpene synthases TPS10 and TPS14 are co-regulated with the two cytochrome P450s (CYP71B31 and CYP76C3) in flowers at anthesis.[83] Furthermore, the protein products are found in the endomembrane system resulting in monoterpene alcohol linalool production.[83] Therefore, it is likely that other secondary metabolite pathways are important to defense in the *G. max-H. glycines* pathosystem (Fig. 3).

Callose is a structural defense element whose genetic components exhibit co-regulation

The recent identification of callose existing at sites of resistance in *G. max* roots parasitized by incompatible *H. glycines* is consistent with numerous observations of its presence at defense sites in many plant pathosystems.[9,84-110] These studies have provided extensive support that callose performs an active role in defense. Consistent with these observations, other functional studies have demonstrated the active role callose plays as it participates in defense.[111-117] However, in contrast, very limited studies have shown that callose may not accumulate to appreciable levels at defense sites or participate in defense at all.[118,119]

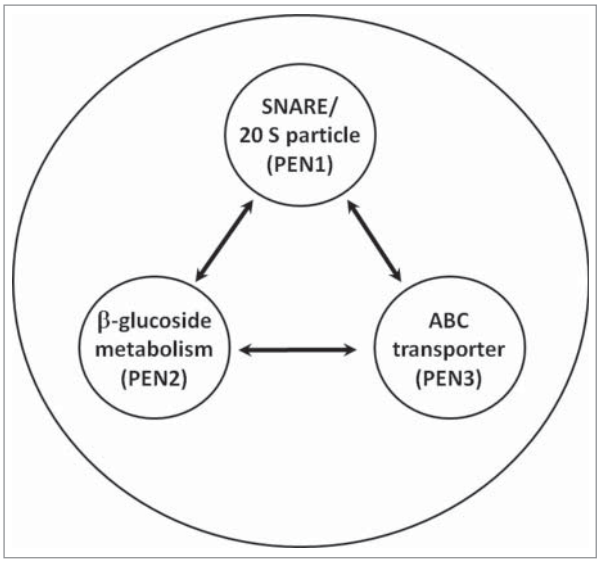

Figure 3. Components of the regulon, composed of PEN1, PEN2 and PEN3, are under co-regulation.[9] Other components functioning within this apparatus are represented by the outer ring.

Callose is a glucose-derived polysaccharide polymerized mainly by callose synthase (CaLS) or GLUCAN SYNTHASE–LIKE (GSL) enzymes, forming β-1,3- and lesser amounts of β-1,6-branches. Conversely, β-1,3-glucanases depolymerize callose. During defense, plants deposit a plate-like structure referred to as a cell wall apposition that is also known as a papillae. It is these papillae that contain callose, among other materials, that are thought to provide a physical barrier to the establishment of an invasion site for the pathogen.[120] The development of papillae at the molecular level requires the vesicle transport system.[115] For example, in *A. thaliana*, papillae formation as a consequence of infection by *Blumeria graminis* f.sp. *hordei* is mediated by PEN1.[115] The transport of callose has been suggested to occur through multivesicular bodies. However, conclusive evidence has not presented.[114,115,120] These observations directly link vesicle transport to the delivery of callose at infection sites, consistent with the observations of Sharma et al. 9 in the *G. max-H. glycines* pathosystem.

Co-regulated gene expression occurring during defense is extensive

The regulon is likely to be an apparatus having a broad structural, biochemical and physiological basis. This concept is supported through gene expression experiments. For example, gene expression experiments examining nematode-parasitized cells undergoing a defense response identified many expressed genes that would be defense candidates in *G. max* that would be predicted to associate with the regulon (Fig. 4; Table 1; Table S1).[23]

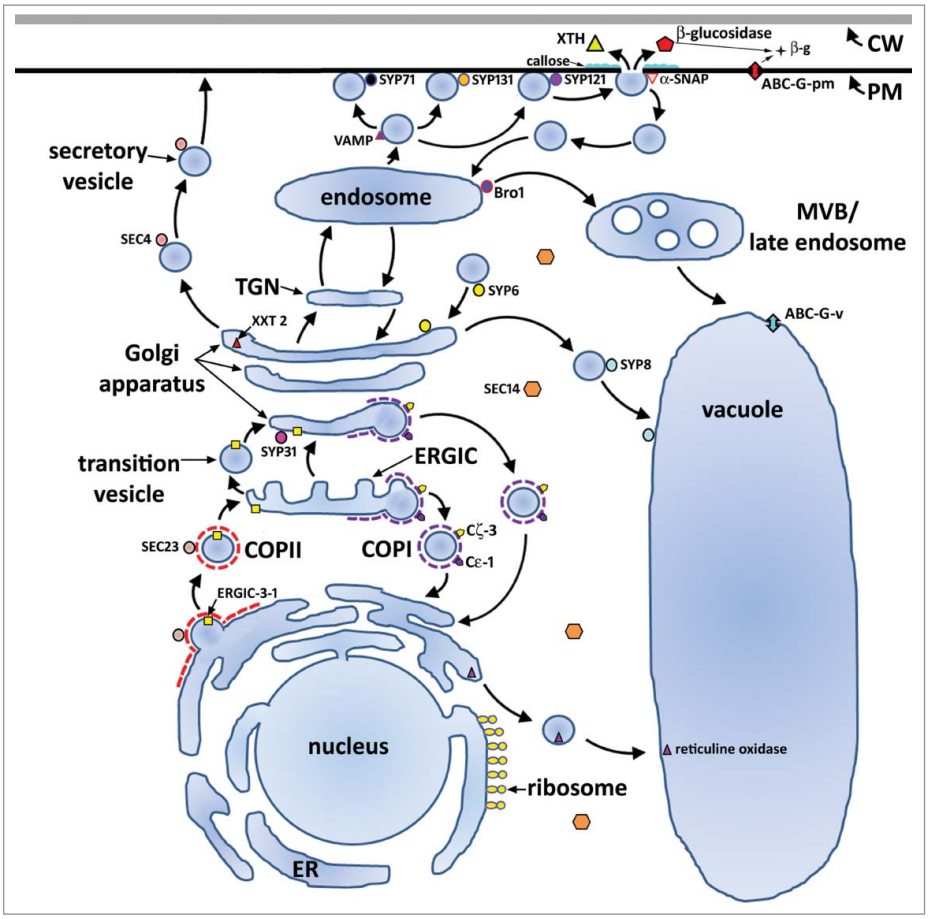

Figure 4. Proteins shown to function during the defense process *G. max* has to *H. glycines* parasitism. The previously identified regulon components including SYP31/Sed5p, PEN1 (SYP121/Sso1p), MUNC18/Sec 1p, SNAP-25/Sec 9p, SYB/VAMP/Ykt6p/Sec 22p, SYT/Tcb3p, NSF/Sec 18p and α-SNAP/Sec 17p, β-glucosidase, ABC-G. Presented here, Sec14, Sec 4, Sec 23, Bro1, SYP6, SYP131, SYP71, SYP8, Bet1, Cε, Cζ, ERGIC3.[7,9,24]

To examine this concept further, experiments have been done examining candidate defense genes functioning in different defense pathways. These genes include *G. max* homologs of Sec 14-1, Sec 4-6, Sec 23-5, Bro1-1, SYP6-1, SYP131-1, SYP71-6, SYP8-2, Bet1-1, ε-COP-1, ζ-COP-3 and ERGIC-3-3. Genetic pathways that associate with the regulon have also been examined, including those incorporating reticulon oxidase-40 (Fig. 5; Table S2), xyloglucan xylosyltransferase 2-5 (Fig. 6; Table S2) and galactinol synthase-3 (Fig. 7; Table S2). As a control to show the expression of any gene will not result in engineered resistance, we have selected a *G. max* homolog of the *A. thaliana APETALA2* (*AP2*) Gm-Apetala2-1 (Glyma01g44130).[121] In *A. thaliana, APETALA2* (*AP2*) is the founding member of a family of transcription factors originally identified to regulate flower development.[121,122] Further analysis has shown Gm-Apetala2-1 exhibits homology to the *A. thaliana AP2* homolog *TINY*.[123] Ectopic expression of *A. thaliana TINY*, caused by a semidominant dissociation insertion mutation, affects fertility, plant height and the elongation of hypocotyls.[123,124] However, Gm-Apetala2-1 is not expressed within the control pericycle cells or syncytia undergoing the process of defense and therefore it would not be expected to contribute to the process (Table S1). A qPCR analysis shows the genetically engineered plants exhibit induced candidate gene expression (Fig. S1). An analysis of genetically engineered roots infected with *H. glycines* reveals suppressed parasitism as compared to control plants (Fig. 8; Table S3).

The regulon apparatus functions broadly in defense

It is not surprising that the presented genes, identified to be expressed within the syncytium undergoing the process of defense, have roles in defense. The *S. cerevisae* Sec 14p is a cytoplasmic protein required for secretory vesicle formation from the Golgi apparatus and is encoded by a phosphatidylinositol (PtdIns) transfer protein (PITP).[25,33,125,126] Sec 14p also functions as a sensor of the PtdIns/phosphatidylcholine (PtdCho) ratio in Golgi by directly regulating PtdCho biosynthesis.[127] The *S. cerevisae* Sec 4p is a Rab GTPase that functions to regulate the assembly of the exocyst through its interaction with Sec 15p.[128,129] The exocyst is a protein complex functioning in vesicle transport and membrane fusion between post-Golgi secretory vesicles to the plasma membrane. This interaction provides a regulatory function, occurring upstream of SNARE-mediated membrane fusion.[130]

The *S. cerevisae* Sec 23p is a cytosolic protein that functions in concert with Sar1p, Sec 24p, Sec 13p and Sec 14p as the minimal unit for COPII carrier formation.[131-133] An important part of this process is phosphorylation of COPII coats.[131-133] During vesicle formation, Sec 12p-mediated activation of Sar1p results in its recruitment to the ER membrane which results in recruitment of Sec 23p-Sec 24p. This action results in the formation of the inner COPII prebudding complex which allows export protein capture.[131-133]

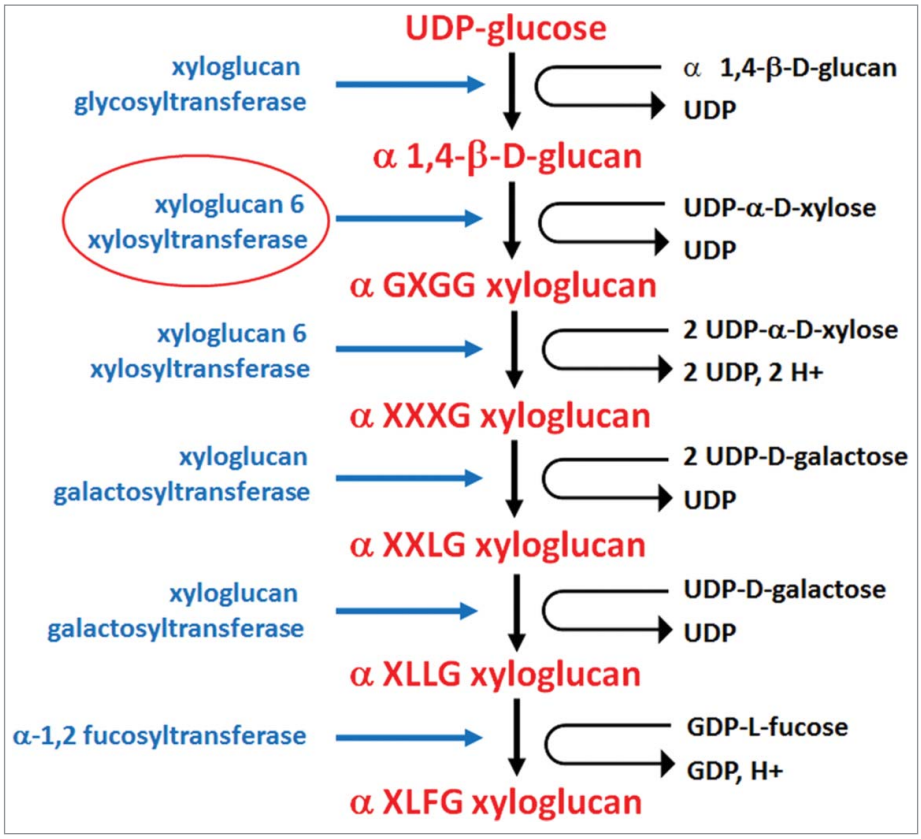

Figure 5. Enzymes functioning in xyloglucan biosynthesis are xyloglucan glycosyltransferase (CSLCS4) (E.C. 2.4.1.168), xyloglucan 6 xylosyltransferase (XXT1, XXT2) (E.C. 2.4.2.39), xyloglucan 6 xylosyltransferase (XXT5) (E.C. 2.4.2.39), xyloglucan galactosyltransferase (KATMARI1) (no E.C. #), xyloglucan galactosyltransferase (no E.C. #), α-1,2 fucosyltransferase (E.C. 2.4.1.69). Xyloglucan terminology: X, glucose residues substituted by α-D-xylose; G, non-substituted glucosyl units within a xylosylglycan; L, glucose residues substituted by α-D-xylose and β-D-galactose; F, glucose residues substituted by α-D-xylose, β-D-galactose and α-L-fucose. Supplemental data is provided (Table S2).[267-275]

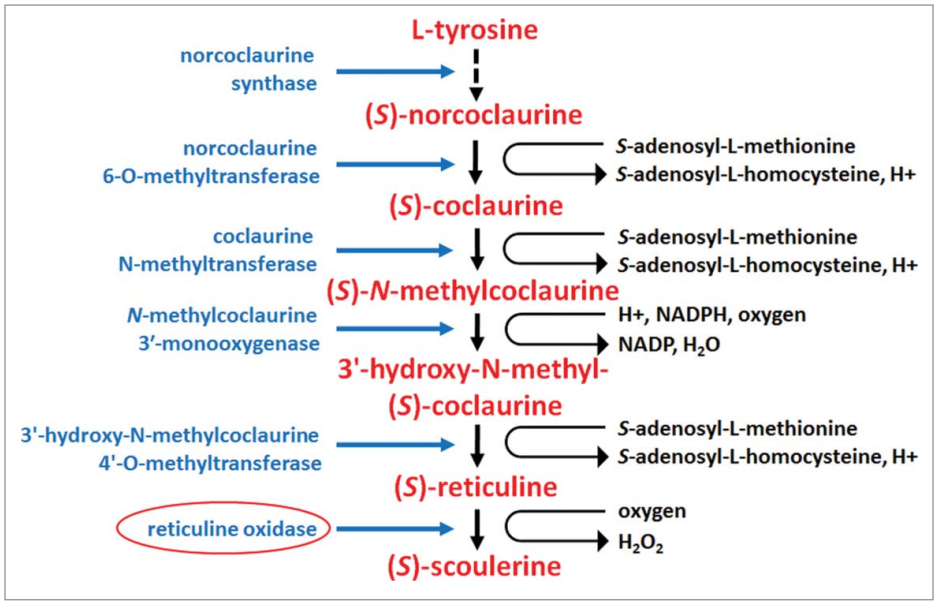

Figure 6. Enzymes functioning in reticuline metabolism. The biogenesis of (S)-reticuline begins with (L)-tyrosine. While a number of subsequent intermediates are made through different pathways, they converge on the production of (S)-noroclaurine via the enzymatic activity of noroclaurine synthase (E.C. 4.2.1.78). Subsequent enzymatic steps include norcoclaurine 6-O-methyltransferase (E.C. 2.1.1.128), coclaurine N-methyltransferase (E.C. 2.1.1.140), the CYP80B1 *N*-methylcoclaurine 3′-monooxygenase (E.C. 1.14.13.71), 3′-hydroxy-N-methylcoclaurine 4′-O-methyltransferase (E.C. 2.1.1.116), berberine bridge enzyme tetrahydroprotoberberine synthase (reticuline oxidase) (EC 1.5.3.9). (S)-reticuline is an intermediate whose subsequent metabolism results in the generation of a wide number of isoquinoline alkaloids that are an important response to pathogen attack. Supplemental data is provided (Table S2).[229-237]

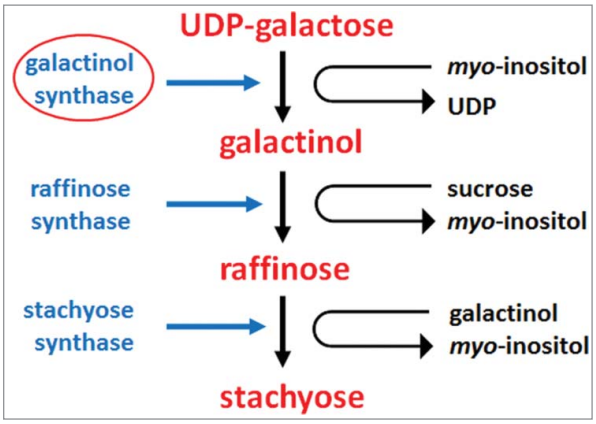

Figure 7. Enzymes functioning in stachyose metabolism. The enzymes involved include galactinol synthase (E.C. 2.4.1.123), raffinose synthase (E.C. 2.4.1.82) and stachyose synthase (E.C. 2.4.1.67). Supplemental data is provided (Table S2).[248-253]

The *S. cerevisae* Bro1p is endosome-associated, functioning in the multivesicular body[134,135] Bro1p is a cytoplasmic protein named for its ~160 aa domain that interacts with the endosomal sorting complex required for transport complexes (ESCRT).[136-138] Mutation of residues within this Bro1p domain interferes with its ability to localize to endosomes.[139] An analysis of Bro1p has been performed, revealing it contains a tetratricopeptide repeat (TPR)-like structure that is known to function as a putative ESCRT-III binding site.[139] Mutant analysis of Bro1p binds sucrose non-fermenting 7 protein (Snf7p) (ESCRT-III). 135 Bro1p-like proteins also function in plants.[140-143]

A number of syntaxins that would function in related ways during membrane fusion function in defense. SYP6 has been shown to localize to the *trans*-Golgi network (TGN) and binds to α-SNAP.[144-152] SYP131 is related to t-SNARE that functions in plant-arbuscular mycorrhizal interactions.[153] SYP131 is closely related to SYP132, shown to function in symbiotic interactions.[154] SYP71 localizes mainly to the plasma membrane and also the cell plate, endosome and ER.[155,156] SYP71 can also be subverted during virus infection to facilitate its pathogenicity.[157] SYP8 (At-SYP51) has been shown to accumulate on tonoplast and small prevacuolar compartments and co-localize with TGN markers and only partially with endocytic compartments with roles in vacuolar sorting, exocytosis and endocytosis.[158]

The *S. cerevisae* Bet1p functions in anterograde transport from the ER to Golgi, found on Golgi membranes and is recruited to COPII vesicles by Sec 24p. Bet1p activates Bos1p with this interaction regulated by the small GTP-binding protein Ypt1p. Bet1p overexpression overcomes mutants of *sec 35*.[159-169]

Sec 35p is novel, implicated in the tethering ER-derived vesicles.[170] This tethering occurs at the Golgi apparatus. 170 Sec 35p binds to the hydrophilic Sec 34p to form a 480 kD complex that facilitates vesicle traffic.[170]

The ε-COP and ζ-COP proteins are part of the COPI coat, a 700 kD structure composed of α, β, β' γ, δ, ε and ζ subunits that function in retrograde transport between the Golgi and ER. Furthermore, COPI functions in the maturation of endosomes and autophagy.[76,171-178] Among these intermolecular interactions, ε-COP (Sec 28p) has been shown to stabilize α-COP (Ret1p).[76] This function was observed out of work done in a number of studies that showed that the proteins composing COPI (Ret1p [α-COP], Sec 26p [β-COP], Sec 27p [β'-COP], Sec 21p [γ-COP], Ret2p [δ-COP], Ret3p [ζ-COP]) are essential for viability at all temperatures with ε-COP being temperature sensitive.[76,172,179,180]

The tubulovesicular ERGIC structure performs an important role in the sorting of proteins destined to be delivered to various cellular compartments. The ERGIC appears to be a stable compartment for COPII-dependent delivery of materials that are subsequently transported to the Golgi. ERGIC3 is a protein involved in this process.[77,181-191]

Secretion functions to drive a defense response

The demonstrated function of the secreted XTH and α-hydroxynitrile glucosidase proteins in defense indicated that the physiological needs of *G. max* resistance *to H. glycines* parasitism ran deep into the central metabolic processes of the parasitized cell. From genomics analyses performed in *A. thaliana*, a fundamental shift in metabolism during plant defense to pathogen attack has been long known and is consistent with observations made in the *G. max-H. glycines* pathosystem.[17,23,192] In agreement with these observations is the demonstration of the involvement of the membrane fusion apparatus that would deliver XTH and α-hydroxynitrile glucosidase to the cell periphery and an ABC transporter that would likely deliver conjugated metabolites to the site of parasitism.[7,9] Therefore, it appears that the defense of *G. max* to *H. glycines* parasitism involves altering the structure of the cell wall through the enzymatic activities of XTH, impeding the ability of *H. glycines* to make a functional syncytium. The defense process also involves the delivery of secondary metabolites to combat pathogen effectiveness. Furthermore, other metabolites are probably important to the defense process.

Hemicellulose: xyloglucan metabolism functions in defense in the root

Cytological observations of *G. max* undergoing the process of resistance to *H. glycines* have shown the cells engaged in the resistant reaction are limited in their ability to expand.[193-196] These observations have indicated cell wall composition and modification are important to the defense process. A large proportion of the cell wall is composed of hemicellulose, a network that functions as a structural stabilizer. The majority of hemicellulose is composed of xyloglucan. Xyloglucan biosynthesis occurs by the stepwise activities of Golgi-localized enzymes and the delivery of these complex polysaccharides are generally believed to occur through bulk flow mediated by secretory vesicles (Fig. 5).[197-199] The location of these materials in the Golgi, along with the secretion of these materials into the apoplast strenghtens the importance of the vesicle transport system in defense. Upon polymerization, the hemicellulose strand can be modified by xyloglucan endotransglycosylase/hydrolase (XTH) (E.C. 2.4.1.207), an ancient gene family found in all plants that may predate land colonization.[200-204] A *G. max* XTH has been shown to be highly expressed in *H. glycines* parasitized root cells undergoing defense and functions in the

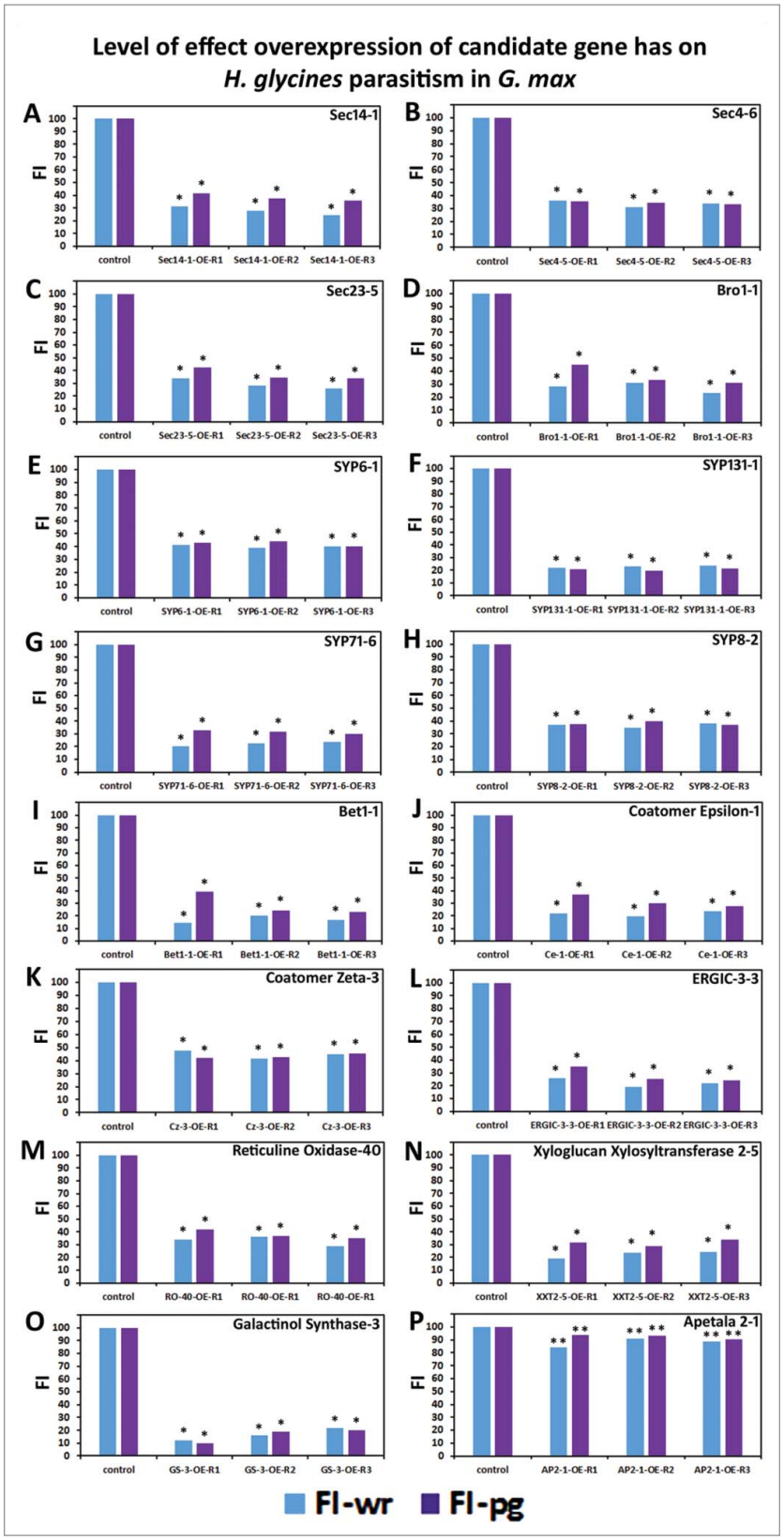

Figure 8. The overexpression of candidate defense genes in *G. max* leads to suppressed parasitism by *H. glycines*. (A) Sec 14-1; (B) Sec 4-6; (C) Sec 23-5; (D) Endosomal Targeting BRO1-Domain-1; (E) SYP6; (F) SYP131-1; (G) SYP71-6; (H) SYP8-2; (I) Bet1-1; (J) Coatomer ε-1; (K) Coatomer ζ-3; (L) ERGIC-3-3; (M) RO-40; (N) XXT 2-5; (O) GS-3; (P) AP2-1. The overexpression experiments present the outcome as the female index (FI) which is a percent of infection in relation to the control plants. wr, analysis examining cysts per whole root sample. pg, analysis examining cysts per gram of root tissue. * Statistically significant, $p < 0.05$; ** Not statistically significant, $p > 0.05$. Analyzed by Mann–Whitney–Wilcoxon [MWW] Rank-Sum Test. [276] Supplemental data is provided (Table S3).

defense process.[7,23] XTHs function to restructure cell walls through cell wall loosening or intercalation of new xyloglucan.[205-209] These two different functions of XTH are possible because it serves as a xyloglucan *endo*-transglucosylase (XET) where xyloglucan polymers are cleaved and joined to different xyloglucan chains.[210] In contrast, the XEH activity of XTH hydrolyzes xyloglucan polymers.[209] Regarding rapid cell wall expansion, auxin is known to regulate this process.[211-213] XTH has a signal peptide and the protein can be *N*-glycosylated, indicating processing through the secretory pathway.[214-220] The overexpression of XTH in *Populus* sp. results in the initial shortening of xyloglucan chain length, providing mechanistic insight into how plants can use XTH to limit cellular expansion during a defense reaction.[7,221]

The demonstration that XTH is highly expressed during the *G. max* defense process toward *H. glycines* parasitism has indicated that the metabolic processes that lead to the generation of the xyloglucan would also be important.[23] In examining this result further, we have identified that mRNA of 4 genes encoding the enzymes that function in xyloglucan biogenesis and metabolism are present in syncytia undergoing he process of defense (Table S2). The enzymes that function in xyloglucan biogenesis and metabolism have been shown to localize to the Golgi apparatus, clearly implicating the importance of a functional secretion system to defense. To test this hypothesis, we have cloned a *G. max* homolog of XXT and overexpressed it in a susceptible genotype, resulting in impaired parasitism (Fig. 7; Table S3). The results emphasize the importance of hemicellulose metabolism to defense. Furthermore, the importance of a functional secretion system is demonstrated since the proteins are processed through the secretory pathway with some functioning within the Golgi apparatus.

Reticuline metabolism functions in defense in the root

Plants produce an astonishing number of secondary metabolites, numbering over a hundred thousand in total.[222] One of these important secondary metabolites, whose subsequent metabolism leads to the production of defense molecules, is (S)-reticuline. (S)-reticuline is a benzylisoquinoline alkaloid belonging to a family of secondary metabolites consisting of more than 2,500 different known small molecules.[223,224] Some of the early biochemistry regarding reticuline has been worked out by understanding its basic structure.[225-228] The biogenesis of (S)-reticuline begins with (L)-tyrosine. While a number of subsequent intermediates are made through different pathways, they converge on the production of (S)-noroclaurine.[229-237] (S)-reticuline is an intermediate whose subsequent metabolism results in the generation of a wide number of isoquinoline alkaloids that are an important response to pathogen attack.[230] A second pathway that leads to (S)-reticuline biosynthesis is also known to have its origins with (L)-tyrosine and leading to (S)-norlaudanosoline production, but the pathway has been described in mammalian cells.[238,239]

Reticuline oxidase (RO) has also been described as berberine bridge enzyme (BBE).[227,229,240] RO originally had been shown to be associated with a particle within the cell.[228] RO has a signal peptide, indicating it is targeted to the ER.[230] In *Berberis wilsoniae* var. subcaulialata, RO has been shown to localize within vesicles with other enzymes involved in the biosynthesis and metabolism of (S)-reticuline.[241] The RO-containing vesicles have been shown to compose a low number of detectable polypeptides (~20), indicating that vesicles with different types of cargo function in defense. 241 In opium poppy (*Papaver somniferum* cv Marianne), RO has been shown to localize in the companion cells of phloem.[242] Furthermore, the subcellular localization of RO is the ER.[223,241,243,244] This observation is consistent with the presence of a signal peptide for the *G. max* RO-40 (Fig. S2). After elicitor treatment, the ER undergoes a major ultrastructural arrangement becoming dilated and producing vesicles that later fuse with the vacuole.[241,243,244] Notably, a vacuolar sorting determinant is present in the N-terminus of the RO protein.[243] The production of the metabolic intermediates leading to the production of to (S)-reticuline and the structural requirements appear to be shared between different plant groups.[230,243-246] We have identified *G. max* homologs of genes functioning in reticuline biogenesis and metabolism (Table S2). Further work using *Coptis japonica* as a model revealed that an ABC transporter was responsible for delivering berberine to its site of function.[247] This observation provided mechanistic insight into the delivery of secondary metabolites synthesized by RO to the vacuole. As observed by Sharma et al.,[9] it is likely that a number of different ABC transporters function during the defense of *G. max* to parasitic nematodes.

Stachyose metabolism functions in defense in the root

Stachyose is produced in an enzymatic process beginning with galactinol synthase (E.C. 2.4.1.123) action involving myo-inositol and UDP-D-galactose.[248] The process leads to the production of galactinol. 248 The only known role for galactinol is for the production of larger soluble oligosaccharides such as raffinose, stachyose, and verbascose.[249] However, galactinol has been shown to be induced in its production during a defense response in roots and functions in signaling during systemic acquired resistance.[250] Subsequent enzymatic activities of raffinose synthase (E.C. 2.4.1.82) and stachyose synthase (E.C. 2.4.1.67) result in the production of stachyose (Fig. 7).[251-253] The enzymes for the biogenesis of stachyose have been identified in *G. max* (Table S2).

The framework of defense involving the co-regulation of regulon in roots

The framework of defense as *G. max* protects itself from its major pathogen, *H. glycines* is based off of cytological observations and genetic experiments relating its *rhg1* locus.[7,9,22,24,28,30,31,193] These results put into context the identification of an *H. glycines* effector that binds *G. max* α-SNAP/Sec 17 that would have the function of disarming its defense response.[63] Evidence has been presented showing that the transcriptional activity of these genes influences each other, indicative of co-regulation.[7,9,24] The co-regulation of components of a structural and biochemical element helps in explaining why so many genes are transcriptionally active in the cells destined to undergo the process of resistance. Additional experimental evidence is presented here to support those observations. The experiments show how the expression of these genes

Table 1. A summary of *G. max* candidate gene expression.

Gene	Accession	Time point (dpi) 0*	3	6
Sec14-1	Glyma14g08180	N/M	N/M	M
Sec4-6	Glyma20g23210	N/M	N/M	M
Sec23-5	Glyma18g00670	N/M	M	M
Bro1-1	Glyma02g10910	N/M	M	M
SYP6-1	Glyma17g07100	N/M	M	M
SYP131-1	Glyma12g32100	N/M	M	M
SYP71-6	Glyma19g36410	N/M	N/M	M
SYP8-2	Glyma14g02120	M	M	M
Bet1-1	Glyma06g10370	M	M	M
Cε−1	Glyma09g03500	M	M	M
Cζ−3	Glyma15g01150	M	M	M
ERGIC-3-3	Glyma13g01920	M	M	M
RO-40	Glyma15g14040	N/M	N/M	M
XXT 2-5	Glyma19g39760	N/M	N/M	M
GS-3	Glyma19g41550	N/M	N/M	M
AP2-1	Glyma01g44130	N/M	N/M	N/M

Footnote. Laser microdissection has been used to collect control cells (pericycle) at 0 days post infection (dpi) and *Heterodera glycines*-induced syncytia undergoing the process of resistance at 3 and 6 dpi. * Uninoculated roots have been used to obtain pericycle cells which have served as the source of the control mRNA samples. M (red) probe has been measured. N/M (blue), probe has not been measured. For details, please see Table S1.

recapitulates the natural defense process that leads to suppressed parasitism by *H. glycines* in *G. max*. Furthermore, the identification of callose at the site of defense expands the biochemical and structural environment pertaining to defense, unifying work done in other plant pathosystems.[5,6,8,15,16,115,254,255] The work likely has identified a genetic program that is functional in other plants against other pathogens or could be altered to facilitate symbiotic interactions. As a polymer, callose has the capacity of rapid building block mobilization to infection sites with their subsequent polymerization into structures that can impede pathogenesis.[9,255-257]

Methods

The data that has been presented in this review has been obtained through the published methods in Sharma et al. 9 Laser microdissection (LM) has been used to collect control cells (pericycle) at 0 days post infection (dpi) and *Heterodera glycines*-induced syncytia undergoing the process of resistance at 3 and 6 dpi. Uninoculated roots have been used to obtain pericycle cells which serve as the source of the control mRNA samples. Microarray hybridizations have been run in triplicate (arrays 1-3). The hybridizations have used probe derived from RNA isolated from LM-collected syncytia obtained from 3 independent replicate experiments. For robustness, the experiments have been run independently in two different *H. glycines*-resistant genotypes. For the gene to be considered expressed at a given time point (3 or 6 days post infection [dpi]), probe signal must have been measurable above threshold on all three arrays for both *G. max*$_{[\text{Peking/PI 548402}]}$ and *G. max*$_{[\text{PI 88788}]}$ (6 total arrays), $p < 0.05$. The Bioconductor implementation of the standard Affymetrix® detection call methodology (DCM) analysis consists of four steps, including (1) removal of saturated probes, (2) calculation of discrimination scores, (3) p-value calculation using the Wilcoxon's rank test, and (4) making the detection call (present [$p < 0.05$]/marginal [$p = 0.05$]/absent [$p > 0.05$]). In the analysis presented here, the probe set is accepted as detecting probe if $p < 0.05$. The PCR and qPCR primers used in the studies are provided (Table S4). The qPCR data has been obtained using $2^{-\Delta\Delta C_T}$ to calculate fold change (Fig. S2).[9,258] The female index (FI), is the community-accepted method to determine the effect a condition has on *H. glycines* parasitism.[259] The results have been presented as female cysts per whole root mass and female cysts per gram (Table S3).

Diclosure of Potential Conflicts of Interest

The authors claim no potential conflicts of interest.

Acknowledgements

The authors thank Dr. Nancy Reichert for providing culture room space that is essential for the transgenic experiments. The Department of Biological Sciences is acknowledged for providing start-up support and teaching assistantships. Support from the Mississippi Soybean Promotion Board, Cotton Incorporated and Mississippi Agricultural and Forestry Experimental Station (MAFES) is acknowledged. Graduate students from the Department of Biological Sciences and Department of Biochemistry, Molecular Biology, Entomology and Plant Pathology contributing to this project and the genomics analyses but are not authors include Prachi Matsye, Prakash Niruala, Tineka Burkhead, Bisho Lawaju and Weasam Aljaafri. Undergraduate students greatly complimenting the research include Christina Jones, Suchit Salian, Nishi Sunthwal, Priyanka Gadre, Dollie Welch, Kim Anderson, Brittany Ginn, John Clune, Hannah Burson, Chase Robinson, Meghan Calhoun, Katherine McCracken, Kayla Moore, Madison Milhoan, Erin Curran, Neil Shannon, Austin Martindale, Keigero Fergusen, Ashley Dowdy, Taylor Henry, Hannah Stimson, Hannah Whitlock, Erin Ball, Henry Pittman, Shelby Janeski, Ashlee Vargason, Annedrea McMillan, Courtney Gagliano, Leslie Canale, Alison Antee, Hannah Miller, Adam Crittenden, Lauren Langston, Jamelle Vance, Carolyn Chacon, Emily Carter, Natalie Rentrop, Eileen Modzeleski, Erica Sowell, Chrissy Miller, Anna Bailey Britt, Chelsea Tittle, Robyn Beattie, Dejanie Dilworth, Aishwarya Dikshit, Harshini Sampathkumar, Samantha Rushing, Meagan Young, Kathryn Stiglet, Makenzie Miller, Morgan Urich, Chase Nash, Kyle Winston, Olivia Long, Jody Clark, Ana Simal, Katherine Thrash, Adrienne McMorris, Rebecca Waters, Haleigh Smith, Kelvin Blade, Jesse Austin, Alex Hammett and Maggie Kuhn. Amanda Lawrence, Imaging and Analytical Technologies Center, Mississippi State University is acknowledged. Dr. Wes Burger (MAFES) and Dr. Giselle Thibaudeau (College of Arts and Sciences) are acknowledged for their interest.

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