AUXIN IN PLANT DEFENSE REACTIONS: NEW INSIGHTS AND POTENTIAL APPLICATIONS FOR RESISTANCE BREEDING PROGRAMMES

Ram Prasad Mainali[1] and Sushil Nyaupane[2*]
[1]National Agriculture Genetic Resources Centre (NAGRC, National Genebank),
Nepal Agricultural Research Council (NARC), Khumaltar, Kathmandu, Nepal
[2]Faculty of Agriculture, Far Western University (FWU), Tikapur, Kailali, Nepal
*Corresponding author email: sunyaupane@gmail.com
Ram Prasad Mainali : https://orcid.org/0000-0002-4637-3676
Sushil Nyaupane : https://orcid.org/0000-0003-4818-6513

ABSTRACT

The role of auxin in growth and development in plants has been studied and documented since its discovery; however, its role in plant defense during pathogenic infection has been studied more recently. With the aim to provide new insights and update knowledge in this field of biology, this review was carried out focusing on the specific role of auxin and associated signaling against a wide array of bacterial and fungal pathogens, as special references. This review illustrates the role of auxin including indole-3-acetic acid, mechanism of auxin-responsive gene action, auxin signaling and associated crosstalk with other defense hormone pathways. Loads of evidence support that the biology of plants favoring repressed auxin signaling, auxin responsive defense gene activation and hormonal crosstalk with other associated defense hormones such as salicylic acid and jasmonic acid signaling pathway is vital for host defense against pathogens. As this paper unravels the role of auxin in plant defense, classical and molecular breeders would get new background insight into engineering plants with enhanced host plant resistance with the introduction of auxin responsive gene or manipulation of desirable auxin signaling or associated pathway. The current knowledge domain on plant defense during pathogen infection could be improved by considering multiple pathogens in nature, their interaction with other beneficial microbes and associated auxin manipulation in future research works.

Keywords: Host defense, pathogen, phytohormone, plant- microbe interactions, signaling.

INTRODUCTION

Plants are the suitable hosts for multitude of microorganisms. Plant surfaces and tissues, which are simply nutritive and with having sufficient moisture content, provide suitability to those fore -mentioned microorganisms (Elnahal *et al.,* 2022). Various microbe attacks on the plants and all of them have their unique mode of invasion, growth and reproduction in the host plants, although there are free-living microbes which simply don't need hosts. Although not all the microorganisms which comes to get contact with plants inflicts diseases to plants (Kunkel & Johnson, 2021). Based on their nature and behavior, microorganism can be grouped into three types i.e. beneficial, pathogenic and neutral to the host plant (Elnahal *et al.,* 2022). Different microorganisms have their own mode of invasion to the host plants. Viruses follow biotrophic mode of invasion whereas, fungus and bacteria exploit both biotrophic and necrotophic invasions. Biotrophs obtain nutrients without destroying host plant tissues whereas, necrotrophs do the opposite. Both of these phenomena cause their host plants to exhibit distinct and changed physiologies (Raman & Suryanarayanan, 2017). However, plant does its best to counter the invasion and effect of those microbes on its own accord through different mechanisms, popularly called as defense responses (Ye *et al.,* 2019).

Plant defense responses could be either primary (performed) or secondary (induced), which largely depend on the conditions. For the plant, first and foremost step to defend is to recognize the invading microorganism and microorganism derived elicitors. This defense response of the plant results the activation of ion fluxes, phosphorylation or dephosphorylation of proteins by enzymes, and formation of signaling molecules such as salicylic acid (SA), jasmonic acid (JA), ethylene, nitric oxide, adapter proteins, etc. This is further followed by sequences of signaling, ultimately regulates the expression of defense related genes. These all are responsible for further responses such as cell wall strengthening, accumulation of phytoalexins and several proteins, cellular death, etc. (Agrawal, 2018). Additionally, various plant hormones also play a role in plant-pathogen interaction (Burger & Chory, 2019), not only having impact to the crop biology but also to the microbes biology (Kunkel & Harper, 2018).

Auxin (from Greek word "auxein" meaning to grow) is a category of phytohormones synthesized by all higher plants. Indole-3-acetic acid **(**IAA) is the most abundant auxin in plants and synthesized from a tryptophan-dependent or a tryptophan-independent pathway (Mashiguchi *et al.,* 2011; Manu & Nemato, 2012). The role of auxin as a regulator of growth and development including role in seed germination has been studied for a long time (Lv *et al.,* 2021). This hormone directs various processes of the plant such as cell cycle, cell elongation, stimuli transfer and organogenesis, which governs the form, shape and strength of the plant. For example, IAA stimulates the cell wall loosening factors and facilitates elongation process determining a shape of the plant. This hormone directs various processes of the plant such as cell cycle, cell elongation, stimuli transfer and organogenesis, which governs the form, shape and strength of the plant. For example, IAA stimulates the cell wall loosening factors and facilitates elongation process determining the shape of the plant. It is involved in root growth regulations such as formation of adventitious roots (Liu *et al.,* 2012) and therefore accounted as an important constituent of tissue culture. Besides its long recognition as an actor of plant growth and development, modern research works have suggested its role in host plant defense against pathogens in the field of biology. It's quite interesting to mention that this hormone act as a microbial signaling molecule responsible for altering pathogen gene expression (Kunkel & Johnson, 2021).

Food crops, like all plants, are prone to biotic and abiotic stresses; this is mainly due to their immobile growing habit and so, plants should be able to tolerate these stresses in order to survive (Zhang & Friml, 2020). Pathogens, like bacteria, fungi, viruses, nematodes and herbivore pests are categorized as biotic stresses (Atkinson & Urwin, 2012; Kunkel & Johnson, 2021). These biotic stresses or parasites act on host plants during favorable environments and cause disease or undesirable phenotype and subsequent loss. The loss in various crops varies between 20 to 40 % due to biotic stressors whereas pathogens alone cause yield losses up to 16% globally (Savary *et al.,* 2012; Ficke *et al.,* 2018). During the infection process, factors causing biotic stresses such as bacteria and fungi enrich the auxin synthesis in the host plant and its level is directly related to the disease development, especially through the loosening cell walls to proliferate different pathogens (Ye *et al.,* 2019). As counteract, plants act against pathogens through a basal defense and host specific defense response, with auxin signaling is involved in both defense systems. Basal defense, also known as the first line of defense, activates on a plant when the plant recognizes microbial components, here microbe or pathogen associated molecular patterns (M/PAMP).

Upon pathogen attack, induced plant microRNA (miRNA) by pathogen effectors down-regulates the auxin signaling. The repressed auxin signaling restricts pathogen growth and assists for basal defense (Navarro *et al.,* 2006). Again, the auxin deficiencies in the plant induced by several auxin such as IAA inactivating genes enhance broad spectrum resistance to both bacterial and fungal disease (Fu *et al.,* 2011). In a different way, the resistance gene of a plant may recognize the specific pathogen effectors leading to host specific defense. Auxin plays a vital role in inducing host specific defense such as hypersensitive response by providing a signal for gene activation (Blank *et al.,* 2009; Lyons *et al.,* 2015). Whatever is the defense system, the mechanism of infection or lifestyle such as biotrophic or necrotrophic habit of the pathogens is equally important as they differentially modulate the auxin signaling or crosstalk with associated defense hormone (Kidd *et al.,* 2011; Qi *et al.,* 2012). In conclusion, the auxin signaling activities, defense gene activation and crosstalk of auxin with other defense hormone signaling pathways play an important role to provide host resistance against pathogens.

This review is carried out to examine the role of the most important hormone, auxin in plant defense against bacteria and fungi as examples of biotic stresses. The current state of knowledge on the role of auxin or auxin conjugates to host defense will be highlighted in the first section of the paper. The second section will discuss the auxin signaling and its crosstalk with special reference to biotrophic and necrotroph. The future prospects and take-home messages for plant breeders, especially in the field of role of auxin in plant defense and its utilization in crop breeding programs is highlighted in this review paper. Research gaps and some suggestions in the above mentioned areas are provided at the end.

METHODOLOGY

The role of auxin or auxin conjugates and it's signaling for plant defense is imperative, however, the information available is scattered and not up to date. For the literature review and search of different research papers and review papers about the role of auxin in plant defense mechanisms, online database Scopus and Google Scholar was used using keywords, 'auxin', 'host defense' and 'biotic stresses'. This review paper is formulated with integration of searched literature and insights of authors in the fore-mentioned topics.

1. AUXIN OR AUXIN CONJUGATES AS HOST DEFENSE AGAINST PATHOGENS

Depending on the pathogen and the type of the contact, host auxin signaling plays a function in either promoting disease or defense during pathogen infection. For instance, auxin signaling activation increases resistance to several plant viruses and necrotrophic diseases. Auxin signaling, on the other hand, frequently tends to encourage disease in plants exposed to biotrophic or hemibiotrophic pathogens (Kunkel & Johnson, 2021). Auxin or auxin conjugates act against a wide range of pathogens either by influencing growth and development or by regulating other cell activities. Indole-3-acetic acid (IAA) is the most common endogenous auxin in most of the plants. Plants also have other natural auxins such as indole-3-butyric acid (IBA), 4-chloroindole-3-acetic acid (4-C1-IAA), phenylacetic acid (PAA) and indole-3-propionic acid (IPA); however, their role in plant activities seems to be very minimal as compared to IAA. IAA either occurs in free form or conjugated to other compounds such as amino acids. The free form of IAA acts directly whereas the conjugated form of IAA is used for catabolism and subsequent action ranging from growth and development to defense mechanism. Evidence supports that the auxin and its conjugates are modulated upon pathogens attack or control, or crosstalk other associated hormone signaling and hence manipulate the system either for increased or reduced resistance (Fu *et al.,* 2011; Zhang *et al.,* 2019).

It has been stated that increased level of auxin is directly related to disease development (Kunkel & Johnson, 2021). Auxin may reduce the natural protective barrier of plant cells to an external biotic agent, here phytopathogens. Most often the bacterial infection induces the production of IAA in the plant cell. IAA subsequently expresses *expansins*, a group of cell wall relaxing proteins and makes the plant vulnerable to external biotic agents (Ding *et al.,* 2008). Similarly, in rice, IAA promotes the loosing of the cell wall, with the same process, and makes the plant susceptible to different fungal and bacterial pathogens (*Magnaporthe grisea, Xanthomonas oryzae* pv *oryzae* and *X. oryzae* pv *oryzicola*) (Fu *et al.,* 2011; Ye *et al.,* 2019). As a different mechanism, pathogens like *Botrytis cinerea* (fungus) and *Pseudomonas syringae* (bacteria) change the auxin metabolism in *Arabidopsis* and accumulate the conjugated form of the auxin, IAA-Asp. This conjugated inactive form of auxin is responsible for pathogen spread and host susceptibility (Gonzalez-Lamothe *et al.,* 2012). Moreover, strong auxin responsive transgenic plants are responsible for increased susceptibility. These transgenic plants can be determined phenotypically on observing increased lateral roots. For example, the transgenic *glip2* mutants show auxin-related gene (AuX/IAA) expression, which finally resulted in a susceptible phenotype (Dong *et al.,* 2009). After all, it is evident that the auxin level is vital for disease susceptibility (Kidd *et al.,* 2011; Zhang *et al.,* 2019) and hence repression of plant auxin signaling could be important to host plant resistance against pathogens.

Gretchen Hagen 3 (GH3) genes belong to a major auxin receptor gene family and *GH3* protein conjugation may alter or stimulate the hormonal signals thereby enhancing plant defense against a range of phytopathogens. The auxin-responsive gene (*GH3-8*) encodes IAA-amino synthetase, which prevents the accumulation of free auxin. The lower level of free auxin increases disease resistance against the rice pathogen, *Xanthomonas oryzae* pv *oryzae* when compared to wild type (Ding *et al.,* 2008). The overexpressing transformants, *OsGH3.1* produces reduced auxin characterized by dwarf phenotype, which has reduced cell growth and cell wall dissipating and ultimately enhances the resistance against the fungal pathogen in rice (Domingo *et al.,* 2009). Similarly, durable promoter *GH3-2*, encodes an indole-3-acetic acid (IAA)-amido synthetase then catalyzes the IAA-amino acid conjugate and inactivates IAA, generally pathogen induced IAA. This results in the suppressions of *expansin* genes, which provides broad-spectrum resistance to bacterial pathogens *Xanthomonas oryzae* pv *oryzae* and *Xanthomonas oryzae* pv *oryzicola* and fungal pathogen *Magnaporthe grisea* (Fu *et al.,* 2011). Hence, the enzyme produced by this auxin-responsive gene may enhance host resistance by reducing auxin conjugates. In general, the reduced level of auxin in a plant is associated with resistant phenotypes.

2. AUXIN SIGNALING AS HOST DEFENSE AGAINST PATHOGENS

The growth, development and biotic stress response of plants is mediated by auxin just after embryogenesis and up to the senescence. This process is accompanied by downstream signaling, which involved the regulation of auxin response genes (Quint & Gray, 2006), DNA-binding auxin response factors (ARFs), auxin/indole acetic acid (aux/IAA) transcriptional repressors (Guilfoyle & Hagen, 2007; Rybel *et al.,* 2010), topless co-repressors (TPL) (Szemenyei *et al.,* 2008) and transport inhibitor response 1 (TIR1) protein family (Terrile *et al.,* 2012). The ARF is connected with the Aux/IAA found in the promoters of the auxin response gene. The ARFs fix with the auxin response DNA elements (AuxRE) and mediate signaling either through stimulation or repression of transcription processes (Guilfoyle &

Hagen, 2007). The high level of auxin in the cell dissociates the ARF from the Aux/IAA repressors through an Aux/IAA destruction process via the ubiquitin-proteasome pathway. The produced ARF activators then activate auxin response genes (Guilfoyle & Hagen, 2012; Wang *et al.,* 2013). The ARFs and Aux/IAAs interaction may directly stimulate auxin-responsive transcriptions in plant (Wang *et al.,* 2013). Besides ARFs, the TIR1 and Aux/IAA proteins assemble as an auxin co-receptor complex. The auxin co-receptor complexes formed by a combination of different TIR1/AFBs and Aux/IAA provide sensing to signaling function in plants for example, in *Arabidopsis thaliana* (Calderon *et al.,* 2012).

Pathogens modify plant auxin physiology and functioning thus affect signaling pathways as a common virulence scheme. The phytopathogenic bacteria, *Pseudomonas syringae* pv. tomato strain PstDC300 induces overexpression of *AvrRpt2* in a normal plant. This situation leads to the higher IAA production and then subsequent disease development. Moreover, in the transgenic *Arabidopsis,* overexpression of *Pseudomonas syringae* type III effector *AvrRpt2* modifies auxin functioning by expressing free IAA at the higher level (Chen *et al.,* 2007). The same effector *AvrRpt2* increases auxin response by modulating the basic transcriptional repressors Aux/IAA and promotes pathogenicity (Cui *et al.,* 2013). In contrast, the wild-type *Arabidopsis* plant, upon attack, derives eubacterial 22–amino acid peptide (*flg22*) from PstDC300. This flagellin-derived peptide induces the plant miRNA, which then down-regulates the auxin signaling through targeting the auxin receptors TIR1, AFB2 and AFB3. The repressed auxin signaling restricts the bacterial pathogen PstDC300 growth and provides resistance to the plant (Navarro *et al.,* 2006). Interestingly, auxin signaling may induce either auxin-responsive pathway or auxin opposing pathway and exhibit immunity or susceptibility (Kazan & Manners, 2010).

Auxin and its crosstalking for host defense: biotroph verses necrotroph

Pathogens may be biotroph or necrotroph based on their mode of nutrition and lifestyle and based on those factors, different plant defense signal is activated. Biotrophs are organisms that live within the interstitial spaces of living plant cells and obtain their nutrition and energy from those cells (Fei & Liu, 2023). Auxin promotes plant growth and development, which is a carbon and nitrogen source for biotrophic pathogens in order to exhibit virulence behavior. In contrast, the necrotroph kills the host plant and utilizes dead tissue from it. In addition to having different food acquisition mechanisms, necrotrophs and biotrophs also have different virulence strategies and disease symptoms (Ghozlan *et al.,* 2020). Besides, the auxin and associated defense hormone signaling is also found to act differentially in different groups of pathogens (Zhang & Soonewald, 2017). Salicylic acid (SA) and jasmonic acid (JA), in addition to phytohormones including ethylene (ET), abscisic acid (ABA), gibberellins (GAs), auxin, and cytokinins (CKs), represent the two main defense-related phytohormones. These phytohormones are microscopic compounds made by plants that regulate a variety of physiological functions and play a key role in activating the plant immunological signaling network (Kim *et al.,* 2022). As defense-related hormonal crosstalk, the salicylic acid (SA) signaling pathway plays a key role in plant defense against biotroph (Wang et al., 2007), however, jasmonic acid (JA) signaling pathway is vital for plant defense against necrotroph (Qi *et al.,* 2012; Birkenbihl & Somssich, 2011). Different hormone responses also depend on a variety of other parameters, including the duration of an infection, the tissues of the plant, and the stage of plant development (Zhang & Soonewald, 2017).

The antagonistic crosstalk between SA and auxin during host defense against biotrophs was documented by many researchers. During infection, biotrophs either synthesize auxin or auxin-like molecules or alter the host plant auxin pathway and then increase host susceptibility (Navarro *et al.,* 2006; Kidd *et al.,* 2011; Wang *et al.,* 2007; Kunkel & Johnson, 2021). Besides, the biotrophs suppress the SA mediated defense responses and promote the pathogenesis mostly via auxin expression (Chen *et al.,* 2007; Spoel & Dong, 2008). In contrast, the defense hormone SA reduces the expression of auxin receptor gene TIR1/AFBs, which then ultimately reduce the auxin responses (Navarro *et al.,* 2006). Generally, SA impedes auxin signaling and therefore checks the biotrophic pathogens (Wang *et al.,* 2007). In addition, author (Zhang *et al.,* 2007a) further illustrated the antagonistic relationship between SA and auxin by qRT-PCR analysis, where SA inducing mutant *Arabidopsis* GH3.5 down-regulates the auxin, which then increases resistant of *Arabidopsis* plant against biotrophs. In conclusion, repressed auxin signaling and increased SA accumulating pathway lead to the host resistance against biotrophic pathogens.

Studies support the *MAP Kinase Kinase 7* (*AtMKK7*) gene effect on the regulation of SA and auxin. *AtMKK7* gene expresses lower level free IAA in the *Arabidopsis* plant as compared to wild type. At the same time, SA production is found maximum with pathogenesis-related (PR) gene expression and subsequent resistance effect against biotrophic pathogens (Dai *et al.,* 2006; Zhang *et al.,* 2007b; Zhang *et al.,* 2008). In addition, the *Arabidopsis*, SA overexpressing mutants such as cpr6 and snc1, measure lower free auxin levels than wild type (Wang *et al.,* 2007). Therefore, it is desirable to develop auxin down regulating or SA overexpressing mutant or both on the same plant to get resistance against biotroph.

The defense hormone JA-induced auxin signaling contributes to the plant resistance against necrotrophic pathogens. The mutants such as *axr1*, *axr2* and *axr6* with defective auxin-stirred SCF ubiquitination pathway express auxin signaling and exhibit increased susceptibility to the fungi *Plectosphaerella cucumerina* and bacteria *Botrytis cinerea* in *Arabidopsis,* where both bacteria and fungi are necrotrophic in nature (Llorente *et al.,* 2008). In a separate study, author (Qi *et al.,* 2012) reported that the necrotrophic pathogen, *Alternaria brassicicola,* helps to activate the auxin biosynthetic genes through the transcription process. This process elevates the auxin level of the host plant. At the same time, the pathogens reduce the PIN family protein of auxin transporters. Therefore, auxin transport capacity is reduced. The JA and auxin are found to interact positively and induce resistance to necrotrophic pathogens where JA-induced auxin signaling probably contributes to plant resistance against necrotrophs (Qi *et al.,* 2012) with some exceptions (Rahman *et al.,* 2012). These findings provide some molecular proof of JA and auxin interaction for host defense against necrotrophs. It is stated that auxin biosynthesis, auxin transport and signaling is antagonistic to SA synthesis and signaling is contributing to biotrophic resistance whereas, auxin and JA signaling possesses common grounds and are contributing to necrotrophic resistance. Furthermore, auxin responses are also equally important for causing development of disease (Kazan & Manners, 2010).

3. FUTURE PROSPECTS: IS MICROBIAL MEDIATED AUXIN SIGNALING HELPS TO COMBAT WITH PATHOGENS?

The trend of research has been greatly shifted from the use of auxin in growth and development to altering biotic stress responses. Recent studies demonstrate the role of auxin on host and microbial biocontrol agent interaction and ultimately to host defense against the pathogen. It is evident that the microbial biocontrol agents produce auxins such as IAA, which then affects the normal auxin physiology of the plant (Contreras-Carnejo et al., 2009). No doubt, the auxin produced by microbial biocontrol agents facilitates the extensive root growth, which may trigger the defense response of the plant such as basal defense against the pathogens. Besides, the local or systemic defense is triggered in the plant when microbial biocontrol agents such as *Pseudomonas fluorescens* strengthen host plants against pathogen attack, where auxin regulation of its role in up-regulation of the defense responsive gene may be vital. For example, during an interaction between bacterium biocontrol, *P. fluorescens* and fusarium head blight (FHB) fungus, bacterium enhances an IAA and ABA production in the host tissue, which then expresses bacterium-primed genes. This process allows the *P. fluorescens* to facilitate control of blight in barley (Petti et al., 2012). These pieces of evidence illustrate the further scope of the role of IAA in bio-control of phytopathogens and priming of host defenses.

Trichoderma and *Bacillus* are considered as few of the important biocontrol agents in recent days. The gene cloned from bioagent was studied in the past along with auxin regulation in the host plant. For example, the recombinant hydrophobin *rHFB2-6*, originally cloned from *Trichoderma asperellum* ACCC30536 induces a gene associated with auxin signal transduction in the Poplar plant (*Populus beijingensis*)). This auxin signal transduction induces the JA and SA signal transduction pathway and ultimately enhances ISR and SAR, giving broad-spectrum resistance to the pathogen (Huang et al., 2015). Moreover, the *B. subtilis* strains *BBG111* produce a different pattern of cyclic lipopeptides (CLP). These cyclic lipopeptides induce auxin signaling along with other defense hormone signaling and trigger an induced defense system against *Rhizoctonia solani* in Rice (Chandler et al., 2015). Hence, the bio-agent or the gene cloned from bio-control agent could be few of the important targets that can regulate auxin signaling and ultimately provide host resistance against pathogens.

CONCLUSION

During pathogen attack on plant and plant's subsequent counterattack, auxin or auxin signaling acts against phytopathogens by the up-regulation or down-regulation process of its signaling network. Generally, the down-regulation of auxin or associated signaling combat with pathogens and inhibits pathogen growth in the plant. The auxin-responsive gene, in general, is responsible for down-regulating the auxin. Again, the role of auxin against pathogens having different lifestyles is equally important as the auxin signaling varies depending upon the pathogen lifestyle such as biotroph pathogen and necrotrophic pathogen. In general, the SA-mediated defense response acts against biotrophs with the antagonistic relationship while GA-mediated defense response acts against necrotrophs with a linear relationship, with some limitations. Both auxin-responsive genes such as GH3 and auxin-associated defense hormone pathways such as GA and SA pathways can be manipulated in order to produce desirable auxin signaling pathways through genetic engineering and this may give an opportunity to the plant breeder to develop the resistant variety without reducing the crop yield. We hope that this background knowledge generates new and wider options for the breeder than in addition to the conventional practice of introducing resistance gene in the plant.

This review paper is mostly focused on the role of auxin in plant defense mechanisms against one or few pathogens, however in nature; a wide array of pathogens exists at the same time. The bio-agents are also simultaneously present in the field. This fact shows the necessity that future research should consider multiple pathogens in nature, their interaction with other beneficial microbes and associated auxin manipulation. This inter-connections study can be strengthened by further chemical biology and genome-wide study. Moreover, the ecological perspective of study was neglected in this theme, which should be conducted in addition to present biological and molecular study of auxin involved plant pathogen interaction and defense.

ACKNOWLEDGMENTS

We would like to acknowledge Dr. Goverse Aska from Laboratory of Nematology, Plant Sciences Group, Wageningen University and Research for providing valuable feedback to prepare this manuscript.

REFERENCES

Agrawal, S. (2018). Arabidopsis thaliana as a model organism to study plant-pathogen interactions. In Molecular Aspects of Plant-Pathogen Interaction (pp. 1-20). Springer, Singapore.

Atkinson, N.J. and Urwin, P.E. (2012). The interaction of plant biotic and abiotic stresses: from genes to the field. *Journal of Experimental Botany*, 63, 3523–3543.

Birkenbihl, R.P., & Somssich, I.E. (2011). Transcriptional plant responses critical for resistance towards necrotrophic pathogens. *Frontiers in Plant Science*, 2, 76. Doi: 10.3389/fpls.20 11.00076

Blank, L., Wolf, T., Eimert, K. and Schröder, M. (2009). Differential gene expression during hypersensitive response in Phylloxera-resistant rootstock 'Börner' using custom oligonucleotide arrays. *Journal of Plant Interactions*, 4(4), 261-269. Doi: 10.1080/17429 140903254697

Bürger, M., & Chory, J. (2019). Stressed out about hormones: how plants orchestrate immunity. *Cell host & microbe*, 26(2), 163-172.

Calderón Villalobos, L. I. A., Lee, S., De Oliveira, C., Ivetac, A., Brandt, W., Armitage, L., ... & Estelle, M. (2012). A combinatorial TIR1/AFB–Aux/IAA co-receptor system for differential sensing of auxin. *Nature chemical biology*, 8(5), 477-485.

Chandler, S., Van Hese, N., Coutte, F., Jacques, P., Höfte M. and De Vleesschauwer, D. (2015). Role of cyclic lipopeptides produced by *Bacillus subtilis* in mounting induced immunity in rice (Oryza sativa L.). *Physiological and Molecular Plant Pathology*, 91: 20-30.

Chen, Z., Agnew, J. L., Cohen, J. D., He, P., Shan, L., Sheen, J., & Kunkel, B. N. (2007). *Pseudomonas syringae* type III effector AvrRpt2 alters Arabidopsis thaliana auxin physiology. *Proceedings of the National Academy of Sciences*, 104(50), 20131-20136.

Contreras-Cornejo, H. A., Macías-Rodríguez, L., Cortés-Penagos, C., & López-Bucio, J. (2009). *Trichoderma virens*, a plant beneficial fungus, enhances biomass production and promotes lateral root growth through an auxin-dependent mechanism in Arabidopsis. *Plant physiology*, 149(3), 1579-1592.

Cui, F., Wu, S., Sun, W., Coaker, G., Kunkel, B., He, P. and Shan, L. (2013). The *Pseudomonas syringae* Type III Effector AvrRpt2 Promotes Pathogen Virulence via Stimulating *Arabidopsis* Auxin/Indole Acetic Acid Protein Turnover. *Plant Physiology*, 162(2), 1018-1029. Doi: http:/ / dx. doi. org/ 10. 1104/ pp. 113. 219659

Dai, Y., Wang, H., Li, B., Huang, J., Liu, X., Zhou, Y., ... & Li, J. (2006). Increased expression of MAP KINASE KINASE7 causes deficiency in polar auxin transport and leads to plant architectural abnormality in Arabidopsis. *The Plant Cell*, 18(2), 308-320.

Dietrich, A., Wolf, K Eimert, T. and Schroder, M.B. (2010). Activation of gene expression during hypersensitive response (HR) induced by auxin in the grapevine rootstock cultivar 'Börner'. *Vitis,* 49(1), 15–21.

Ding, X., Cao, Y., Huang, L., Zhao, J., Xu, C., Li, X. and Wang, S. (2008). Activation of the indole-3-acetic acid-amido synthetase GH3–8 suppresses expansin expression and promotes salicylate and jasmonate independent basal immunity in rice. *Plant Cell*, 20: 228–240.

Domingo, C., Andrés, F., Tharreau, D., Iglesias, D.J. and Talón, M. (2009). Constitutive expression of *OsGH3.1* reduces auxin content and enhances defense response and resistance to a fungal pathogen in Rice. *The American Phytopathological Society*, 22(2), 201–210. Doi: 10.1094/MPMI -22-2-0201

Dong, S.L., Kim, B.K., Kwon, S.J., Jin, H.C. and Park, O.K. (2009). *Arabidopsis* GDSL lipase 2 plays a role in pathogen defense via negative regulation of auxin signaling. *Biochemical and Biophysical Research Communications*, 379, 1038–1042.

Elnahal, A. S., El-Saadony, M. T., Saad, A. M., Desoky, E. S. M., El-Tahan, A. M., Rady, M. M., ... & El-Tarabily, K. A. (2022). The use of microbial inoculants for biological control, plant growth promotion, and sustainable agriculture: A review. *European Journal of Plant Pathology*, 162(4), 759-792.

Fei, W., & Liu, Y. (2023). Biotrophic Fungal Pathogens: a Critical Overview. *Applied Biochemistry and Biotechnology*, 195(1), 1-16.

Ficke, A., Cowger, C., Bergstrom, G., & Brodal, G. (2018). Understanding yield loss and pathogen biology to improve disease management: *Septoria nodorum* blotch-a case study in wheat. *Plant disease*, 102(4), 696-707.

Fu, J., Liu, H., Li, Y., Yu, H., Li, X., Xiao, J. and Wang, S. (2011). Manipulating broad-spectrum disease resistance by suppressing pathogen-induced auxin accumulation in Rice. *Plant Physiology*, 155, 589–602.

Ghozlan, M. H., Eman, E. A., Tokgöz, S., Lakshman, D. K., & Mitra, A. (2020). Plant defense against necrotrophic pathogens. *American Journal of Plant Sciences*, 11(12), 2122-2138.

Gonzalez-Lamothe, R., Oirdi, M.E.I., Brisson, N. and Bouarab, K. (2012). The conjugated auxin Indole-3-Acetic Acid–Aspartic Acid promotes plant disease development. *The Plant Cell*, 24, 762–777.

Guilfoyle, T.J. and Hagen, G. (2007). Auxin response factors. *Current Opinion in Plant Biology*, 10, 453-60. Doi: 10.1016/j.pbi.2007.08.014

Guilfoyle, T.J. and Hagen, G. (2012). Getting a grasp on domain III/IV responsible for Auxin Response Factor-IAA protein interactions. *Plant Science*, 190, 82-88. Doi: 10.1016/j. plantsci. 2012.04.003

Huang, Y., Mijiti, G., Wang, Z., Yu, W., Fana, H., Zhang, R. and Liu, Z. (2015). Functional analysis of the class II hydrophobin gene HFB2-6 from thebiocontrol agent Trichoderma asperellum ACCC30536. *Microbiological Research*, 171, 8–20.

Kazan, K., & Manners, J. M. (2009). Linking development to defense: auxin in plant–pathogen interactions. *Trends in plant science*, 14(7), 373-382.

Kidd, B. N., Kadoo, N. Y., Dombrecht, B., Tekeoglu, M., Gardiner, D. M., Thatcher, L. F., ... & Kazan, K. (2011). Auxin signaling and transport promote susceptibility to the root-infecting fungal pathogen Fusarium oxysporum in Arabidopsis. *Molecular plant-microbe interactions*, 24(6), 733-748. Doi: 10.1094/MPMI -08-10-0194

Kim, C. Y., Song, H., & Lee, Y. H. (2022). Ambivalent response in pathogen defense: A double-edged sword?. *Plant Communications*, 100415.

Kunkel, B. N., & Harper, C. P. (2018). The roles of auxin during interactions between bacterial plant pathogens and their hosts. *Journal of Experimental Botany*, 69(2), 245-254.

Kunkel, B. N., & Johnson, J. M. (2021). Auxin plays multiple roles during plant–pathogen interactions. *Cold Spring Harbor Perspectives in Biology*, *13*(9), a040022.

Liu, L., Guo, G., Wang, Z., Ji, H., Mu, F. and Li, X. (2010). Auxin in Plant Growth and Stress Responses. *In:* Phytohormones: A Window to Metabolism, Signaling and Biotechnological Applications (Editors: Lam-Son Phan Tran, Sikander Pal). ISBN: 978-1-4939-0490-7 (Print) 978-1-4939-0491-4 (Online). Springer New York (pp. 1-35). Doi: 10.1007/978-1-4939-0491-4.

Llorente, F., Muskett, P., Sanchez-Vallet, A., Lopez, G., Ramos, B., Sanchez-Rodriguez, C., ... & Molina, A. (2008). Repression of the auxin response pathway increases Arabidopsis susceptibility to necrotrophic fungi. *Molecular plant*, 1(3), 496-509.

Lv, Y., Pan, J., Wang, H., Reiter, R. J., Li, X., Mou, Z., ... & Yu, D. (2021). Melatonin inhibits seed germination by crosstalk with abscisic acid, gibberellin, and auxin in Arabidopsis. *Journal of Pineal Research*, 70(4), e12736.

Lyons, R., Stiller, J., Powell, J., Rusu, A., Manners, J.M. and Kazan, K. (2015). *Fusarium oxysporum* triggers tissue-specific transcriptional reprogramming in *Arabidopsis thaliana*. *PLoS ONE,* 10 (4), e0121902. Doi:10.1371/journal.pone.0121902

Mano, Y. and Nemoto, K. (2012). The pathway of auxin biosynthesis in plants. *Journal of Experimental Botany*, 63 (8), 2853-2872. Doi: 10.1093/jxb/ers091

Mashiguchi, K., Tanaka, K., Sakai, T., Sugawara, S., Kawaide, H., Natsume, M., ... & Kasahara, H. (2011). The main auxin biosynthesis pathway in Arabidopsis. *Proceedings of the National Academy of Sciences*, *108*(45), 18512-18517.

Navarro, L., Dunoyer, P., Jay, F., Arnold, B., Dharmasiri, N., Estelle, M., ... & Jones, J. D. (2006). A plant miRNA contributes to antibacterial resistance by repressing auxin signaling. *Science*, 312(5772), 436-439.

Petti, C., Reiber, K., Ali, S.S., Berney, M., and Doohan, F.M. (2012). Auxin as a player in the biocontrol of *Fusarium* head blight disease of barley and its potential as a disease control agent. *BMC Plant Biology*, 12: 224.

Qi, L., Yan, J., Li, Y., Jiang, H., Sun, J., Chen, Q., ... & Li, C. (2012). Arabidopsis thaliana plants differentially modulate auxin biosynthesis and transport during defense responses to the necrotrophic pathogen Alternaria brassicicola. *New Phytologist*, 195(4), 872-882.

Quint, M., and Gray, W.M. (2006). Auxin signaling. *Current Opinion in Plant Biology*, 9(5), 448–453. Doi: 10.1016/j.pbi.2006.07.006

Rahman, T.A.E., Oirdi, M.E., Gonzalez-Lamothe, R. and Bouarab, K. (2012). Necrotrophic pathogens use the Salicylic Acid signaling pathway to promote disease development in Tomato. *MPMI*, 25(12), 1584-1593. Doi: 10.1094/MPMI-07-12-0187-R

Raman, A., & Suryanarayanan, T. S. (2017). Fungus–plant interaction influences plant-feeding insects. *Fungal ecology*, 29, 123-132.

De Rybel, B., Vassileva, V., Parizot, B., Demeulenaere, M., Grunewald, W., Audenaert, D., ... & Beeckman, T. (2010). A novel aux/IAA28 signaling cascade activates GATA23-dependent specification of lateral root founder cell identity. *Current Biology*, 20(19), 1697-1706. Doi: 10.1016/j.cub.2010.09.007

Savary, S., Ficke, A., Aubertot, J.N. and Hollier, C. (2012). Crop losses due to diseases and their implications for global food production losses and food security. *In:* Food Security the Science, Sociology and Economics of Food production and Access to Food, Springer ISSN 1876-4517. Doi: 10.1007/s12571-012-0200-5

Spoel, S.H. and Dong, X.N. (2008). Making sense of hormone crosstalk during plant immune responses. *Cell Host Microbe*, 3(6), 348–351.

Szemenyei H., Hannon, M. and Long, J.A. (2008). TOPLESS mediates auxin-dependent transcriptional repression during *Arabidopsis* embryogenesis, *Science*, 319, 1384–1386.

Terrile, M.C., París, R., Calderón-Villalobos, L.I., Iglesias, M.J., Lamattina, L., Estelle, M. and Casalongué, C.A. (2012). Nitric oxide influences auxin signaling through S-nitrosylation of the Arabidopsis TRANSPORT INHIBITOR RESPONSE 1 auxin receptor. *The Plant Journal,* 70(3), 492-500. Doi: 10.1111/j.1365-313X.2011.04885.x

Wang, D., Pajerowska-Mukhtar, K., Culler, A.H. and Dong, X. (2007). Salicylic acid inhibits pathogen growth in plants through repression of the auxin signaling pathway. *Current Biology*, 17, 1784–1790. Doi: 10.1016/j.cub.2007.09.025

Wang, S., Hagen, G., and Guilfoyle, T.J. (2013). ARF-Aux/IAA interactions through domain III/IV are not strictly required for auxin-responsive gene expression. *Plant Signaling and Behavior*, 8(6), e24526.

Ye, J., Zhong, T., Zhang, D., Ma, C., Wang, L., Yao, L., ... & Xu, M. (2019). The auxin-regulated protein ZmAuxRP1 coordinates the balance between root growth and stalk rot disease resistance in maize. *Molecular plant*, 12(3), 360-373.

Zhang, H., & Sonnewald, U. (2017). Differences and commonalities of plant responses to single and combined stresses. *The Plant Journal*, 90(5), 839-855.

Zhang, H., Tan, X., Li, L., He, Y., Hong, G., Li, J., ... & Sun, Z. (2019). Suppression of auxin signaling promotes rice susceptibility to Rice black streaked dwarf virus infection. *Molecular plant pathology*, 20(8), 1093-1104.

Zhang, J., Peng, Y. and Guo, Z. (2007a). Constitutive expression of pathogen-inducible *OsWRKY31* enhances disease resistance and affects root growth and auxin response in transgenic rice plants. *Cell Research*, 18: 508-521.

Zhang, X., Dai, Y., Xiong, Y., DeFraia, C., Li, J., Dong, X. and Mou, Z. (2007b). Overexpression of *Arabidopsis* MAP kinase kinase 7 leads to activation of plant basal and systemic acquired resistance. *The Plant Journal*, 52, 1066–107.

Zhang, X., Xiong, Y., DeFraia, C., Schmelz, E. and Mou, Z. (2008). The *Arabidopsis* MAP kinase kinase 7 A crosstalk point between auxin signaling and defense responses? *Plant Signaling & Behavior*, 3(4), 272-274.

Zhang, Y., & Friml, J. (2020). Auxin guides roots to avoid obstacles during gravitropic growth. *The New Phytologist*, 225(3), 1049.