CHAPTER

18

Use of plant-defense hormones against pathogen diseases

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List of abbreviations

4-CL 4-coumarate CoA ligase
ABA Abscisic acid
ACC 1-aminocyclopropane-1-carboxylic acid
APX ascorbic acid peroxidise
BPH brown plant hopper
BR brassinosteroids
C4H cinnamate-4-hydroxylase
CAT catalases
CHI chitinase
CHS chalcone synthase
CK cytokinin
Cmm *Clavibacter michiganensis* subsp. *michiganensis*
D-E-L-L-A aspartic acid–glutamic acid–leucine–leucine–alanine
EBR 24-epibrassinolide
ET ethylene
FHB fusarium head blight

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GA gibberellic acid
GID1 gibberellin insensitive dwarf1
GLU β-1,3-glucanase
HAMPs herbivore-associated molecular patterns
HR hypersensitive response
HRGP hydroxyproline-rich glycoproteins
IAA indole acetic acid
ICS isochorismate synthase
IPT isopentenyl transferase
ISR induced systemic resistance
JA jasmonic acid
JA-Ile jasmonyl-isoleucine
MeJA methyl jasmonate
OsACS oryza sativa ACC synthase
OsGLP oryza sativa Germin like protein
PAL phenylalanine ammonia-lyase
PAMP pathogen-associated molecular pattern
POD peroxidase
PPO polyphenol oxidase
PRRs pattern recognition receptors
PsT *Pseudomonas syringae* pv. *tomato*
PYL PYR1-LIKE
PYR1 pyrabactin resistance 1
RCAR regulatory components of ABA receptors
SA salicylic acid
SAM S-adenosylmethionine
SAR systemic acquired resistance
SCF Skp, Cullin, F-box containing complex
SLR1 slender rice 1
SNF1 sucrose nonfermenting 1-related protein kinases 1
SOD superoxide dismutase

1. Introduction

A wide variety of bacterial and fungal diseases infect plants frequently, which has driven plants to develop a variety of biotic stress responses. The disease is more of an occurrence than a rule since plants have well-developed defense mechanisms against numerous pathogens. Plants usually employ complex multiple layers of defense mechanisms to protect themselves from several dreadful diseases. Plants naturally include physical and chemical barriers that restrict microbes from invading and colonizing them. These barriers include waxy cuticles, glucosinolate aggregates, and lignin (Atkinson and Urwin, 2012; Burger and Chory, 2019). Plants have also developed a number of inducible defenses that are activated when a pathogen is encountered. Numerous molecular, biochemical, and morphological alterations, such as an oxidative burst, the expression of defense-related genes, the synthesis of antimicrobial substances, and/or programmed cell death are among the inducible defenses

(van Loon et al., 2006). When pathogenic bacteria invade plant tissues, pattern recognition receptors (PRRs) in plant cells recognize the pathogen-associated molecular pattern (PAMP), initiating the first line of defense known as PAMP-triggered immunity. Complex hormonal signaling networks govern how plants respond to different biotic stresses (Arnaud and Hwang, 2015; Bezemer and van Dam, 2005; Fujita et al., 2006; Yang et al., 2022). Phytohormones are naturally occurring molecules with low molecular weight. Salicylic acid (SA), jasmonic acid (JA), ethylene (ET), abscisic acid (ABA), auxin, cytokinins (CKs), gibberellins (GA), and brassinosteroids (BR) are only a few of the plant hormones that are being explored. These hormones regulate a variety of developmental and signaling processes (Gray, 2004). The plant hormones JA and SA are the primary regulating constituents against pathogenic microbes and insects, even though most of them have been linked to defense system pathways (Bari and Jones, 2009; Tamoki et al., 2013). According to experimental findings, treating plant leaves with SA increases systemic acquired resistance (SAR) against infections. SA signaling in plants favorably induces defense against biotrophic pathogens, which feed and reproduce on living host organisms (Mandal et al., 2009; Reymond and Farmer, 1998; Akbudak et al., 2022). Although necrotrophs and insects that generally feed on living host cells through specialized feeding mechanisms are resistant to the JA/ET hormonal controls, this resistance is not absolute (Glazebrook, 2005; Yang et al., 2015). Even while other phytohormones including CKs, ET, ABA, and auxins occasionally interact with the SA or JA-induced hormonal signaling systems, they each have distinct effects on the plant's defense mechanism when used alone (Ma and Ma, 2016; Atamian and Harmer, 2016; Yang et al., 2022). By synthesizing phytochemicals or functional analogs, microbes have the ability to alter plants' immunological responses, which has caused hormonal imbalance and the onset of unwarranted defensive reactions (Robert-Seilaniantzetal, 2007). The major focus of this chapter analysis is the recent progress in the discovery of several hormonal components linked to plants' resistance and management to different insects and diseases (Table 18.1). A massive, complex, and unnamed network of interrelated systems called the plant hormone network operates. For instance, additional hormones such as ET, ABA, auxin, GAs, and CKs pathways are thought to modulate the SA-JA signaling cascade (Pieterse et al., 2012; Poveda, 2020; Kim et al., 2022). This chapter encompasses the latest developments in understanding the function and regulation of plant hormones, molecular mechanisms, and their synergistic and antagonistic interactions against various disease-causing pathogens.

2. Salicylic acid

Salicylic acid (2-hydroxy benzoic acid) (Fig. 18.1) is one of the most important phenolic compounds produced by plants. However, basal level production of this compound may vary among the different plant species. Joahn Buchner, a German chemist, in 1828 isolated this compound from the inner bark of the *Salix alba* and named it Salicin, which has been used as a pain reliever for decades. Several studies have also revealed their role in the phenomenon of thermogenesis in the reproductive organs of both gymnosperms and angiosperms (Raskin, 1992; Vlot et al., 2009). SA production in plants occurred via two significant pathways, viz. the isochorismate synthase (ICS) and the phenylalanine

TABLE 18.1 Role of plant hormones in plan protection against the various disease and pathogen.

Plants/ Fruits/ Vegetables	Use of hormone in combination with	Disease	Pathogen	Mechanism	Reference
Pea	SA	Pea powdery mildew	*Erysiphe pisi*	Induction of systemic resistance	Frfy and Carver (1998)
Tomato	SA	Tomato stem canker	*Alternaria alternata* f.sp. *lycopersici*	Induction of systemic resistance	Esmailzadeh et al. (2008)
Faba bean	SA + Benzithiadiazole	Rust	*Uromyces viciae-fabae*	Induction of systemic resistance	Sillero et al. (2012)
Jujube	SA	Postharvest disease of *Zizyphus jujuba*	*Monillinia fructicola Alternaria alternata*	Enhancement in GLU and POD actvity	Cao et al. (2013)
Orange	SA + *Penicillium membraefaciens*	Blue and green mold decay in citrus (postharvest)	*Penicillium digitatum*	Enhancement in PAL, POD, PPO, GLU activity	Zhou et al. (2014)
Mandarin	SA	Rot of citrus (postharvest)	Unknown bacterial and fungal pathogens	Aggregation of metabolites involved in defense and H_2O_2	Zhu et al. (2016)
Tomato	SA	Tomato leaf curl disease	*Begomovirus Tomato leaf curl Philippines virus* (ToLCPV)	Induction of SAR	Ong and Cruz (2016)
Grape fruit	SA + chitosan	Rot of grapefruit (postharvest)	*Penicillium digitatum*	Not stated	Shi et al. (2019)
Orange	SA + *Rhodotorula mucilaginosa*	Fungal infection of orange	*Penicillium italicum*	Enhancement in PAL, POD, PPO, GLU, CAT and APX activity	Ahima et al. (2019)
Guava	SA	Black spot disease (post harvest)	*Penicillium capitalensis*	Immediate fungitoxic activity and elicit a defensive response	Arafat (2019)
Potato	SA + *B. subtilis*	Stored potato tubers	*Penicillium infestans Fusarium oxysporum*	Rise in antioxidant activity	Lastochkina et al. (2020)
Pakchoi	SA	Club root disease of *Brassica campestris*	*Plasmodiphora brassicae*	Rise in antioxidant enzymes *viz*., SOD, APX,CAT, GR	Xi et al. (2021)
Sweet pepper	SA	Black mold disease	*Alternaria alternata*	Rise in gene expression were for the PR-1, beta-1, 3-glucanase, and peroxidase	Abdel-Rahman et al. (2021)

TABLE 18.1 Role of plant hormones in plan protection against the various disease and pathogen.—cont'd

Plants/ Fruits/ Vegetables	Use of hormone in combination with	Disease	Pathogen	Mechanism	Reference
Cassava plant	SA	Fusarium root rot	*Fusarium solani*	Not stated	Saengchan et al. (2022)
Bean plant	SA + BA (benzoic acid)	Common blight of beans	*Xanthomonas axonopodis* pv. *phaseoli* (*Xap*)	Not stated	Abo-Elyousr et al. (2022)
Tomato	JA (MeJA)	Anthracnoserot	*Colletotrichum coccodes*	Volatiles were retained	Tzortzakis et al. (2007)
Pear	JA (MeJA)	Blue mold decay of pears	*Penicillium expansum*	Not stated	Zhang et al. (2009)
Peach	JA (MeJA)	Postharvest diseases	*Penicillium expansum* *B. cinerea*	Enhancement in PAL, POD, PPO, GLU, CHI activity	Jin et al. (2009)
Apple	JA (MeJA)	Postharvest diseases	*Penicillium expansum*	Not stated	Quaglia et al. (2011)
Cherry tomato	JA (MeJa)+Chitosan	Postharvest diseases	*Alternaria alternata*	Enhancement in enzyme activity viz., POD, PPO, PAL	Chen et al. (2014)
Chinese bayberry	JA (MeJA)	Postharvest diseases	*Penicillium citrinium*	Enhancement in PAL and CHI activity	Wang et al. (2014)
Strawberry	JA (MeJA)	Postharvest diseases	*B. cinerea*	Enhancement in GLU and CHI activity	Saavedra et al. (2017)
Tomato	JA + melatonin	Postharvest diseases	*B. cinerea*	Enhancement in defense related enzyme activity	Liu et al. (2019)
Apple	JA (MeJA) + *Meyerozyma guilliermondii*	Postharvest blue mold decay	*Penicillium expansum*	Enhancement in enzyme activity viz., POD, PPO, PAL, CAT and flavonoids	He et al. (2020)
Kiwi fruit	JA (MeJA)	Soft rot disease	*Botryosphaeriadothidea*	Enhancement in enzyme activity viz., POD, PPO, PAL, CAT and CHI	Pan et al. (2020)
Grape fruit	JA (MeJA)	Gray mold disease	*Botrytis cinerea*	Enhancement in enzyme activity viz., PPO, PAL, CAT and CHI	Suhui et al. (2022)

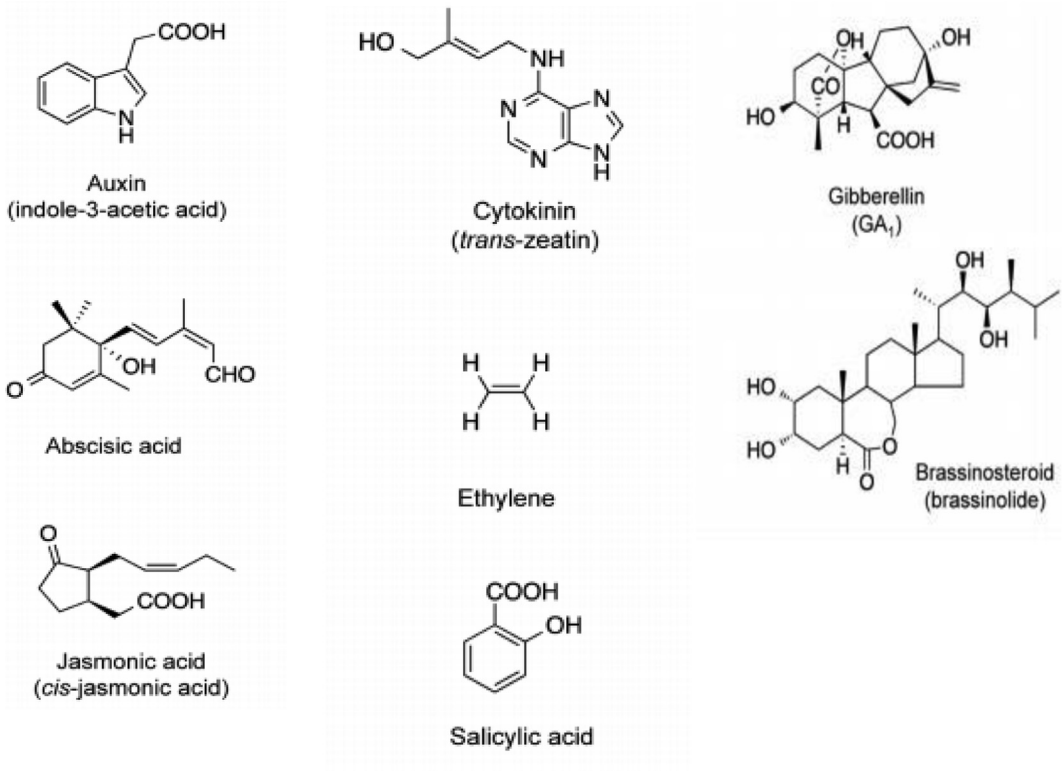

FIGURE 18.1 Structure of plant hormones. *Adapted from Asami and Nakagawa (2018).*

ammonia-lyase (PAL). Both biochemical routes begin with chorismate in plastids (Rekhter et al., 2019; Mishra and Baek, 2021). It is well recognized that SA plays a crucial role in modulating plant defense (Durant and Dong, 2004; Glazebrook, 2005), which includes both biotrophic as well as hemibiotrophic pathogens such as viruses. The hormone is also found responsible for activating pathogen-resistant (PR) genes as well as SAR pathways for generating long-term immunity in plants (Shah, 2003; Fu and Dong, 2013; Zhou and Zhang, 2020). One of the plants' early defense responses to pathogen invasion is the hypersensitive response (HR). It stands out for its quick induction of apoptosis in the host cell at the location of the initial infection, which also gets rid of the pathogen and prevents infection spread. The plant can thereby prevent the pathogen from spreading further by sacrificing a little quantity of tissue. Against biotrophic infections, this response is very effective (Glazebrook, 2005). Endogenous SA has been found responsible for mediating HR; as a result, there has been the prenecrotic aggregation of SA observed in the leaves of cucumbers. Mutations in SA pathways revealed changed patterns in HR. Some components of HR appear to be universal throughout pathosystems. To begin with, afflicted cells undergo apoplasm alkalinization due to ions shift (Xu and Heath, 1998). Emerging calcium channel studies have demonstrated that blocking calcium transport also inhibits cell death whenever they are invaded by bacteria. Calcium plays a critical role in these HR-related fluxes (Levene et al., 1996; Ali et al.,

2007). Reactive oxygen species production is known to occur before HR-related cell death when combined with ion fluxes because of their high reactivity which ultimately leads to the destruction of the cellular machinery (Mur et al., 2013). In response to exogenous SA, plant disease resistance is generally enhanced by activating PR proteins and associated defense enzymes like polyphenol oxidase, chitinase as well as phenylalanine aminolyase (Wang and Liu, 2012; Kaltdorf and Naseem, 2013). Yao and Tian (2005) studied the effects of pre and postharvest application of SA on inducing pathogen resistance in cherry fruit. They concluded that exogenous application of 2 mM SA to sweet cherry fruit dramatically reduced lesion sizes induced by the fungus *Monilinia fructicola* because of resistance induction and enhanced enzymatic activity of β-1,3-glucanase (GLU), PAL, and peroxidase (POD). Application of 2 mM SA was also effective against damage caused by *Penicillium expansum* on cherries and reduced pathogen-induced oxidative stress due to enhanced activity of catalases (CAT) and glutathione peroxidases (Xu and Tian, 2008). The same outcomes were also recorded by Tareen et al. (2012), who verified enhanced antioxidant enzymic activity such as CAT and superoxide dismutase (SOD) on peaches after the exogenous application on peaches. The concentration of SA ranging from 0.5 to 5 mM alleviated the harm caused by *Rhizopus stolonifer* on peaches (Panahirad et al., 2012). Adss et al. (2017) studied the postharvest resistance in tomato fruits using SA elicitors against rot disease caused by *Alternaria solani*. The treatment decreased the diameter of the fruit-rotted area after the application of 2 μM SA. A sudden decline in polygalacturonase activity was observed; however, significant increases in defense enzymes, viz. PAL, peroxidase, polyphenol oxidase (PPO), and POD were seen. Tannuri et al. (2021) also studied the exogenous application of SA for preventing coffee rust. They reported that 150 mg/L of SA after foliar application lowered the intensity and occurrence of coffee rust in plants with low crop load and activated the production of enzymes such as ascorbate peroxidase, superoxide dismutase along with catalase after initial application. However, very little impact was observed after consecutive applications. Akbudak et al. (2022) evaluated the treatment of SA on white mold disease caused by *Sclerotinia sclerotiorum* on two lettuce cultivars Melina and Fuzila (*Lactucasativa* L.) Decline in fungal infection was observed up to 19% and 14% for Melina and Fuzila after exogenous applications of SA and increased in the number of marketable and nonmarketable leaves of lettuce and also protected the plant from future fungal infections.

3. Jasmonic acid

The major endogenous chemical jasmonic acid (3-oxo-2-20-*cis*-pentenyl-cyclopentane-1-acetic acid) (Fig. 18.1) discovered in higher plants has significantly contributed to the development of numerous signaling systems that are in charge of regulating plant growth and stress reactions (Smith et al., 2009). JAs belong to a class of fatty acid derivatives that include its methyl ester (MeJA), as well as the isoleucine conjugate (JA-Ile) (JAs). This hormone regulates several physiological functions. The octadeconoid pathway produces oxylipins, or oxygenated fatty acids, which include JA and its derivatives. They were first recognized as a stress-related hormone (Balbi and Devoto, 2008). JAs play a role in the regulation of critical developmental processes. Opening of stomata, for instance, prevents the formation of Rubisco affecting both nitrogen and phosphate intake as well as the transport of organic

materials like glucose, maturation of pollen, development of flower as well as fruit, photosynthesis, senescence, and root growth (Bari and Jones, 2009). By stimulating the expression of some genes, JAs can effectively serve as a signaling molecule that regulates reactions to environmental stimuli. The first study to establish a connection between Jas' defense mechanisms was on the modulation of the expression of digesting proteinase inhibitors in the *Solanum lycopersicum* (Farmer and Ryan, 1990). Two decades after that first finding, it is now well established that JA is a crucial cellular signal involved in the activation of immunological responses to the majority of insects, herbivores, and necrotrophic microorganisms. The early signaling phases involved in recognizing an attack by herbivores or pathogens are the identification of herbivore and damage-associated molecular patterns (HAMPs and DAMPs, respectively) and the activation of JA biosynthesis (Broaderson et al., 2006; Ruan et al., 2019). There is indeed a plethora of studies on the function of jasmonates in the response to insect herbivory. To put it simply, when insects infiltrate plant tissues, membrane lipids release linolenic acid which then enters the octadecanoid route where it is transformed into an active JA, leading to further simulation of resistance pathways and, ultimately, the plant's defense genes. JA in conjunction with other plant hormones like ET acts synergistically in defense responses. Proteinase inhibitors, volatile chemicals associated with defense, nicotine, active phenolics, and phytoalexins are all secondary metabolites that have been linked to jasmonate induction. An increase in herbivores and their natural predators have also been seen as a result of the emission of induced plant volatiles that can deter pests (Browse and Howe, 2008; Verhage et al., 2011). The rhizobacteria-induced systemic resistance (ISR), which is similar to but different from the SA-mediated SAR, is a long-lasting, systemic defense response that is triggered in *Arabidopsis* when specific rhizobacteria are present. The utilization of mutants for SA, JA, and ethylene signaling proved that rhizobacteria-ISR only needs JA and ethylene response stimulation and is not dependent on SA induction. This rhizobacteria-ISR provides resistance to a range of microbe-borne illnesses and insects, demonstrating that ethylene and JA play a part in long-term defensive reactions (Pangesti et al., 2016). In response to exogenous application methyl jasmonate (MeJA), it has shown to minimize the appearance of chilling injury in peaches due to abrupt temperature changes in storage (from 5 to 20°C). As a result, diseases are less likely to infect the fruit because the damaged tissues provide an easy entry point for microbes. When it comes to peach defensive responses, vaporized MeJA (1 μmol/L) has been shown to reduce the occurrence of *Penicillium expansum*, *Botrytis cinerea*, and *R. stolonifer* and other fungus-causing postharvest infections (Meng et al., 2009). Due to the activation of a defensive response mediated by JA/ET, it was possible to significantly reduce *B. cinerea* damage in peaches by employing another JA derivative, such as n-propyl dihydrojasmonate, at 400 μM concentration (Suktawee et al., 2019). Fugate et al. (2012) studied the exogenous administration of JA (100 μM) and observed that it considerably lessens the damage that pathogens like *B. cinerea*, *Penicillium claviforme*, and *Phoma betae* cause to postharvest sugarbeat. Further administration of MeJA at a concentration of 500 μM on vegetables like broccoli and cauliflower enhanced the concentration of secondary metabolites including glucosinolates and gluconasturtiin (Ku et al., 2013a, b). Root vegetables were also given 10 μM MeJA exogenously, and this increased the expression of genes related to plant defense systems (de Oliveira et al., 2017). In rice plants infected with *Magnaporthe oryzae*, Wang et al. (2020) examined the endogenous levels of JA as well as the effects of exogenous JA on rice blast symptoms and the activation of genes involved in

defense. To determine the optimum JA treatment, a variety of spraying strategies were investigated. Before, during, and after the pathogen invasion, changes in the endogenous levels of JA were noticed. JA also restricted the fungal growth and appressorium formation. In rice leaves, treatment with JA (400 μmol/L) dramatically improved the viability of the cell as well as the natural production of JA. Furthermore, among all treatment modalities, rice plants inoculated with *Magnaporthe oryzae* and treated with JA, 72 h after inoculation demonstrated the greatest symptom alleviation and enhanced endogenous JA biosynthesis. Yang et al. (2022) investigated exogenous JA administration against *Phytophthora infestans* illness in (SD20) potato plants. They observed the activation of early pathogen defense response against a virulent strain of *P. infestans*. In response to exogenous JA, "SD20" plants quickly increased the expression of genes involved in immunological regulation, JA synthesis, signal transduction, and early defense responses. The majority 2927 of differentially expressed genes encoding transcription factors, kinases, bioactive compounds, enzymes associated with defense, and pathogen resistance proteins were identified.

4. Auxin

Plant hormones are essential for controlling plant responsiveness to a range of biological and environmental sources of stress as well as signaling networks involved in developmental processes. Recent research suggests that hormones like auxin are involved in signaling pathways for plant defense, although their function in plant defense is yet unknown. A wide range of plant developmental activities are regulated by the phytohormone auxin, obtained from the Greek word "auxein," which means to grow. The defense of plants has been linked to plant hormones like auxins (Yamada, 1993; Robert-Seilaniantz et al., 2007; Navarro et al., 2008). The key precursor for the formation of indole acetic acid (IAA) is tryptophan. The trp genes encode the five-step synthesis of tryptophan, which is started from chorismate. The shikimate route, which is a typical mechanism for the formation of aromatic amino acids and numerous bioactive metabolites, is used to create the branch point chemical chorismate. It begins with the synthesis of phosphoenolpyruvate and erythrose 4-phosphate (Dosselaere and Vanderleyden, 2001; Casanova-Sáez et al., 2021). A crucial aspect of bacterial phytopathogenesis is auxin physiology. Auxin signaling in *Arabidopsis* is down-regulated in response to the plant recognizing a biotrophic leaf pathogen, reducing the disease's susceptibility (Navarro et al., 2006). The pathogen is able to combat this by activating a Hrp system (Chen et al., 2007). The four-phased model of the plant defense system's effector-triggered susceptibility, pathogen-related molecular pattern-triggered immunity, and effector-triggered susceptibility, all of which are regulated by host auxin signaling, is demonstrated by the results in outstanding detail (Jones and Dangl, 2006). Additionally, both studies showed that the host's external auxin supply boosts sensitivity to bacterial infection and the onset of disease symptoms. As most bacterial diseases are known to produce IAA, it is conceivable that this characteristic is a part of the pathogen's strategy to bypass the plant's immune system. The same could be true for auxin-producing rhizobacteria that support plant development. It is yet to be shown if the IAA generated by these bacteria actually improves the interactions between the bacteria and the roots of plants. There is evidence that IAA controls the expression of

various plant-associated microorganisms (Argon et al., 2014; Kunkel and Harper 2018; Djami-Tchatchou et al., 2020). It has been established that auxin-responsive GH3 genes support plant defensive responses in *Arabidopsis* and Rice. Recent research has demonstrated that, during pathogen infection, SA and auxin signaling pathways are both regulated by GH3.5 in a bifunctional fashion. Similarly, it was established that overexpression of GH3-8 was responsible for the increased resistance to the rice pathogen *Xanthomonas oryzae* pv. *oryzae* (Xoo), which is responsible for inducing bacterial blight disease, and this resistance was unrelated to SA and JA signaling. SA- and JA-sensitive genes were expressed less frequently in GH3-8 overexpressing plants than in wild-type plants (Ding et al., 2008; Ludwig-Müller, 2011; Denancé et al., 2013). The recent suppression of auxin signaling, either by alterations in the auxin signaling components or interference with auxin transport, has reduced the resilience of *Arabidopsis* plants to the necrotrophic fungi *Plectosphaerella cucumerina* and *Botrytis cinerea*. Moreover, *Arabidopsis* auxin response genes are downregulated as a result of infection with aggressive necrotrophs like *P. cucumerina* (Llorente et al., 2008). As a result, auxin signaling may play a significant role in regulating how plants react to necrotrophic fungi. Auxin signaling elements are another mechanism that viral infections exploit to increase virulence and spread illness. For instance, in *Arabidopsis* and tomato, interactions between tobacco mosaic virus (TMV) replicase and Aux/IAA proteins influence the transcriptional activation of auxin-responsive genes and enhance the emergence of disease symptoms. Furthermore, it has been shown that the TMV replicase connects with a number of related *Arabidopsis* Aux/IAA proteins and interferes with their localization in the nuclear region (Padmanabhan et al., 2006; Müllender et al., 2022). This suggests that TMV may interfere with Aux/IAA activities to change the cellular milieu to promote viral reproduction and dissemination (Padmanabhan et al., 2008; Checker et al., 2018). Together, the new data point to auxin as a key player in the hormone signaling network controls the body's defenses against a variety of infections caused by biotrophs and necrotrophs. Auxin regulates the creation, degradation, and expression of genes connected to other hormone signaling pathways as well as the development and defense responses (Paponov et al., 2008; Gomes and Scortecci, 2021). Nevertheless, it is still unknown how auxin levels affect the balance of other hormones and hone defensive reactions specific to particular illnesses (Checker et al., 2018; Gomes and Scortecci, 2021).

5. Abscisic acid

ABA is a crucial phytohormone that controls several aspects of plant development, especially germination of seeds and fruit development, particularly in response to a variety of abiotic stresses like heat, salinity, and water stress (Finkelstein et al., 2002; Tuteja, 2007; Graeber et al., 2012; Lee and Luan, 2012). Contrary to abiotic stress, the pathosystem determines how well ABA functions under biotic situations. For instance, ABA increased resistance to one fungus (*Cochliobolus miyabeanus*) but increased sensitivity to another (*Magnaporthe oryzae*) in the same plant (De Vleesschauwer et al., 2010; Jiang et al., 2010). ABA performs a variety of roles in plant immunity in addition to its functions in abiotic stress and development (Ton et al., 2009). A plant's immune responses are either stimulated or inhibited by ABA depending on the type and lifestyle of the pathogen. For instance, ABA

inhibits *Pseudomonas syringae* bacterial pathogen resistance. Based on the type of pathogen, ABA either stimulates or inhibits a plant's defense mechanisms. For instance, in *Arabidopsis thaliana*, ABA enhances resistance to the necrotroph *Alternaria brassicicola* while suppressing resistance to *Pseudomonas syringae* (Mohr and Cahill, 2003; Garcia-Andrade et al., 2011). It is also known that ABA coordinates a number of plant defense mechanisms against viruses. Superoxidase dismutases and catalases were expressed as a result of ABA therapy, which also prevented the buildup of reactive oxygen species. It has also been demonstrated that the function of ABA in plant immunity is stage-dependent. By causing stomatal closure and callose deposition during the early stages of infection, ABA can favorably control plant defense. However, in later stages, when SA or JA signaling is antagonized, ABA has a detrimental impact on plant immunity (Ton et al., 2009; Bharath et al., 2021). The diverse functions of ABA in response to bacterial or fungal infections have been extensively investigated, but little is known about how ABA interacts with plant viruses, and the only studies that have been published have indicated that ABA increases resistance. Zeaxanthin epoxidase catalyzes the conversion of zeaxanthin to violaxanthin as the first crucial step in the production of ABA (Audran et al., 1998; Dong et al., 2015). Neoxanthin synthase, isomerase, and 9-*cis*-epoxycarotenoid dioxygenases progressively catalyze the conversion of violaxanthin to xanthoxin (Tan et al., 2003; Dong et al., 2015). Following that, a short-chain alcohol dehydrogenase converts xanthoxin to ABA (Gonzalez-Guzman et al., 2002) and an abscisic aldehyde oxidase (Yang et al., 2014; Cardoso et al., 2020). By means of hydroxylation and conjugation, ABA can be physiologically inactivated (Nambara and Marion-Poll, 2005). The most common form of ABA catabolism is thought to be ABA 8′ hydroxylation (ABA8ox), which is mediated by cytochrome P450 monooxygenase (Saika et al., 2007; Cardoso et al., 2020). ABA is sensed and conducted by three primary components such as PYR/PYL/RCAR, an ABA receptor (Ma et al., 2009; Park et al., 2009), type 2C protein phosphatase, a negative regulator (Leung et al., 1997) and SNF1-related protein kinase, a positive regulator (Fujii et al., 2011). Plants typically have low amounts of cellular ABA under normal circumstances, and 2C protein phosphatase binds to SNF1-related protein kinase and inactivates it by dephosphorylation (Raghavendra et al., 2010). The PYR/PYL/RCAR receptor proteins bind ABA when it has accumulated as a result of stress circumstances, blocking the 2C protein phosphatase-mediated dephosphorylation of the SNF1-related protein kinase. Downstream transcription factors are phosphorylated and activated by the active SNF1-related protein kinase, and they then start the transcription of ABA-responsive genes (Hauser et al., 2011). In the initial studies of ABA-virus interactions, TMV had an impact on ABA accumulation (Whenham et al., 1986). By promoting callose deposition, ABA also reduced viral mobility (Rezzonico et al., 1998). Moreover, numerous investigations showed a direct correlation between ABA concentrations and antiviral resistance. Exogenous ABA treatment in tobacco improved TMV and tobacco necrosis virus resistance (Iriti and Faoro, 2008). By working either synergistically or antagonistically with the JA system, ABA has been shown to enhance or decrease host plant innate immunity (Anderson et al., 2004; Garcia-Andrade et al., 2011; Avramova, 2017). In *Arabidopsis*, ABA and JA worked together to promote resistance to the necrotrophic fungal disease *Botrytis cinerea* (Garcia-Andrade et al., 2011). In the irregular interaction between Arabidopsis and Pythium, ABA synthesis was demonstrated to be necessary for the formation of JA (Adie et al., 2007). ABA strengthens the plant's defense against the fungus that causes brown spots in rice (De Vleesschauwer et al., 2010). Nevertheless, it reduced

the host's defenses against *Magnaporthe oryzae* (Jiang et al., 2010). The improved plant antiviral response caused by ABA has been linked to its effectiveness against viral infections (Alazem and Lin, 2017). RNA silencing and callose deposition at plasmodesmata (PD) are believed to be the primary ABA-dependent antiviral mechanisms (Alazem and Lin, 2017). Numerous studies have shown a connection between ABA levels and tobacco mosaic virus immunity (Chen et al., 2013; Whenham et al., 1986). By inducing callose accumulation at PD and limiting viral cell-to-cell migration, the use of ABA enhances resistance to TMV (Rezzonico et al., 1998). A variety of ABA-deficient mutants have been used to demonstrate ABA-mediated resistance against the bamboo mosaic virus (Alazem et al., 2013), Thus the induction of AGO families in *Arabidopsis* is mostly responsible for the ABA-mediated immunity to bamboo mosaic virus (Alazem et al., 2017). To what extent ABA could potentially mediate vulnerability to viral infection is unknown, though, given the paucity of available data. According to research by Deng et al. (2018), the expression of several genes related to ABA production and signaling may shift in response to *P. digitatum* invasion in citrus fruits. However, it is still unknown if ABA has a role in the citrus fruit's disease resistance. Similar investigations were also reported by Lafuente et al. (2019), *P. digitatum* capacity to infect both the wild-type Navelate orange fruit (*Citrus sinesis* (L.) Osbeck) and the mutant Pinalate fruit (which lacks ABA) was evaluated. The ABA-deficient mutant's increased susceptibility to infection supports the notion that ABA protects citrus fruit against *P. digitatum* infection. Exogenous ABA decreased the proportion of infected fruits and the maceration area of the mutant without changing in vitro microbial overgrowth. Additionally, it has been reported that proline, sphingolipids, and polyamines can also accumulate more readily when exposed to ABA (Bharath et al., 2021). Boba et al. (2022) also elucidated that ABA activates flax's defensive mechanisms and plays a key role in the plant's ability to successfully combat off infection.

6. Ethylene

The gaseous plant hormone ethylene is the simplest unsaturated hydrocarbon (Fig. 18.1) produced by plants at specific developmental stages as well as in response to both biotic and abiotic stressors. It participates in the ripening of fruit, senescence, and leaf abscission (Abeles et al., 1992). In general, ACC synthase (ACS) catalyzes the transformation of the amino acid methionine into S-adenosylmethionine (SAM), which will then be transformed into 1-aminocyclopropane-1-carboxylic acid (ACC), and eventually, ET is produced from ACC by the catalysis of another biocatalyst, the 2-oxoglutarate ACC oxidase (ACO) (Wang et al., 2002). Ethylene has demonstrated a variety of developmental impacts, including the inhibition of shoot growth in dicots and conversely promotion of growth in many hydrophytes. Long-term ethylene treatment of plants caused the stems to develop calluses (Wallace, 1928). It is still unclear what functions ethylene plays in morphogenesis. Available findings indicate that morphogenesis is frequently activated at a critical concentration. Many researchers have also noted the importance of ethylene in the development of bulbs on tulip shoots (Alderson and Taeb, 1990). According to the available data, ethylene inhibits embryogenesis. Additionally, it also accelerated the synthesis of secondary metabolites. ET is the key regulator of the plant immune signaling network, which controls pattern-triggered immunity against biotrophic infections (Kim et al., 2014). The hormone affects a variety of plant pathogens through

both endogenous and exogenous mechanisms. On plant–pathogen interactions, there is a sharp rise in endogenous ethylene synthesis, which enhances plant defensive responses by activating defense genes such as L- PAL, 4-coumarate CoA ligase (4-CL), chalcone synthase (CHS), and hydroxyproline-rich glycoproteins (HRGP). Exogenous treatments of ET and/or its precursors have shown direct connections between this volatile plant hormone and a plant's protective mechanisms against pathogens (Beckman, 2000). The microarray analysis revealed that the transcripts of some tomato genes involved in ET production and response were altered in response to infection with *Clavibacter michiganensis* subsp. *michiganensis* (*Cmm*). In comparison to wild-type plants, never-ripe (Nr) mutant tomato plants with a compromised ethylene perception character and transgenic plants with a lower capacity for ET production both significantly delayed the onset of wilt symptoms after *Cmm* infection. These findings suggest that the regulation of the tomato's susceptibility to disease progression is significantly influenced by host-derived ET (Balaji et al., 2008). Currently, it is widely acknowledged that ET works with JA to trigger defences against necrotrophic infections and that it opposes SA-dependent resistance to several biotrophic pathogens (Fig. 18.2) (Knoester et al., 1999; Yang et al., 2015). Conversely, there have been reports of collaborative linkages involving ET and SA pathways, and ET has been linked to the stimulation of defenses against certain biotrophs and hemibiotrophs, so this is probably an exaggerated view (van Loon et al., 2006). The intricacies of the regulation of multigene families associated with ET production, signal transduction, and crosstalk determine how much ethylene contributes to choosing the best protective reaction to every specific biotic stress (Adie et al., 2007). Numerous studies demonstrate that following pathogen administration, the signaling of jasmonate- and ethylene frequently collaborates to promote the stimulation of a number of genes involved in defense (Glazebrook, 2005). The increase of PAL mRNA in carrot root (*Daucus carota*) was found to be induced by 10 ppm ethylene, which allowed researchers to explore the hormone's role in plant defense (Ecker and Davis, 1987). Ethylene was also compared with the interferons in humans that send alerts to other distance cells. According to Broglie et al. (1986), ethylene treatment resulted in the enhancement of chitinase levels in bean seedlings. These results have drawn attention to the notion that ethylene may play a part in how the host reacts to pathogen infection. Additionally, locally generated ethylene, which is a gas, is ideally suited to activate defense-response genes in nearby and plant tissues before pathogen transit. Ethylene levels also increased on melon (*Cucumis melo*) after the invasion of *Colletotrichium lagenarium* (Toppan and Esquerre-Tugaye, 1984) followed by an increase in chitinase activity and hydroxyproline accumulation in the cell wall (Roby et al., 1986). *Arabidopsis* plants when administered with an inhibitor of ET biosynthesis noticeably draw more nematodes toward their roots, whereas mutants with excessive ET production harbored fewer nematodes than that of the control (Fudali et al., 2013). In addition to controlling local induced resistance, ET also controls SAR. It can get beyond vascular restrictions because it is a gaseous hormone this might be particularly significant in tall plants because vascular signals might only go a few centimeters and volatile signals can travel meters. ET sensitivity is necessary for TMV-infected leaves to develop SAR, which in turn causes SA to build up and lead to the development of SAR in unaffected leaves (Robert-Seilaniantz et al., 2011; Pietrse et al., 2012). Foreign pathogenic attackers may employ ET signaling to enter the host by either creating ET on their own or ethylene-inducing effectors that congregate to target ET signaling to reduce plant immunity (Wi et al., 2012). For instance, the pathogen

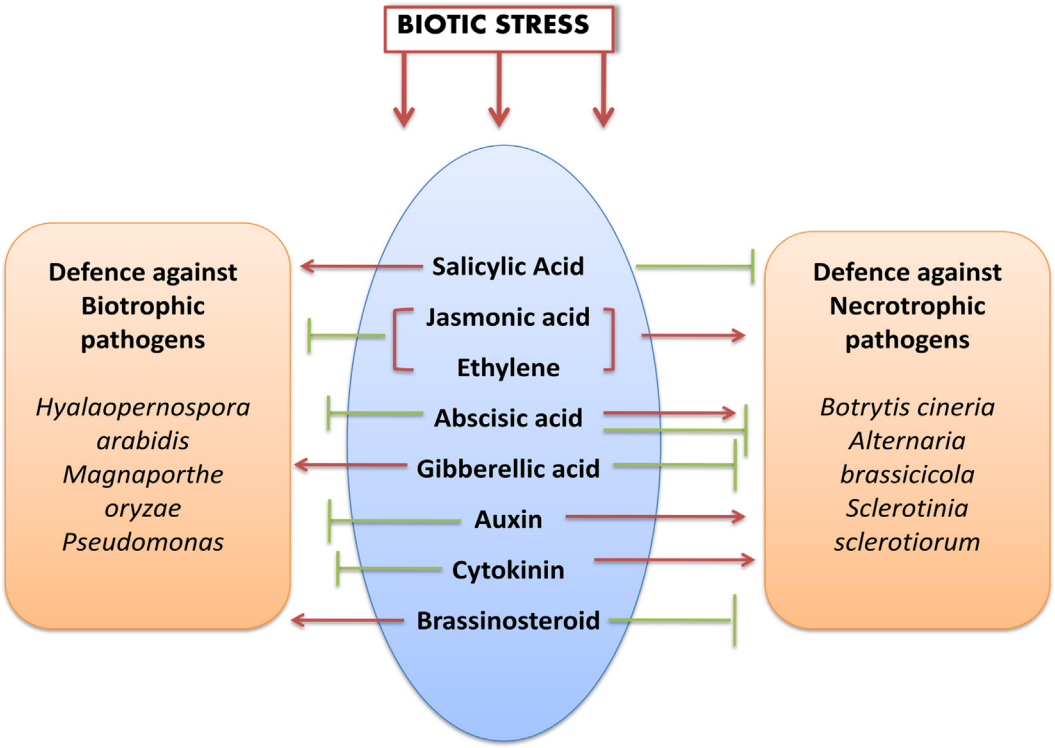

FIGURE 18.2 An abridged representation of the role of numerous hormones in the controlling of plant defense against several biotrophs and necrotrophs. A favorable interaction is indicated by an arrow, and an unfavorable interaction is indicated by a blocked line. For additional information and acronyms, see text. *Modified from Bari and Jones (2009).*

Ralstonia solanacearum's ET modifies the expression of genes related to the host (Valls et al., 2006). Astonishingly, *Botrytis cinerea* can create ET in tomatoes and in vitro (Cristescu et al., 2002). However, there is enough ET present to activate plant defenses when there is an invasion (Chague et al., 2006). The virulence of *Fusarium oxysporum* involves the ethylene receptor ETR1. The *Fusarium* wilt symptoms are statistically less severe in etr1-1 mutant *Arabidopsis* plants than in Col-0 (*Arabidopsis* ecotype) plants, which is demonstrated by the pathogen's diminished vascular growth and enhanced gene expression that responds to SA (Pantelides et al., 2013). According to Seo et al. (2016), L-histidine stimulates ET signaling in tomatoes and *A. thaliana*, which results in resistance toward *R. solanacearum* and *B. cinerea*. Yariyama et al. (2019) also investigated the administration of L-histidine exogenously and its effects in inhibiting bacterial diseases in rice seedlings. L-histidine treatment of germinated rice seeds elevated the expression of the ET-responsive OsGLP8-12 and the ET biosynthesis-related OsACS2. These findings implied that ET signaling may be necessary for the control of bacterial rice seedling illnesses. Xue et al. (2021) elucidated the significance of the foliar application of Silicon (Si) against the late blight-causing oomycete pathogen *Phytophthora infestans*. It was discovered that Si treatment of potato plants enhanced resistance to

LB in detached leaves and plants, along with stimulation of reactive oxygen species (ROS) and expression of genes involved in pathogenesis. In terms of the hormone mechanisms underlying Si-mediated LB resistance, we discovered a sharp rise in the concentration of ET 15 min after Si spraying. Plants were shown to have higher levels of JA, JA-Ile (jasmonyl-isoleucine), and lower levels of SA 1 day after Si spraying and another 1 day after *P. infestans* EC1 infection. All of these findings show that ET is critical for triggering plant immune responses and that it can be used to protect crops against disease infestation (Li et al., 2019; Zhang et al., 2020).

7. Cytokinins

Cytokinins are adenine molecules substituted with isoprenoid units (Fig. 18.1). The enzyme catalyzing the conversion of isoprenoid to other different forms of CKs was called isopentenyl transferase (IPTs). Caplin and Steward (1948) discovered that coconut milk contained an active metabolite that had the strongest favorable effect, on cell proliferation. Skoog and Miller discovered in the 1950s that autoclaved herring sperm DNA was an effective stimulator for the proliferation of cultivated tobacco pith cells (Miller et al., 1955). The active constituent was identified as 6-furfurylaminopurine and was called kinetin. Zeatin was later discovered to be the first naturally produced cytokinin in immature maize endosperm (Letham, 1973). Since their discovery, cytokinins have been associated with almost every phase of plant development including cell multiplication, shoot initiation, leaf senescence, and photomorphogenic development (Mok, 1994). The effects of cytokinins on seed germination are less evident; yet, multiple investigations have indicated that cytokinins can influence germination, especially when germination conditions are less than ideal. In addition to plants, microorganisms, microalgae, and insects that are connected with plants can also produce CKs. Plant cells transduce cytokinin signals using a two-component phosphorelay mechanism, which is widely utilized by microbes to recognize and respond to environmental cues. There has long been speculation that cytokinins play a role in plant-pathogen interactions. The exogenous administration of molecules with cytokinin activity changed the amounts of host immune responses or vulnerability to infections in plants. For instance, plant cell cultures maintained in elevated cytokinin concentrations exhibited upregulation of defense and stress genes (Schafer et al., 2000). Even though CK's role in plant defense is unclear, there is evidence that it is involved in the control of some pathogen-specific defense responses. Club root disease in *Arabidopsis*, which is brought on by *Plasmodiophora brassicae*, is largely influenced by CK (Siemens et al., 2011). Tumor-inducing fungi such as *Ustilago maydis*, *Fusarium pseudograminearum* (Sørensen et al., 2018), and *Claviceps purpurea* (Hinsch et al., 2015) can create CKs. Since many years ago, it has been understood that CKs can help plants survive microbial diseases as reported in tobacco and *Arabidopsis* (Choi et al., 2010; Grosskinsky et al., 2011; Naseem et al., 2014). Studies on *Arabidopsis* demonstrated that host cytokinins play a crucial part in plant immunity. White clover mosaic potexvirus susceptibility was decreased and defense gene expression was also increased when cytokinins were applied to bean plants (Clarke et al., 2000). Before pathogen administration, large doses (10−100 μM) of the synthetic cytokinin 6-benzyl amino purine (BA), produced from isoprenoids, were exogenously given to

Arabidopsis plants. The biotrophic oomycete *Hyaloperonospora arabidis (Hpa)* became less susceptible as a result (Argueso et al., 2012). Identical outcomes against *Pseudomonas syringae* pv. *tomato (Pst) DC3000* were obtained in *Arabidopsis* when it was treated with 1 μM of transzeatin, a naturally occurring isoprenoid with a considerable affinity for cytokinin receptors (Choi et al., 2010). The *ahk2,3* plants, which have mutations in two of the three genes that encode cytokinin receptors (*ARABIDOPSIS HISTIDINE KINASE 2* and *3*), did not, however, display the same cytokinin disease-protective effect, demonstrating that the action of cytokinins in this circumstance itself is brought on by cytokinin-regulated biological mechanisms (Albrecht and Argueso, 2017). The fact that most of the trials used exogenous hormone applications made it difficult to discern how much cytokinin is created naturally and from external sources, which was another issue when describing the importance of cytokinins in plant defense. The introduction of transgenic *Arabidopsis* plants overexpressing IPT genes, in which the natural levels of cytokinin are increased by 100 times, offered an obvious remedy for this. A reduction in *Pst* growth was observed in these plants suggesting that elevated levels of cytokinins can both exogenously and endogenously inhibit pathogen growth (Kakimoto, 2001; Choi et al., 2010). In addition to reduced pathogen development, plants treated with cytokinin showed increased expression of defense genes as well as callose accumulation to levels significantly higher than those reported with pathogen exposure alone (Argueso et al., 2012). It is important to note that plants treated just with cytokinin do not display appreciable amounts of defense reactions in the absence of pathogens or elicitors. In this regard, the function of cytokinin in plant defense is akin to that of priming agents, which increase defensive reactions, activated only when a pathogen or elicitor is detected. Grosskinsky et al. (2011) studied the exogenous application of kinetin (1−18 μm) on *Nicotina tobaccum* infected with *Pseudomonas syringae* pv. *tomato* (PsT) DC3000. The application of 10 μM kinetin to detached leaves of tobacco for 24 h increased resistance to PsT infection by up to 95%. High cytokinin concentrations applied to rice plants increased the expression of defense genes against the *Magnaporthe oryzae* (Jiang et al., 2013). According to numerous studies published in the literature, elevated concentrations of cytokinins in plants are associated with providing viral resistance and even nematodes (Pogany et al., 2004; Shanks et al., 2016). Studies on many plant species, including *Arabidopsis*, have shown that SA content and signaling are, at the very least, primarily responsible for the decrease in pathogen growth seen in cytokinin-induced immunity (Naseem et al., 2012). According to reports, cytokinins also helped plants defend themselves against potentially dangerous bacteria in the living soil by altering their physiological and morphological characteristics (Zhang et al., 2021). Veselova et al. (2021) reported that the application of exogenous zeatin improved wheat resistance to *Stagnospora nodorum* by suppressing the ethylene signaling pathway and activating genes that depend on SA. As a result, ethylene prevented the SA-dependent resistance mechanism from being activated, by further inhibiting the cytokinin signaling pathway. Recent research also established that CK played a beneficial effect in triggering rice resistance to brown plant hopper (BPH). In addition, also showed that CK and JA work together to cause BPH resistance in rice. To fully comprehend the molecular processes underlying the interactions between CK and JA, additional research is still needed (Zhang et al., 2022).

8. Gibberellic acid

The tetracyclic diterpenoid compound known as gibberellins (GAs) has played a significant role in plant development (Fig. 18.1). GAs are plant growth regulators that control several developmental events, such as stem elongation, germination, dormancy, blooming, and fruit senescence. GAs substantially encourages intact plants to grow longer cells. They are produced by the terpenoid pathway and require three enzymes *viz*., cytochrome P450 monooxygenase (P450), 2-oxoglutarate dependent dehydrogenase, and terpene synthase (TPSs) (2 ODDs). Gibberellin (GA) was first discovered to be a chemical secreted by the fungus *Gibberella fujikuroi* that causes the rice disease known as "bakanae" (Kurosawa, 1926). GA stimulated the degradation of DELLA proteins, which are growth inhibitors that are detrimental to plant growth. The rice DELLA protein slender rice1 (SLR1) interacts with the rice GA receptor gibberellin insensitive dwarf1 (GID1) in a GA-dependent manner. A ubiquitin E3 ligase SCF complex as well as 26S proteasome are involved in the ubiquitination and destruction of DELLA as a result of GID1 binding to DELLA. The earliest discovery of GAs in plants came from their isolation, primarily from immature seeds where the concentrations were high. Data from pure, derivatized extracts obtained through gas chromatography and mass spectrometry (GC-MS) have served as the major source for their identification from natural sources since the early 1970s. In addition to higher plants, fungi and bacteria also synthesize GAs (MacMillan, 2001). GAs are thought to be secondary metabolites produced by bacteria and fungi that serve as signaling agents to develop interactions with host plants. The explanation of signaling elements involved in defensive responses has not given GA much attention. However, recent research indicates that elements of GA signaling contribute significantly to both plant disease resistance and vulnerability. Recent research has revealed that DELLA proteins in *Arabidopsis* modulated SA- and JA-dependent plant immunological reactions (Navarro et al., 2008). A quadruple-*della* mutant of *Arabidopsis* lacking four DELLA genes was found vulnerable to necrotrophs, viz. *B.cinerea*, however, plant was found immune to biotrophs (Navarro et al., 2008). These results indicate that DELLA proteins increase necrotroph resistance by stimulating JA/ET-dependent immunological responses. However, susceptibility toward biotrophs increased due to the suppression of SA-dependent defense responses in *Arabidopsis*. As a result, SA- and JA/ET-based plant defensive response pathways seem to be integrated by DELLA proteins. DELLA proteins have also been shown to integrate responses to various hormonal and environmental signals. Plant defense responses have been demonstrated to be impacted by mutations that impair GA perception. In contrast to wild-type plants, the GID1 rice mutants, which lack the GA receptor, aggregate additional GA and exhibit improved immunity to the blast fungus *Magnaporthe grisea* (Tanaka et al., 2006). The functions of SA and JA have been thoroughly studied in cereals suffering from Fusarium head blight (FHB), but the significant functions of phytohormones are still less clear. Shaul et al. (1996) studied the effect of GAs on the prevention of postharvest Botrytis blight in cut roses. The disease was suppressed after the application of 1 mM GA3. Paclobutrazol (PBZ) was added to the GA3 solution to improve the control of Botrytis blight caused by GA3. Hed et al. (2011) employed GAs to treat Chardonnay and Vignoles grapes for bunch rot. *Botrytis cinerea* was principally responsible for harvesting bunch rot of wine grapes, a persistent issue that limits the output of eastern vineyards,

particularly on varieties with compact clusters. To lessen bunch rot and reduce the compactness of Chardonnay and Vignoles clusters, GA sprays were tested for effectiveness. In Vignoles and, to a slightly smaller extent, in Chardonnay, GA applications reduced the quantity of berries per centimeter as well as the frequency and severity of bunch rots for 3 years; nevertheless, the efficiency of GA was frequently reliant on the timing and rate of application. Ding et al. (2013) investigated gibberellic acid's defense toward the potato purple top (PPT) phytoplasma infection in tomatoes. The goals of this study were to understand how GA affects plant defenses against phytoplasmal infection and to determine whether pretreatment with GA would protect tomatoes from a future phytoplasmal attack. Results showed that, after exogenous application of hormone and simultaneous phytoplasma graft inoculation, an upregulation of SA synthesis, signaling, and downstream defense response genes *ICS1*, *NIM1*, and *PRP-1* was observed, which are responsible for systemic acquired resistance. However, downregulation of gene encoding GA signaling component, as well as growth inhibitor DELLA protein (*GAI*), was observed. Given that the differential regulation of the aforementioned genes was associated with an increase in the activity of the defense-related enzymes GLU and chitinase (CHI), it makes sense that GA took part in the synchronization of signaling networks to aid the host's defense and sustain regular growth amid PPT phytoplasma's stress-inducing activities. Burhow et al. (2016) investigated the relationship between a variety of phytohormones and wheat FHB pathogenesis. Rigorous research of endogenous hormone profiles revealed altered cytokinin, GA, and JA metabolism in a cultivar resistant to FHB. However, plants infected with *Fusarium graminearum* enhanced ABA, JA, and SA in both FHB-susceptible as well as resistant cultivars. Later research found that ABA or GA coapplication with a fungus challenge boosted or lowered the spread of FHB. Because ABA enhanced the activation of early-infection genes involved in cytoskeletal rearrangement as well as hydrolases while gibberellins decreased the activation of genes involved in nitrogen metabolism, these phytohormones' actions may be linked to changes in the *F. graminearum* transcriptome. In axenic circumstances, neither ABA nor GA had a discernible impact on the growth or sporulation of the *F. graminearum* fungus. Moosavi (2017) studied the impact of GAs on plant defense mechanisms and the severity of *Meloidogyne javanica* disease in tomato plants. The activity of the cytoplasmic enzymes, viz. PAL, PPO, and CAT, every other day for the first week following inoculation with *Meloidogyne javanica*, then weekly for 8 weeks, it was determined whether gibberellin could cause biochemical defense responses in tomato roots. Gibberellin induced a plant defense response because the PAL, POD, and PPO activity was at its peak in the first week following spraying. After that, the amount of enzymes began to decrease during weeks 2 and 3, climbed during the fourth week, and then resumed its downward trend until the experiment's end. Gibberellin promoted plant innate defensive responses and increased plant resistance based on nematode reproduction and plant growth. DELLA proteins have also been shown to be essential for weakening *Arabidopsis* defense mechanisms in response to the labial saliva of the beet armyworm (*Spodoptera exigua*) (Lan et al., 2014). However, exogenous GA treatment reduces the root-knot nematode (*Meloidgyne graminocola*) resistance of rice (*Oryza sativa*) by degrading DELLA and decreasing JA signaling (Yimmer et al., 2018). According to research, GA3 may be utilized to enhance pest control in rice by promoting rice's resistance to brown planthopper *Nilaparvata lugens* (Stål) (Wang et al., 2021a, b).

9. Brassinosteroids

Brassinosteroids, which have recently been recognized as a novel family of plant hormones, seem to be present throughout the whole plant kingdom and have specific physiological effects on plant growth and development. The use of these hormones in agriculture to boost productivity, improve plant disease resistance, and reduce environmental stress has enormous potential (Ahanger et al., 2018). In 1979, *Brassica napus* was used to extract the first brassinosteroid known as brassinolide. Since then, several research works have been conducted globally on the isolation, characterization, and uses of brassinosteroids from various plant sources. In addition to being common, brassinosteroids have been found in dicotyledons, monocotyledons, gymnosperms, and algae. Although it is present in almost every part of the plant, pollen and immature seeds have the greatest concentration. Possibly biosynthesized in all organs and tissues of plants (Fujioka et al., 1996; Schmidt et al., 1997; Tong and Chu, 2018). The concentration of endogenous brassinosteroids differs from one tissue and one organ to another. They are known to have an impact on a variety of plant developmental processes, including seed germination, cell proliferation and elongation, flowering, sexual maturity, and senescence (Ahammed et al., 2020; Peres et al., 2019). However, the impact on biotic stress has not yet been fully investigated. Brassinosteroids are known to move from roots to shoots through the xylem, according to several radiolabeled experiments. At nano to micromolar doses, it has remodulated how cells develop and differentiate (Clouse and Sasse, 1998; Anfang and Shani, 2021). They are proven to be potential PGRs in agriculture as well (Fujioka and Sakurai, 1997; Sasse, 1997; Peres et al., 2019). Plant sterols are used to make brassinosteroids, with campesterol and its analogs serving as the precursors of brassinolide. Early C-6 oxidation and Late C-6 oxidation are the two routes from campesterol that brassinolide production is predicted to follow (Fujioka et al., 2000; Zhao and Li, 2012). Early C-6 oxidation pathway is the name of the biosynthetic route that produces brassinolide by hydroxylation and epimerization. Similar to how teasterone is transformed into typhasterol, 6-deoxoteasterone is epimerized to 6-deoxotyphasterol via the 3-oxo forms in the late C-6 oxidation pathway (Abe et al., 1994; Yokota et al., 1994; Choi et al., 1997; Ye et al., 2011). Between auxins and brassinosteroids, there is a strong synergism (Yopp et al., 1981; Takeno and Pharis, 1982; Arteca et al., 1983; Meudt and Thompson, 1983; Choi et al., 1990; Cao and Chen, 1995; Sankar et al., 2011; Chaiwanon and Wang, 2015). It is well known that BR has a role in controlling the plant's defensive response. It demonstrates effects on a wide range of microorganisms, including bacterial, viral, and even fungal plant diseases (Coll et al., 2015). Recent research has discovered various previously unrecognized functions of BR in disease response, despite the fact that BRs have long been recognized as a favorable modulator in plant resistance to disease (Nahar et al., 2013). According to reports, BR increases tobacco's resistance to *Oidium* sp. Similar to this, BR has also demonstrated effects in raising rice plant resistance to *Magnaporthe grisea* infection (Nakashita et al., 2003). The development of disease symptoms brought on by *Verticillium dahlia* will be prevented by the exogenous application of a kind of BR called 24-epibrassinolide on tomato plants. The application of BR to potato plants also demonstrates their resistance to *Phytophthora infestans* (Vasyukova et al., 1994). It was studied that resistance was linked to rising levels of ethylene and abscisic acid. This suggested that BR cross-talks with other hormonal signaling in plants to mediate their defensive

response (Gruszk, 2018). For plants to defend themselves against RNA viruses, it is seen as crucial. Even at a dose of 5 mg/ha, epibrassinolide (epiBL) application in barley during the tillering period considerably reduced leaf disease brought on by *Helminthosporium teres Sacc*. and boosted grain production (Pshenichnaya et al., 1997; Volynets et al., 1997). On the cucumber plant field, epiBL has been shown to have yet another protective effect (Churikova and Vladimirova, 1997; Hayat et al., 2003; Dehghan et al., 2020). Zhang et al. (2018) elucidated the beneficial role of BR in enhancing tea plant resistance to *Colletotrichum gloeosporioides*. Exogenous EBR (24-epibrassinolide) pretreatment significantly lowered lesion progression in tea leaves inoculated with pathogen followed by decreased aggregation of H_2O_2 in tea leaves. Exogenous EBR enhanced PAL activity, indicating that it activated the phenylpropanoid pathway. By accelerating the deposition of callose and lignin, brassinosteroids have been demonstrated to promote innate immunity toward *Colletotrichum acutatum*, responsible for causing anthracnose disease in plants such as strawberries (Furio et al., 2018). Song et al. (2022) demonstrated that after inoculating mango fruit with *C. gloeosporioides*, 1 μmol L^{-1} of EBR treatment substantially decreased disease severity. However, an in vitro test showed that EBR had no direct antifungal action against *Colletotrichum gloeosporioides*. EBR dramatically enhanced the accumulation of flavonoids, lignin, and total phenolics in mango fruit as well as the activities of PAL, POD, chitinase, cinnamate-4-hydroxylase (C4H), GLU, and 4-hydroxycinnamate CoA ligase.

10. Conclusions

The signaling networks and developmental mechanisms that enable plants to adapt to a range of biotic and abiotic stressors are crucially regulated by plant hormones. Significant progress has been made in recognizing SA, JA, and ET, which play critical roles in plants' reactions to biotic stress. Recent research works have also suggested that other hormones, including auxin, GA, CK, and BR, are involved in the signaling pathways of plant defense, though it is less clear how these hormones operate in plant defense. The current review focuses on the most recent developments in our comprehension of the function of these hormones in controlling how plants react when they are under attack from different diseases and pests. The majority of the times, plant defense systems are multilayered and complicated, making them efficient against a wide range of diseases. The physical and chemical barriers that are naturally present in plants prevent pathogen access and infection. Different hormones have favorable or negative effects on various biotrophs and necrotrophs, depending on the nature of plant-pathogen interactions. Plant hormone signal transduction pathways are not distinct but rather interwoven in a complex regulatory network that encompasses numerous defense signaling pathways including developmental activities (Fig. 18.2). Additionally, it has been discovered that crosstalk across phytohormone pathways is important for controlling viral defense response. But despite technical advancements in the 21st century, we still do not have precise knowledge of the genes that control phytohormones and the pathways that impact viral development. It is important to emphasize how the pathosystem affects the kinds of interactions and how plants respond to stimuli, as well as the quantity, type, and tissue where hormones are produced. Understanding, how plants coordinate various hormonal components in response to

diverse developmental and environmental cues will be a major area of future research. Some hormones alter the host physiology, protein synthesis, and plant defenses against microbial attack when given to plants. Exogenous administration of JA and SA after harvest considerably lessens the harm caused by many diseases, directly and via the induction of antioxidant and defense mechanisms in various plant organs. Numerous plant diseases develop phytohormones or their functional imitators in addition to the hormones that plants naturally make to influence the regulatory network that controls plant defense. Recent research reveals that plant infections can modify the biosynthesis and signaling of hormones, causing hormonal imbalances and changes to plant defense mechanisms. One of the tactics certain infections employ to increase their virulence and spread illness is this. The mechanisms by which pathogen effectors modulate hormone signaling components to induce virulence are, however, largely unknown. The many elements engaged in the intricate connections between hormone-regulated defense signaling pathways are the subject of current world wide transcriptome profiling analyses in relation to pathogenic threats, which are revealing important information. However, further research on in-depth time course experiments and a deeper comprehension of the intricate regulatory molecular systems that control plant hormone signaling and defense responses are crucial for developing successful plans for genetically modifying crops to resist disease and pests in time ahead.

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