ROLE OF SALICYLIC ACID IN PLANTS DEFENSE MECHANISMS AGAINST PATHOGENS

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Abstract

Salicylic acid (SA), a signaling molecule known for its role in plant defense against disease. SA can trigger plant defensive mechanisms, making it a viable alternative to biocidal agrochemicals. The agricultural sector's growing demand for global food supplies is a primary motive behind developing appropriate methods of controlling diseases that are effective not just against target pathogens but also against those that may emerge in the future. Plants have specific structures, compounds, and sophisticated mechanisms that help them fight diseases. Pathogens are continually inventing new ways to break plant defenses, therefore knowing these defense mechanisms and pathways is essential for developing novel disease-prevention treatments. Several signaling molecules that control the production of defense-related compounds are involved in plant defense pathways. SA, ethylene (ET), jasmonic acid (JA), and abscisic acid (ABA) are all involved in these mechanisms. SA is the subject of this review article because of its importance in various ways the plants tolerate to biotic stress. This review article focuses on the role of SA in plant pathogen defense. The mechanisms of action of SA in plant defense, SA and systemic acquired resistance, SA in plant-pathogen resistance, effects of SA on antioxidant systems, and future directions of SA were all discussed in depth. Scientists will be able to create more efficient techniques for safeguarding plants from infections for sustainable agriculture if they have a deeper grasp of plant defense systems.

1.INTRODUCTION

Plants have mechanisms that allow them to tolerate drought, salt, severe temperatures, poisons, and pathogen-caused diseases (Purohit et al., 2019; Suzuki et al., 2014). Many bacteria, fungi, viruses, and nematodes attack plants, frequently with disastrous consequences. Plants are sensitive to a variety of diseases that may be present in the environment, as well as environmental variables that affect plant immunity, from the time they germinate. Some pathogens infect, reproduce, and complete their life cycles within a living host, while others kill the host as the infection progresses. However, plants have developed a number of defense systems to combat infections (Kissoudis et al., 2014). To protect plants from deadly diseases that can cause significant economic losses, modern agriculture mainly relies on integrated disease management strategies. Researchers would be able to create better disease management approaches for crop protection if they had a better grasp of plant defense signaling. Recent advances in systems biology, transcriptomics, metabolomics, and genomics have considerably increased our awareness of plant defense and contributed to crop protection technology advancements (Bektas & Eulgem, 2015).

SA is a signal molecule that stimulates the plant's defense mechanisms and response to biotic and abiotic stressors (Martín-Mex et al., 2015; Pieterse et al., 2012; Robert-Seilaniantz et al., 2011). Since constitutive SA buildup is usually connected to retarded plant development and lower plant fitness, SA production and SA-mediated signaling are strictly regulated (Chandran

et al., 2014; Pajerowska-Mukhtar et al., 2012). SA is a plant growth inhibitor that is water-soluble (Muthulakshmi & Lingakumar, 2017). Through morphological, physiological, and biochemical routes, SA, a plant hormone, stimulates plant defense against several biotic and abiotic stresses (Prodhan et al., 2018). SA has numerous physiological effects in plants such as enhancing the response of plants to pathogens challenges, improving phytosynthesis, and triggering or modifying endogenous signaling to tolerate a variety of pressures. One of SA's most essential functions is to increase antioxidant synthesis. Antioxidants protect the plants from negative of reactive oxygen species (ROS). SA improves the plants' response to tolerate and resist several ailments by triggering the activities of pathogenic pathogens when their internal concentration is increased (Kumar, 2014). SAR is the most essential sort of plant immunity in terms of agronomy (Klessig et al., 2018), and it can be triggered by signal molecules linked to plant disease resistance, such as SA and a variety of synthetic chemicals (Shahmoradi & Naderi, 2018). Functional analogs of SA are among these molecules, and they can activate plant defensive responses, making them appealing alternatives to traditional biocidal agrochemicals (Bektas & Eulgem, 2015). Despite their potential to activate resistance to a variety of diseases in vitro by inducing SAR genes produced by SA inducers, producers must consider a number of variables when utilizing them in crop protection, including disease control (Canet et al., 2010). Several signaling molecules that control the production of defense-related compounds are involved in plant defense pathways and among them include SA, ET, JA, and ABA. SA is considered in this review because it is important in various processes in plants such as biotic stress regulation. This review article focuses on the mechanism of action of SA in plant defense, SA and systemic acquired resistance, SA in plant-pathogen resistance, effects of SA on antioxidant systems, and future directions of SA will all be discussed in depth. Scientists will be able to create more efficient techniques for safeguarding plants from infections for sustainable agriculture if they have a deeper grasp of plant defense systems.

2. MECHANISM OF ACTION OF SA IN PLANT DEFENSE

Many plant hormones, including salicylic acid (SA), act as endogenous signals to activate plant immunity and improve plant defense against disease. Biotrophic pathogens predominantly activate and inhibit the SA pathway, which is frequently impeded by feedback loops and cross-talk with other phytohormones such as JA and ET modify the SA signal (Pieterse et al., 2012; Vlot et al., 2009). Exogenous injection of SA enhances resistance to tobacco mosaic virus (TMV) (Vlot et al., 2009), cauliflower mosaic virus (CMV) (Love et al., 2007), and turnip crinkle virus (TCV) (Kachroo et al., 2000) in Arabidopsis thaliana. The Agrobacterium tumefaciens-induced developed gall symptoms are diminished when Nicotiana benthamiana is treated with SA (Anand et al., 2008). It also helps to prevent the bacteria Erwinia amylovora from causing pear fire blight (Sparla et al., 2004). It was also tested for resistance to the powdery mildew pathogen Oidium sp. (Nakashita et al., 2002), as well as other pathogens such as Alternaria solani-caused tomato leaf blight (Spletzer & Enyedi, 1999), and Monilia fructicola-caused cherry fruit rot (Cao et al., 2008).

Two separate and segregated routes are used to make SA (Ferrari et al., 2003). Decarboxylation of trans-cinnamic acid to benzoic acid, followed by hydroxylation to SA, is how the phenylalanine pathway produces it. Cinnamic acid is hydroxylated to produce o-coumaric acid, which is then decarboxylated to produce SA (Lee et al., 1995). Isochorismate synthase (ICS), which converts chorismate to isochorismate, is linked in SA synthesis in the isochorismate pathway (Wildermuth et al., 2001). Calmodulin-binding protein 60 g, for example, affects ICS1 expression (CBP60g). PAMP recognition activates isochorismate synthase and SA biosynthesis by causing calcium influx in the cytosol, which is then conveyed to the calmodulin-binding proteins CBP60g and WRKY28 (Reddy et al., 2011). Cyanogenic glycosides like prunasin and mandelonitrile have recently been discovered in the third technique for peach SA generation (Diaz-Vivancos et al., 2017). EDS1 (for improved disease susceptibility) and PAD4 (for phytoalexin insufficiency) are two lipase-like proteins that function upstream of SA in Arabidopsis (Cui et al. 2017). EDS1 is a crucial node that regulates the generation of SA, which aids in the enhancement of defense signals. It forms a heterodimer with PAD4 that

transmits ROS-derived signals, leading to increased SA synthesis by buildup of benzoic acid (BA) and conversion to SA by the enzyme benzoic acid 2-hydroxylase (BA2H) (Rietz et al., 2011; Rustérucci et al., 2001).

SID2 encodes for an ICS implicated in SA biosynthesis, as a mutation in it decreases SA production and PR1 gene expression in A. thaliana. The SA control is aided by EDS5, also known as SID1. It is necessary for the expression of PAD4 and has a function in the transfer of SA precursors (Nawrath et al., 2002; Serrano et al., 2013). EDS4 is another component implicated in SA signaling and SA-induced SAR (Gupta et al., 2000). SID2, which produces SA, is activated by EDS1, PAD4, and EDS4 (Glazebrook, 2005). The EDS5 protein transports SA from the chloroplast to the cytosol, where it is glycosylated or methylated and rendered inactive (Yamasaki et al., 2013). After pathogen infection, SA 2-O-D-glucoside (SAG) is moved to the vacuole and digested, releasing free SA (Park et al., 2007). Methylation of SA results in the formation of methyl SA (MeSA), a mobile SAR signal that transports from infected to uninfected parts before reverting to SA and triggering resistance. SA levels in inoculated leaves increase significantly after pathogen infection, but SA methyl transferase transforms it to physiologically inactive MeSA (SAMT). SA binds to the active site of the SA-binding protein 2 (SABP2) when the concentration of SA reaches a particular level, preventing MeSA from being converted back to SA (Park et al., 2007). The redox potential of the chloroplast cell wall is altered by methylation of SA, allowing it to penetrate farther into the cytoplasm of uninfected tissue. Because the levels of SA in the distal tissue are inadequate to prevent SABP2, MeSA is transferred and transformed to active SA, activating systemic defensive responses (Park et al., 2007).

NPR1 and NPR3/4 homeostasis impact defense signaling downstream of SA in a concentration-dependent way. During pathogen infection, this establishes the degrees and types of defense responses that should be activated. The master controller of SA-mediated defense genes is assumed to be NPR1 (Fu et al., 2012). Two cysteine residues (521 and 529, respectively) bind to SA (Wu et al., 2012). Pathogen-induced SA promotes NPR1 production and trafficking into the nucleus to interact with TGA transcription factors bound to the PR1 promoter's AS-1 (activation sequence-1) like region (Lebel et al., 1998). In the absence of infection, NPR1 is constantly destroyed by the proteasome, which is interfered by NPR3 and NPR4, the adaptors for the Cullin 3 ubiquitin E3 ligase (Fu et al., 2012). NPR4 keeps the levels of NPR1 low, but following infection, SA attaches to NPR4 in greater numbers, disrupting the NPR1–NPR4 connection and boosting NPR1 to enhance the occurrence of defense signaling. NPR3 binds NPR1 in plants with a sufficient level of SA, boosting NPR1 turnover and optimizing defense mechanisms while also resetting NPR1 levels (Moreau et al., 2012).

3. SALICYLIC ACID AND SYSTEMIC ACQUIRED RESISTANCE

Systemic acquired resistance (SAR) is a critical component of plant defense that relies on the accumulation of SA (Ghanbari et al., 2015; Maruri-López et al., 2019). SAR describes a plant's potential to establish long-term resistance to diseases in previously unaffected areas. If Non-expresser of PR genes interacts with transcriptional cofactors, SA can enhance the amount of pathogenesis-related (PR) proteins having antimicrobial properties (Ali et al. 2018; Innes 2018; Klessig et al., 2018; Sudisha et al., 2012). Although SA is an important and vital part of SAR, it is not the main mobile signal. Methyl salicylate, azelaic acid, pipecolic acid, and its derivative N-hydroxypipecolic acid are among the mobile and volatile signals indicating systemic acquired resistance (Bernsdorff et al., 2016; Hartmann & Zeier, 2019; Park et al., 2007; Shah et al., 2014). During SAR, plants create one or more translocated signals, which trigger the plant's resistance mechanism in non-infected parts, preparing the plant for future attacks (Shah & Chaturvedi, 2013). After TMV infection, SAR has been found to develop in both local and systemic organs, with elevated plant defense-related genes such as pathogenesis-related (PR) gene families.

Tobacco, Arabidopsis, and cucumber plants have all been used to study PR protein-mediated defense responses (Bektas & Eulgem, 2015). The PR1 protein, according to a new study, binds to and sequesters host sterols that infections require for growth. The sterol-binding activity of PR 1 protein shows the route of action of an antibacterial protein (Gamir et al., 2017). SAR requires the accumulation of SA in addition to the expression of PR gene families. In infected host tissues,

SA levels rise locally and less systemically (Bektas & Eulgem, 2015; van Loon, 2016). The involvement of SA in SAR signaling was confirmed in studies using transgenic plants overexpressing a bacterial salicylate hydroxylase gene (the nahG gene), which efficiently decreases the quantity of endogenous SA which makes the plant sensitive to diseases (Bektas & Eulgem, 2015). According to previous labelling experiments, SA is mobile during SAR induction in TMV-infected tobacco and cucumber, and the majority of SA accumulates systemically in upper non-infected leaves of infected plants (van Loon, 2016). It was considered that SA produced via the phenyl ammonia-lyase (PAL) pathway was the main driver of disease resistance. The PAL pathway is important for SA generation in response to local cell death, according to an Arabidopsis mutation study published in 2001 (Kumar et al., 2015) and during SAR development, the isochorismate synthase-mediated pathway is more critical for continuous SA production.

SAR signal systems in plants can pursue a variety of pathways, according to several lines of research. SA functions as a systemic signal, according to investigations looking for the SAR systemic signal. In these studies, high SA in phloem sap was found in local and systemic tissues of infected plants (Conrath et al., 2015; Lyon, 2014). Tests on grafting nahG and wild-type tobacco plants, as well as cucumber leaf excision studies (Lyon, 2014), backed up this theory. Later research discovered that signaling takes place via the volatile molecule MeSA, which can cause tolerance to both infected and non-infected sections of the same plant (Kumar, 2014). When a virus attacks, endogenous MeSA levels rise dramatically. The SA carboxyl methyltransferase (SAMT) enzyme transforms SA to inactive MeSA in infected Arabidopsis tissues, producing MeSA (Kumar, 2014). In systemic tissues, MeSA is converted back to SA, resulting in resistance (Kumar, 2014). This conversion is catalysed in tobacco plants by salicylic acid-binding protein 2 (SABP2), a high-affinity SA-binding protein with SA methylesterase activity (Kumar, 2014). According to biochemical studies (Kumar, 2014), its esterase activity is required for the conversion of MeSA to SA and the synthesis of SAR in systemic tissue.

The production of SAR by an infecting pathogen is only effective for future pathogen infection because the first SAR-inducing infection usually results in sufficient tissue damage. This demonstrates that pathogen-induced SAR has a lower impact on disease resistance than SAR triggered before infection (Lyon, 2014). SA and its synthetic analogs have been shown to induce defense responses, including SAR when administered exogenously. The activation of defense-related genes and cell priming can be triggered by systemic resistance, resulting in more efficient elicitation of various defensive responses. Because of its rapid glycosylation and phytotoxicity, SA's efficacy as a plant protection agent has been limited. These substances promote other defensive systems, such as the JA pathway, to produce defense in addition to the SA signaling system (Conrath et al., 2015; Silverman et al., 2005). Plant defense responses are triggered by a number of synthetic and natural chemicals, in addition to SA analogs. Abiotic agents that function as resistance-inducing chemicals include natural metabolites, inorganic substances, and synthetic chemicals. A qualified resistance inducer must cause the plant's reaction to shifting from affinity to dislike (e.g. defense gene expression), and it cannot be antimicrobial or convertible into an antimicrobial chemical by the plant (Walters et al., 2013).

4. THE BIOSYNTHESIS OF SA IN PLANTS

4.1. Biosynthetic pathways

Two significant SA biosynthesis routes in plants are isochorismate (IC) and phenylalanine ammonia-lyase (PAL). Chorismate, a byproduct of the shikimate pathway, is often used to create SA via both routes (Dempsey & Klessig, 2012). The enzymes IC synthase (ICS) and PAL are required for these reactions. Arabidopsis, tobacco, tomato, Populus, sunflower, and pepper all have homologs for the ICS and PAL genes, demonstrating the importance of these SA biosynthesis mechanisms in surviving evolution (Catinot et al. 2008; Dehghan et al., 2014; Seyfferth & Tsuda, 2014; Yuan & Lin, 2008). Mutations in ICS1 in Arabidopsis result in an almost complete loss of pathogen-induced SA accumulation (Wildermuth et al., 2001). However, Arabidopsis quadruple PAL mutants, which have a 10% drop in PAL activity, accumulate 50% less SA than the wild type when infected with pathogens (Huang et al., 2010). The PAL pathway is necessary for plant immunity, while SA is produced mostly through the IC pathway.

ICS catalyses the conversion of chorismate to IC, which is then converted to SA in chloroplasts (Huang et al., 2020; Wildermuth et al., 2001; Zhou, 2018). In some bacteria, IC pyruvate lyases speed up the conversion of IC to SA (Dempsey & Klessig, 2012). Genes associated with bacterial IPLs, on the other hand, are absent from plant genomes. Because of the presence of bacterial enzymes that catalyze this conversion, as well as ICS, SA accumulates in chloroplasts over time (Mauch et al., 2001; Verberne et al., 2000). Plants' SA biosynthesis may be more advanced than bacteria's. SA export from chloroplasts is regulated by the MATE-transporter EDS5 (increased disease susceptibility 5) (Serrano et al., 2013). Because EDS5 mutants have trouble accumulating SA, this export appears to be required for SA accumulation and dispersion within the cell (Ermakova et al., 2021; Nawrath et al., 2002).

4.2. Regulation of SA biosynthesis

Salicylic acid synthesis is strictly regulated because constitutive SA accumulation is detrimental to plant fitness (Chandran et al., 2014; Ermakova et al., 2021). According to current research, calcium signaling regulates ICS1 transcription, which is necessary for the start of SA biosynthesis (Figure 1). The concentration of calcium ions (Ca2+) in the cytosol increases transiently when immunological receptors are activated. Calmodulin (CaM) and Ca2+ -dependent protein kinases are Ca2+ sensor proteins that detect an increase in intracellular Ca2+, commonly known as the Ca2+ signature (Brandt et al., 2015; Gao et al., 2013; Seybold et al., 2014). CaM binding regulates target protein function by transmitting Ca2+ signatures to downstream pathways. ICS1 transcription is regulated by the CaM-binding transcription factor CBP60g (Calmodulin Binding Protein 60g) and its homolog SARD1 (Systemic Acquired Resistance Deficient 1) during Arabidopsis immunity (Hartmann & Zeier, 2019; Qin et al., 2018). SARD1 does not appear to be a CaM-binding protein, although CBP60g requires CaM interaction to function (Nair et al., 2021). Despite their differences, CBP60g and SARD1 are partially redundant for ICS1 expression and SA accumulation during immunity.

The simultaneous control of ICS1 transcription by CBP60g and SARD1 appears to influence the temporal dynamics of SA biosynthesis: CBP60g contributes to SA biosynthesis early after P. syringae infection, but SARD1 contributes later (Nair et al., 2021). In response to CaM binding, CBP60a, which is very similar to CBP60g, inhibits ICS1 expression (Truman et al., 2013). CBP60g and SARD1 may bind to the ICS1 promoter in response to pathogen infection and boost its expression, displacing the negative regulator CBP60a from the promoter, at least in part (Wang et al., 2010). Unlike CaM, CDPK proteins have Ca2+ sensors and response sites built-in, allowing them to relay Ca2+ signatures to downstream components via phosphorylation. The CDPKs, CPK4, 5, 6, and 11, have been shown to relocalize to the nucleus, interact with, and phosphorylate the WRKY transcription factors, WRKY8, 28, and 48, during ETI mediated by the plasma membrane-associated immune receptors RPS2 (Resistance to P. Syringae 2) or RPM1 (Resistance to P. Syringae 1) (Gao et al., 2015). In WRKY8 or WRKY48 mutants, pathogen-induced ICS1 expression is decreased. WRKY28 interacts directly with the ICS1 promoter, which can be phosphorylated by CPK4, 5, 6, or 11, according to Bhardwaj et al. (2011). These findings suggest that during ETI, CDPKs send Ca2+ signals to WRKY transcription factors, activating ICS1 transcription (Wang et al., 2014).

Calcium signaling controls the maintenance of SA accumulation via regulating the transcription of enhanced disease susceptibility 1 (EDS1), a key regulator of the SA accumulation positive feedback loop (Du et al., 2009; Feys et al., 2001). CAMTA3/SR1 (Calmodulin binding transcription activator 3/Signal responsive gene 1), a CaM-binding transcription factor, binds to the EDS1 promoter to suppress transcription, and mutants of CAMTA3/SR1 have greater SA levels and enhanced immunity to P. syringae and Botrytis cinerea. CAMTA3/SR1 and its homologs CAMTA1/2, according to combinatorial mutant analysis, suppress the expression of CBP60g, SARD1, and ICS1 (Kim et al. 2013). The three CAMTA homologs thus cooperate to inhibit SA accumulation; however, it is unknown whether the CAMTA transcription factors directly target the promoters of CBP60g, SARD1, or ICS1.
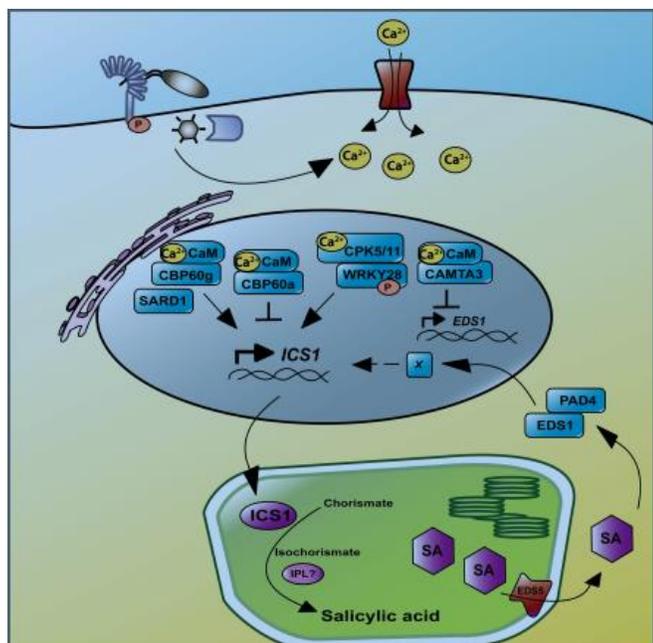
Figure 1. Calcium signaling controls the buildup of SA. MAMP or effector recognition raises intracellular Ca2+ levels, which controls calcium sensor proteins including CaM and CDPKs. CBP60g and CBP60a, both CaM-binding transcription factors, are positive and negative regulators of ICS1 transcription, respectively. SARD1, a CBP60a/g homolog, is not a CaM-binding protein but works in tandem with CBP60g to regulate ICS1 transcription. WRKY28, a DNA-binding protein whose activity is controlled by the CDPKs CPK5 and CPK11, also plays a role in ICS1 expression. By converting chorismate to the SA-precursor isochorismate, ICS1 mediates SA synthesis in chloroplasts. SA may be delivered into the cytosol via the MATE-transporter EDS5. The EDS1/PAD4 complex participates in the SA buildup positive feedback loop. The Ca2+/CaM-binding transcription factor CAMTA3 represses EDS1 transcription, which is a fine-tuning mechanism for SA buildup.

Recently, researchers discovered a CAMTA3/SR1-interacting protein that links CAMTA3/SR1 to ubiquitin-mediated protein degradation, boosting EDS1 production and resistance to P. syringae (Zhang et al., 2014). Finally, these findings show that Ca2+ signaling regulates SA accumulation during immunisation through transcriptional control of genes involved in SA production and maintenance. However, how plants coordinate positive and negative regulators of SA biosynthesis and accumulation in space and time is currently unknown (Wu et al., 2012).

5. SA IN PLANT-PATHOGEN RESISTANCE

SA is a plant hormone that defends plants from microbial diseases such as viruses, bacteria, fungus, and oomycetes (Kunkel & Brooks, 2002; Vlot et al., 2009). Endogenous SA levels and plant resistance to biotrophic and hemibiotrophic diseases are well understood (Glazebrook, 2005). Exogenous SA confers local and systemic acquired resistance to many diseases such as Fusarium oxysporum, Alternaria alternata, Magnaporthe grisea, Colletotrichum gloeosporides (Jendoubi et al., 2015; Kundu et al., 2011; Le Thanh et al., 2017; Wang & Liu, 2012). Exogenous treatment of 1 mM SA nearly totally reduced the development of bacterial wilt disease in chili plants (Chandrasekhar et al., 2017). Treatment of SA to broad beans reduced red light-induced resistance to Botrytis cinerea but had no effect on black light-induced vulnerability (Khanam et al., 2005). SA increased tomato susceptibility to B. cinerea in a dose-dependent manner. Surprisingly, SA-induced increased B. cinerea resistance has also been found in tomato and Arabidopsis plants (Ferrari et al., 2003; Li & Zou, 2017). Defense signaling based on SA is thought to be inferior to defense signaling based on JA/ET (Glazebrook, 2005). Because SA and ET/JA hormone communication routes aren't always hostile (Robert-Seilaniantz et al., 2011), it's critical to investigate them in a range of plant-pathogen systems and field situations.

6. EFFECT OF SA ON ANTIOXIDANT DEFENSE SYSTEM OF PLANTS

Exogenous substances, such as the plant growth regulator salicylic acid (SA), can cause the rapid and coordinated activation of plant defense genes, with the matching gene products boosting disease resistance (Catinot et al., 2008; Nie, 2006; Soylu et al., 2003). This is a promising strategy for reducing reliance on chemical bactericides to prevent crop bacterial infections (Abd-El-Kareem et al., 2009). SA is engaged in signal transmission, which causes the expression of pathogenesis-related (PR) proteins, some of which have non-enzymatic roles, as well as particular enzymes that catalyze processes to create defensive chemicals like polyphenols (Chaturvedi & Shah, 2007; Vimala &

Suriachandraselvan, 2009). SA, also known as ortho-hydroxy benzoic acid, is a naturally occurring substance. SA is a signal molecule that stimulates the body's defense mechanisms (Halim et al., 2006; Jalil & Ansari, 2019; Joseph et al., 2010). Plants' secondary metabolite pathways are altered when they are exposed to biotic and abiotic stressors (Khan et al., 2014). SA interacts with proline metabolism and ethylene production to minimise the negative effects of heat stress on wheat photosynthesis, according to Khan et al. (2013). Similarly, when SA is applied foliar to wheat, the rooting media has been shown to regulate growth and rate of photosynthesis (Arfan et al., 2007).

According to Wang et al. (2021), SA treatment increased the activity of many antioxidant enzymes in wheat. SA's foliar application mitigated the adverse impacts of several abiotic stressors and regulated plant photosynthetic processes, according to (Zafar et al., 2021). In a few papers, exogenous application of SA enhanced wheat tolerance to pathogen stressors (Sultana et al., 2019). Under severe drought stress, SA inhibited the activity of 1-amioncyclopropane carboxylic acid synthase (ACS) and hence lowered the production of ethylene. These findings suggest that applying SA to plants reduces drought-induced reductions in growth and photosynthesis through boosting proline concentration (Nazar et al., 2015). SA has been shown to reduce oxidative damage in rice seedlings by upregulating the methylglyoxal (MG) antioxidant defense and detoxification system (Mostofa et al., 2015). Furthermore, the application of SA raised the amounts of proline, soluble carbs, and soluble protein as well as superoxide dismutase (SOD), peroxidase (POD), catalase (CAT), and ascorbate peroxidase (APX) (Liu et al., 2016). Exogenous and endogenous SA improved cold tolerance by modulating oxidants and antioxidant enzyme activity in both cold-sensitive and cold-tolerant barley cultivars (Salih Mutlua et al., 2016). Acetylsalicylic acid and 24-epibrassinolide were used to control basal heat tolerance in tomato seedlings (Khan et al., 2015). The exogenous SA treatment activated two signaling arms of the Arabidopsis UPR (Mishiba et al., 2013). The foliar application of SA protects the PSII complex from photo-damage by enhancing transcription of the psbA gene (producing D1 protein) and moderating photo-oxidation caused by high antioxidant enzyme activity, allowing PSII to recover from heat stress more quickly (Wang et al., 2012).

The application of salicylic acid to maize seedlings enhanced the percentage survival under abiotic factors such as heat, implying that SA enhances maize seedling stress resistance. Salicylic acid has been found to alter the Halliwell-Asada pathway in maize roots during high-temperature stress (Khanna et al., 2017). Chilling stress increased the production of endogenous SA, according to Wang et al. (2018). The enhanced antioxidant enzymes activity may contribute to maize seedlings' freezing resistance, particularly in the roots. Stevens et al. (2006) discovered that the effective dose of SA for reducing water stress injury in tomato and bean plants was between 0.1 and 0.5 mM. At low quantities, SA is safe for the plant. As a result of the high activity of antioxidant enzymes caused by low SA concentrations, plants become resistant. However, high levels of SA in the plant due to toxicity result in inadequate antioxidant enzyme activity (War et al., 2011).

7. FUTURE DIRECTIONS OF SA

Despite extensive research into the route of action and potential applications of SA, many topics remain unanswered. In addition, the current "Special Issue" has six research pieces. These studies discuss a wide spectrum of mechanisms of SA in plant defense. They include a "traditional" demonstration of SA's role in biotic stress (Shi et al., 2019), as well as the importance of using various mutations linked to SA (Pluhařová et al., 2019; Tajti et al., 2019) and an emphasis on interaction with other chemicals linked to stress (Tajti et al., 2019). There are also studies looking at the influence of environmental factors on SA signaling (Cappellari et al., 2020; Pál et al., 2020), as well as the role of several SA analog chemicals (Palmer et al., 2019). SA's role in biotic stress-related mechanisms has received a lot of attention. Shi et al. (2019) were the first to show that SA is involved in biosynthesis genes and molecular functions in tea plants' anthracnose disease response. Colletotrichum fungi produce this illness, which can result in a 5–20 percent reduction of tea output. According to the findings, SA and its accompanying signaling links, specifically the production of pathogenesis-related protein 1 (PR1) and links with other plant hormones, are thought to induce tea immunity to anthracnose disease activation. They also provided a transcriptome dataset for analyzing gene

expression and metabolic networks in tea plants connected to anthracnose resistance (Shi et al., 2019).

The fact that over forty Arabidopsis mutants or transgenic lines have been described with altered amounts of SA signaling pathways demonstrates the relevance of SA signaling. Pluharová et al. (2019) mutant collection is a highly useful tool for better understanding the mechanisms underlying plant growth-defense trade-offs. They propose a novel study that sheds fresh light on the relationship between SA and plant behavior under stress (Pluhařová et al., 2019). There was a negative correlation between SA concentration and rosette size, but not root growth, according to the researchers. This is particularly essential since SA applied hydroponically frequently inhibits root growth more than shoot growth. When comparing data collected under diverse growth conditions, their findings also highlight the critical functions of light intensity. Signaling in South Africa is not a straightforward process. SA has the potential to interact with several different stress-related chemicals. Polyamines have long been recognized as important components of living cells.

Their signaling significance, on the other hand, has just recently been clear (Pál et al., 2020). Tajti et al. (2019) recently revealed that the SA and polyamine signaling pathways may interact. They discovered that SID2 plants, SA-deficit Arabidopsis mutant, have a unique polyamine metabolism. Exogenous polyamine treatments elicited different responses in the SID2 mutant plants than in the naturally occurred plants. Significant differences in SA content and production were identified between wild-type and SA-deficit mutant Arabidopsis plants after polyamine treatments. Abiotic and biotic environmental factors both have an impact on SA signaling. It has been demonstrated that inoculating Mentha x piperita plants with different Rhizobacteria strains promotes endogenous SA synthesis (del Rosario Cappellari et al., 2019). Cappellari et al. (2020) discovered that inoculating M. piperita with plant-growth-promoting Rhizobacteria could change the effects of exogenous SA or methyl jasmonate therapy. Exogenous SA increased the overall phenol content of this plant, and depending on the dose used, particular Rhizobacteria could boost this effect. The synthesis of the major monoterpene compounds was similarly altered by exogenous SA (Cappellari et al., 2020). These findings show that combining salicylic acid with plant growth-promoting Rhizobacteria could help aromatic plants produce more secondary metabolites.

8. CONCLUSION

SA is a signaling molecule that can alter various plant functions under normal and stressful situations. The way it operates is determined by several factors, including the environment, plant type, and SA concentration. At low doses, it serves as a mediator, affecting the plant's oxidative state and decreasing reactive oxygen species by increasing antioxidant enzymes and preventing the plant from biotic stress. As a result of SA's antioxidant properties, it's possible that this molecule could be employed to make plants resistant to pathogen infections.

9. ACKNOWLEDGEMENTS

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