



## SALICYLIC ACID DRIVEN DEFENCE: ENHANCING RICE RESISTANCE TO BACTERIAL BLIGHT

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Rice (*Oryza sativa* L.) is the most important staple food for more than two-third of the world population providing more than 20 percent of the calorie consumed worldwide. The bacterial blight disease caused by *Xanthomonas oryzae* pv. *oryzae* is considered as one of the major diseases of rice across the globe which causes large production losses. Salicylic acid is a possible treatment for illness management strategy. It has been demonstrated that salicylic acid is essential for fostering rice resistance to bacterial blight. Recent studies indicate that application of salicylic acid to rice plants can stimulate their defence systems and boost their resistance to *X. oryzae* pv. *oryzae*. One of these defence strategies is the overexpression of genes linked to disease. Salicylic acid is a potent disease management technique for rice that successfully fights bacterial blight by making rice plants resistant to the infection and triggering defence mechanisms against it. By increasing defence genes and triggering defence mechanisms in rice plants, salicylic acid creates resistance, shielding the plants from infection and lessening the intensity of bacterial blight outbreaks. The present study mainly focuses on the multifaceted role of salicylic acid in the management of bacterial blight disease in rice.

**Keywords :** Rice, bacterial blight, *Xanthomonas oryzae* pv. *oryzae*, salicylic acid, defense mechanisms.

### ABSTRACT

### Introduction

Rice (*Oryza sativa* L.) is a staple cereal for more than 3.5 billion people, providing over 20% of daily calorie intake. Though low in protein, rice has one of the highest nutritional quality ratings among cereals. Globally, it supplies 15% of protein and 21% of energy consumed by humans (Rao *et al.* 2017). In India, rice is cultivated under diverse altitudes and climates across almost all states, with West Bengal, Uttar Pradesh, Madhya Pradesh, Bihar, Odisha, Andhra Pradesh, Assam, Tamil Nadu, Kerala, Punjab, Maharashtra, and Karnataka contributing 92% of total production. In 2021–22, rice production reached a record 127.93 million tons, 11.49 million tons higher than the last five-year average (Anonymous, 2022).

There are about 50 different biotic factors that can cause potential yield loss in rice including fungi,

bacteria, viruses, nematodes and insect pests. Amongst the diseases that causes significant yield loss in rice, bacterial blight (BB) is reported to be one of the most devastating bacterial diseases. Yield loss due to BB disease can reach as high as 50% depending on the rice variety, growth stage, the geographic location and environmental conditions (Liu *et al.* 2018). Losses resulting from the BB kresiek syndrome can amount to up to 75%. BB is caused by pathogenic bacteria namely *Xanthomonas oryzae* pv. *oryzae* (*Xoo*). It is a gram-negative bacterium with a size of around 0.55 to 0.75 x 1.35 to 2.17  $\mu\text{m}$ . Colonies are light yellow, circular, convex, and smooth. The bacterium is systemic in the xylem of the rice host. A type III protein secretion system exists in this bacterium to directly inject virulence factors into the host. The typical symptoms of the disease include initial small water-soaked lesions on leaf surface which gradually

transitions into a yellowish-white coloration and exhibit a characteristic expansion pattern, typically symmetric along both lateral margins. Lesion margins interface directly with healthy tissue and are marked by a distinct undulate or wavy outline, which serves as a diagnostic feature of the disease and is prominently visible on the lamina. In several instances, bacterial ooze resembling milky dew drop are observed early in the morning on young lesions (Fig.1 a and b). So far, over 30 races of *Xoo* are reported to occur. *Xoo* survives primarily in rice stubble and on weed hosts, notably *Leersia oryzoides*, *Zizania latifolia*, *Leptochloa chinensis*, *L. panacea*, *Cyperus rotundus* and so on. In tropical areas, the bacterium may also survive in irrigation water. The pathogen enters the rice leaf typically through the hydathodes at the leaf tip and leaf margin. Bacterial cells on the leaf surface enter the plant through guttation fluid, multiply in epithelial cells, and spread through the xylem. *Xoo*, is reported to access the xylem through wounds or openings (Jiang *et al.*, 2020).



**Fig. 1 (a) :** Bacterial blight infected rice plants,   
**(b)** Bacterial ooze resembling milky-yellow dew drops on young lesions

#### Plant innate immunity

Being sessile, plants go through a variety of physiological and metabolic transformations to survive in the ever-changing natural world. In the past twenty years, a plethora of genetic and molecular research on the interactions between microbes and plants in several model systems has emphasise that plants have evolved two intricate immune defence mechanisms to ward off pathogen attacks: effector-triggered immunity (ETI) and pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI) (Li *et al.*, 2022). Using their cell surface receptors, plants identify PAMPs and trigger PTI, which in turn triggers the appropriate defence mechanism or pathways and ultimately prevents pathogen invasion and infection. Conversely, ETI is essentially an intrinsic response that results from a specific interaction between pathogen-effector

proteins and the host's nucleotide-binding domain, sometimes known as leucine-rich repeat-containing receptors, or NLRs (Hurley *et al.*, 2014). In this regard, phytohormones play a significant role by establishing a connection between host-pathogen recognition and subsequent cellular responses for activating the defence pathways (Kamiya, 2010).

#### Pseudo Response Regulator (PRR) and Pathogen Triggered Immunity (PTI)

Plant PRRs are cell-surface receptors that sense PAMPs produced into the extracellular environment by invading pathogens. PAMP detection by PRRs triggers PTI reactions. Plant PRRs are represented by receptor-like proteins (RLPs), which do not have a kinase domain, and transmembrane receptor-like kinases (RLKs), which usually include external leucine-rich repeats and an intracellular kinase domain. RLPs attract proteins with kinase domains to activate the downstream signalling pathways because they lack a cytoplasmic kinase domain. In the rice genome, more than 1131 RLK genes have been found. There are 90 RLP genes encoded in the rice genome, making RLPs a second important class of cell-surface receptors in plants. All of these receptor classes react to a broad range of external stimuli, including infections and host-derived endogenous danger signals, which activate receptors (lipid, protein, nucleic acids, carbohydrate, etc.). Research has consistently demonstrated that rice cells are capable of sensing conserved PAMPs, including bacterial flagellin, peptidoglycan, lipopolysaccharide, and fungal chitin, which may then initiate innate immunity. A number of PRR proteins in rice have been well characterised, including XA21, OsFLS2, CEBiP, OsCERK1, LYP4, and LYP6. One of the earliest innate immune receptor genes to be identified was the rice RLK gene Xa21, which offers resistance against a variety of *Xoo* strains. Numerous prior investigations have discovered multiple *Xoo* Rax genes that trigger the XA21-mediated immune response (which is necessary for Xa21 activation) Tyrosine sulfotransferase (RaxST) and three parts (RaxA, RaxB, and RaxC) of a type 1 secretion system are located in a single operon (raxSTAB) that contains these genes. The type 1-secreted protein that is tyrosine-sulfated triggers the XA21-mediated immune response. This theory is supported by the discovery that the induction of XA21-mediated resistance requires a sulfated, 21-amino acid (AA) synthetic peptide (RaxX21-sY) that is generated from the RaxX protein released by *Xoo* (Pruitt *et al.* 2015).

## R gene, ETI and Interaction

Leucine-rich repeat domain (NLR) and nucleotide-binding proteins are widely established to serve as immunological receptors in plants. It has been estimated that roughly 480 NLR genes are present in the rice genome (Le *et al.*, 2017). In plant genomes, NLR genes are typically grouped together in clusters; of the 480 NLR genes in rice, for instance, 263 are found in 44 different clusters (Read *et al.*, 2020). One of these NLR gene clusters on chromosome 6 contains the rice R genes *Pi2*, *Pi9*, and *Piz-t*. In both wild and cultivated rice, at least eight R genes are found at this locus. The transcription activator-like effector (TALE)-dependent resistance to bacterial blight in rice is conferred by both executor R proteins, XA10 and Xa23 (Wang *et al.* 2015). In the endoplasmic reticulum (ER), the XA10 protein localises as hexamers, and this localization is associated with both ER  $\text{Ca}^{2+}$  depletion and XA10-induced cell death in plants. These findings imply that XA10 is an inducible protein that, through a conserved mechanism involving ER disruption and cellular  $\text{Ca}^{2+}$  homeostasis, initiates programmed cell death. The projected transmembrane helices structure of the Xa23 and XA10 executor R proteins is comparable, and they share 50% identity (Tian *et al.*, 2014). TALE AvrXa23 is the particular activator of Xa23 transcription, and XA23 can elicit a potent immunological response in rice (Read *et al.* 2020). A TALE-binding element found in the promoters of *Xa10* and *Xa23* is necessary for cognate TALE-induced resistance (Wang *et al.*, 2015). An executor R gene family that has developed in the rice genome recognises the corresponding TALEs in *Xoo* to aid in disease resistance. Over the past 20 years, numerous genetic researches on rice resistance to BB have been carried out. More than 40 resistance (R) genes have been found to date, and 11 of them *Xa1*, *Xa3/Xa26*, *Xa4*, *Xa5*, *Xa10*, *Xa13*, *Xa21*, *Xa23*, *Xa25*, *Xa27*, and *Xa41* have been cloned. These genes confer host resistance to different strains of *Xoo*. Based on the proteins that these R genes encode, they can be divided into four groups: genes that encode receptor-like kinase (RLK) (*Xa21*, *Xa3/Xa26*, and *Xa4*); genes that encode sugar transporter (SWEET) (*xa13*, *xa25*, and *xa41*); executor genes (*Xa10*, *Xa23*, and *Xa27*); and other genes (*Xa1* and *xa5*) (Nino-Liu *et al.*, 2006). The *xa5* gene in rice encodes the  $\gamma$  subunit of transcription factor IIA (*TFIIA $\gamma$ 5*), playing a crucial role in conferring resistance to bacterial blight caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*). Unlike many resistance genes, *xa5* is recessive, meaning that resistance is exhibited when two copies of the allele are present. The resistance mechanism involves a mutation

in the *TFIIA $\gamma$ 5* subunit, which interferes with the pathogen's transcription activator-like effectors (TALEs). These TALEs typically bind to specific DNA sequences in the host to activate susceptibility (*S*) genes that facilitate infection. The mutation in *xa5* disrupts this interaction, thereby reducing the expression of *S* genes and enhancing resistance. Research has shown that the effectiveness of *xa5* is dependent on major TALE genes in *Xoo* and correlates with reduced expression of the corresponding *S* genes. For instance, the presence of *xa5* can diminish the activity of TALEs that target *S* genes like *OsSWEET14* and *OsSWEET13*, which are involved in sucrose transport and are exploited by the pathogen to access nutrients. However, *xa5* may be less effective against strains of *Xoo* that possess TALEs capable of robustly activating other *S* genes, such as *OsSWEET11* (Yuan *et al.*, 2016).

## Hormone-mediated immunity in rice

It has been discovered that oxidative status is crucial in protecting from numerous biotic and abiotic stressors. Several signalling pathways that result in the synthesis of several defensive proteins and non-protein substances mediate plant defence against stresses. Plant phytohormones like salicylic acid (SA), ethylene, jasmonic acid, and abscisic acid (ABA) are crucial parts of several signalling cascades related to plant defence. JA mediates the octadecanoid route and SA mediates the phenylpropanoid pathway. While JA and ET are thought to be involved in resistance to necrotrophic which degrade plant tissue during infection, and insect pests, SA signalling positively regulates plant defence against biotrophic pathogens, which require living tissue to complete their life cycle (War *et al.* 2011). Antioxidative enzyme activity is one of the physiological, biochemical, and molecular processes that are manipulated in plants by the exogenous application of SA and jasmonic acid. In addition, SA not only interacts with other signalling pathways that mediate plant resistance, but it also controls the elements of its own signalling pathway. It has been suggested that SA influences nutrient intake, water relations, stomatal control, and photosynthesis in plants under stress. The primary elements of induced plant defence against biotic and abiotic challenges are peroxidase (POD), polyphenol oxidase (PPO), superoxide dismutase (SOD), phenylalanine ammonia lyase (PAL), and others. It controls the activity of these enzymes. PODs are a significant class of defence enzymes that protect plants against a range of environmental challenges (Wang *et al.* 2021).

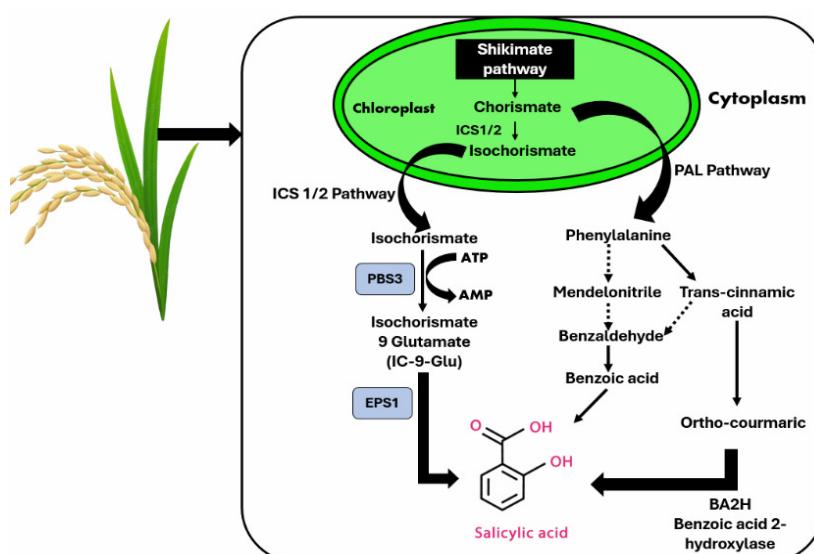
### Salicylic acid (SA) - an elicitor for plant disease response

Plant hormone salicylic acid (SA) is involved in the morphological, physiological, and biochemical processes that trigger plant defence against a range of biotic and abiotic stressors (War *et al.*, 2011). SA is produced as a secondary metabolite, chemically known as 2-hydroxybenzoic acid, is a plant hormone involved in various aspects of plant growth, development, and defence (Ding and Ding, 2020). Isomochorismate synthase and phenylalanine ammonia-lyase-dependent pathways are the two different pathways that biosynthesize SA from chorismate (Dempsey and Klessig, 2017). Phenylalanine ammonia-lyase (PAL) in the phenylpropanoid biosynthesis pathway changes Phe into trans-cinnamic acid, which acts as a substitute precursor for SA (Bagal *et al.*, 2012). SA serves as a signaling molecule in plant defence responses, activating defence genes and pathways to combat pathogens such as bacteria, fungi, and viruses. SA induces SAR, where a localized response to pathogen attack primes the entire plant for enhanced defence against subsequent infections, even in distant tissues. By affecting physio-biochemical processes like stress regulation and hormonal crosstalk during seed germination, cell growth, preserving redox homeostasis and rubisco activity during photosynthesis, flowering, senescence, stomatal regulation, crop yield, and plant response to abiotic stresses like drought, thermo-tolerance, osmotic stress, and heavy metal toxicity, it plays a critical role in the development and maturation of plants (Liu *et al.*, 2022). According to a number of research studies conducted thus far, SA plays a crucial,

pathogen-type-specific, connected, and overlapping function in plant immunity and defence (Mishra *et al.*, 2024). The primary cause of triggering defence responses against biotrophic and hemibiotrophic infections is salicylic acid (Ullah *et al.*, 2023). The invasion of pathogens activates pathways in plant tissue that lead to the production of SA. Following biosynthesis, most of the SA is modified through processes such as sulfonation, hydroxylation, methylation-demethylation, glucosylation, and amino acid conjugation. However, the majority of these conjugated SA forms are biologically inactive (Qiu *et al.*, 2022). SA-mediated signalling pathways may function independently, cooperatively, or antagonistically, depending on the type of invading pathogen (Li *et al.*, 2022). From this perspective, the interaction between SA and other plant hormones such as ethylene (ET), jasmonic acid (JA), and abscisic acid (ABA) results in complex crosstalk that is often dose-, species-, tissue-, and inducer-specific (Wang *et al.*, 2021)

### SA Biosynthesis

In order for plants to produce SAR and initiate an immunological response, SA must first accumulate inside the plant. On the other hand, their indiscriminate accumulation could also be detrimental to plant fitness. As a result, there is strict control over the genes involved in SA production and its metabolic pathways (Wang *et al.* 2017). Plants possess two primary routes for the biosynthesis of SA: the isochorismate pathway and the phenylpropanoid pathway (Fig. 2).



**Fig. 2 :** Salicylic Acid (SA) Biosynthesis Pathways in Rice

These processes rely on chorismate, which is obtained via the shikimate system. The isochorismate and phenylpropanoid pathways, respectively, depend on the enzymes isochorismate synthase (ICS) and phenylalanine ammonia lyase (PAL) (Chen *et al.* 2021). Around 90 percent of pathogen-induced SA accumulation is explained by the isochorismate pathway, as shown by the practically complete lack of pathogen-induced SA accumulation in *Arabidopsis* mutants deficient in ICS1 (Wu *et al.* 2023). Kim and Lim. (2023) report that enhanced disease susceptibility (EDS5) facilitates the transfer of synthesised SA or its precursors from plastid to cytosol, hence acting downstream to the isochorismate pathway. Another protein called amido transferase *avrPphB* susceptible (PBS3) converts glutamate to isochorismate in the cytosol, which then breaks down into 2-hydroxy-acryloyl-N-glutamate and (Kabir and Das, 2021).

### SA-responsive genes and proteins: Mode of Action

In the host plants, SA biosynthesis is regulated by a group of regulatory genes like non expressor of pathogenesis-related genes 1(NPR1) and (NPR3/NPR4) Both, NPR1 and NPR3/NPR4 act antagonistically to each other. At lower SA concentrations, NPR1 acts as a transcription activator and induces resistance (R) gene's expression upon detecting the presence of a pathogen or its invasion (Dalio *et al.*, 2021). NPR1 has a core ankyrin repeat, a C-terminal transcriptional activation motif, and an N-terminal BTB/POZ domain. When plants perceive pathogen invasion, they elevate their SA levels. SA acts as a signalling molecule that triggers the oligomerization and subsequent nuclear translocation of NPR1. Increasing evidence suggests that NPR1 serves as an SA receptor. In the nucleus, NPR1 interacts with transcription factors, such as TGA factors, initiating transcriptional reprogramming. This reprogramming leads to the upregulation of defence-related genes, including pathogenesis-related (PR) genes. Among them, TGA2, TGA5, and TGA6 have redundant roles in disease resistance and SA-induced Pathogenesis-related (PR) gene expression. These PR proteins play diverse roles in plant defence, such as antimicrobial activity, reinforcement of cell walls, and signalling. Thus, the SA-NPR1 pathway serves as a central regulator in orchestrating the plant's defence response to pathogen attack. Likewise, NPR3 and NPR4 redundantly act as co-repressors of defence-responsive gene expression. Elevated SA level results in hypersensitive response (HR)-mediated programmed cell death (Radojičić *et al.*, 2018). The studies have revealed that not only the endogenous SA regulate the plant immune system, but their exogenous application

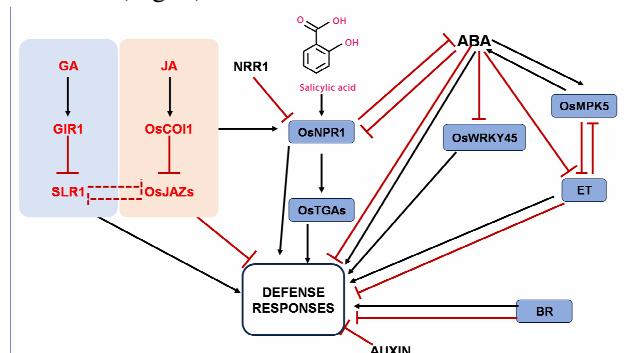
also enhances plant disease resistance against fungal, bacterial, and viral pathogens and promotes plant growth and development through modulating immune-related genes at transcriptional and translational levels (Singh *et al.*, 2023). In this context, the present review focuses on SA-mediated immune response in plants against invading pathogens and discusses its different molecular components, interactions, and cross-talk with other associated cellular components of the signalling cascade responsible for resistance to pathogens. EDS1 is a key player in plant SA-mediated immunity, acting as a signalling node that connects various defence pathways. It enhances the plant's ability to develop SAR and combat various pathogens. EDS1 acts as a transcriptional coactivator, promoting PR1 production under the presence of active NPR1 and SA. It works in collaboration with other proteins, including Phytoalexin Deficient 4 (PAD4), to create a complex that aids in signal transduction in response to pathogen-derived signals. The EDS1-PAD4 complex modulates NPR1 activity, promoting the transcription of defence-related genes and the synthesis of antimicrobial compounds. The interaction with NPR1 facilitates the recruitment of CDK8, a transcriptional coactivator, to the Mediator complex, a large protein complex that regulates immune-responsive genes (Khan *et al.*, 2022). Both NPR1 and EDS1 maintain their expression during pathogenic infection at the protein level. The EDS1/PAD4 complex promotes ICS1 expression and contributes to SA accumulation through a positive feedback loop. EDS1/PAD4 is an alternative route for defence against pathogens or genetic perturbations, upholding SA-related defence mechanisms and enhancing the strength of the innate immune system (Chen *et al.*, 2021). The PR proteins function in both systemic and local defence processes and are the building blocks of a plant's innate immune system. One of the main ways that SA affects the immune response to pathogens at the translational level is through the induction of PR proteins (Khan *et al.*, 2022). By supporting the activity of translation initiation factors or bolstering the stability of PR protein messenger RNAs, the SA can increase the translation of PR proteins (Shirokikh, 2022). They affect the translation of other proteins involved in the expression of PR genes in addition to directly regulating PR proteins. In the salicylic acid signalling pathway, several key enzymes and pathogenesis-related (PR) proteins play crucial roles in mediating defence responses against pathogens. Upon perception of pathogen invasion, plants activate SA biosynthesis pathways, involving enzymes such as phenylalanine ammonia-lyase (PAL), isochorismate synthase (ICS), and salicylate hydroxylase (NahG). PAL catalyzes the

conversion of phenylalanine to cinnamic acid, initiating the phenylpropanoid pathway leading to SA production. ICS mediates the conversion of chorismate to isochorismate, a precursor of SA biosynthesis. Conversely, NahG hydroxylates SA to form catechol, reducing SA levels. Following SA accumulation, defence responses are triggered, leading to the induction of PR proteins. These PR proteins, encoded by PR genes, encompass various classes such as PR-1, PR-2 ( $\beta$ -1,3-glucanases), PR-3 (chitinases), PR-4 (thaumatin-like proteins), and PR-5 (thaumatin-like proteins). PR proteins exhibit antimicrobial activities by degrading fungal cell walls (PR-2 and PR-3), interfering with pathogen cell membrane integrity (PR-4), or exerting direct toxicity against pathogens (PR-5). Moreover, PR proteins contribute to reinforcing plant cell walls, eliciting systemic acquired resistance (SAR), and modulating defence signal transduction pathways (Han and Schneiter, 2024). Since it initiates HR at the infection site and interacts with defence response-related TFs (NAC and WRKY), ROS is essential to plant defence responses against pathogen invasion and other stressors. By modifying the activity of antioxidant enzymes, salicylate can control the amount of reactive oxygen species (ROS) in cells, preserving the delicate balance between defence and oxidative damage (Mishra *et al.*, 2024).

### Interaction of SA with other phytohormones

Resistance to necrotrophic infections is not affected by the SA-mediated defence system, and it may even be negatively impacted. As a result, defence signalling mediated by JA and ET is known to be antagonistic to SA-dependent signalling. However, plant-pathogen interaction systems control the signalling pathways that connect SA and ET/JA and are not exclusively antagonistic. For example, it was shown that Arabidopsis mutants lacking in SA, which are expected to be competent in the JA defence response, are vulnerable to *Pythium irregularare*, a soil-borne necrotrophic disease. Furthermore, introducing low concentrations of SA and JA to tobacco plants resulted in a transient upregulation of gene expression associated with SA (PR1 [ $\beta$ -glucuronidase in tobacco]) and JA (defensin and thionin) signalling. This shows that these phytohormones operate in symphony. They are dependent on NPR1 and Coronatine Insensitive 1 (COI1), two signalling molecules linked to the JA and SA pathways, respectively. Nevertheless, the administration of high doses of SA and JA causes these genes to express antagonistically. The antagonistic relationship between the JA- and SA-pathways has received a lot of attention (Xiang *et al.*, 2020). SA appears to

significantly impede the JA signalling pathway in *A. thaliana*, which results in the repression of several genes linked to JA production (LOX2, AOS, AOC2, and OPR3) as well as downstream response genes including PDF1.2 and VSP2 (Leon-Reyes *et al.*, 2010). A common component of the signalling pathways mediated by JA and SA is found to be WRKY TFs. Resistance to the fungal disease *Erysiphe cichoracearum* is increased when WRKY70 expression is upregulated (mediated by SA), while it is weakened when it comes to *Alternaria brassicicola* (mediated by JA). On the other hand, the SA-mediated reactions are also suppressed by the activation of the ET/JA pathway. Higher levels of SA and enhanced resistance to the biotrophic pathogen *P. syringae* are the results of deleting the JA receptor COI1 and JA-responsive MYC. Also, NPR1 is a necessary conjugator for SA-ET/JA crosstalk, either directly or indirectly by transcriptionally activating the glutaredoxin and/or WRKY TF genes. Arabidopsis npr1 mutant plants that lacked the antagonistic relationship between SA on JA signalling demonstrate the importance of NPR1 as a mediator (Fig. 3).



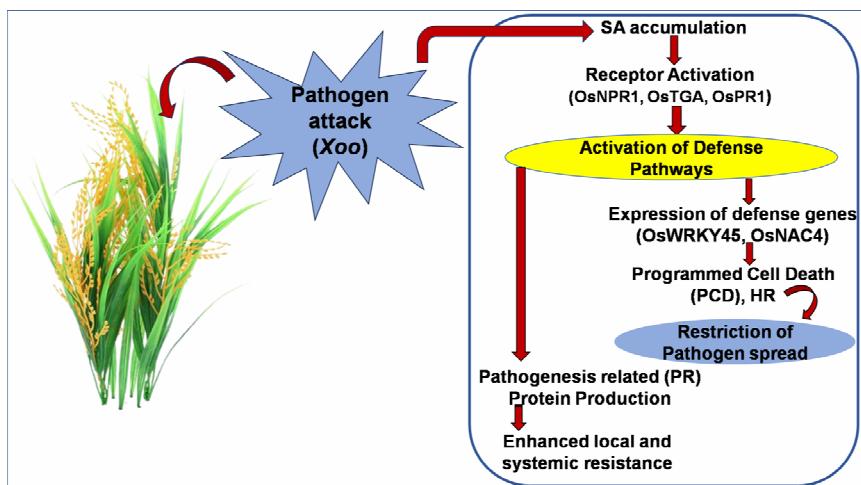
**Fig. 3 :** Hormonal Crosstalk in Rice Immune Response

### SA-Mediated Resistance in Rice

The defence responses of rice plants against BLB after treatment with exogenous SA and challenge inoculation with *Xoo* by monitoring biochemical changes associated with plant defence mechanisms was studied. The disease severities of rice plants treated with 1 mM SA were 26.19% and 38.57% at 7 and 14 DAI, respectively (Le *et al.*, 2017). SA has been identified as a key player in disease resistance in rice plants (Fig. 4). SA is synthesized in rice from cinnamic acid via benzoic acid, similar to tobacco. Brassinolide (BL), a brassinosteroid, induced disease resistance in rice against rice blast and bacterial blight diseases. Similarly, the Xa5 gene in rice, which encodes a novel form of disease resistance was identified. OsNPR1 is the rice orthologue conferring disease resistance through SA-mediated systemic acquired resistance. OsWRKY13 plays a pivotal role in rice disease

resistance by regulating defence-related genes in SA- and jasmonate-dependent signalling pathways. OsEDR1 adversely influence rice bacterial resistance by inducing ethylene biosynthesis (Shen *et al.*, 2011). The synergistic relationship between SA and cytokinin signalling in the generation of disease-resistant rice employing regulatory components of induced disease resistance. OsWRKY67 was a positive regulator of rice resistance to bacterial blight and blast. The possibility

of bioengineering and molecular modification of the SA signalling pathway to induce plant immune responses for agricultural disease control was brought to light by (Vidhyasankar, 2020). OsNPR3.3-dependent SA signalling is important in Xa5-mediated immunity to rice bacterial blight highlights the significance of SA in rice plant disease resistance (Jiang *et al.*, 2020).



**Fig. 4 :** Salicylic Acid (SA) mediated defense mechanism in rice plant

## Conclusion

SA has demonstrated its well-established function in biotic stress in addition to its adaptability in plant development, growth, and responses to abiotic stresses. Although endogenous SA synthesis is closely associated with pathogen invasion and disease resistance, it is also significantly regulated by intricate molecular networks. An environmentally friendly method of comprehending plant-pathogen interactions and manipulating strategies to enhance resistance is through SA-mediated plant immunity. The strong genetic control over SA accumulation and production in plant cells can be used to enhance SA-mediated immunity. SA-associated molecular players have been shown to be implicated in defence signalling for broad-spectrum and non-host resistance in plants. These players include genes (like NPR) and proteins (including TGA, SABP, and PR). Rice plants can develop disease resistance to the bacterial blight (BB) disease by applying salicylic acid (SA), a plant growth regulator. Applying SA to rice plants can lessen the severity of leaf blight (LB) disease by 38%. SA can also be used as an elicitor against LB disease. Without negatively impacting plant yield, priming rice plants with SA can also regulate the activity of antioxidant enzymes including Photosystem II.

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