Journal of Plant Growth Regulation
https://doi.org/10.1007/s00344-024-11264-4

Jasmonic Acid (JA) in Plant Immune Response: Unravelling Complex Molecular Mechanisms and Networking of Defence Signalling Against Pathogens

Rajib Roychowdhury[1] · Alkesh Hada[2] · Sabarni Biswas[3] · Sapna Mishra[1] · Manas Ranjan Prusty[4] · Soumya Prakash Das[5] · Shatrupa Ray[1] · Ajay Kumar[6] · Umakanta Sarker[7]

Received: 1 December 2023 / Accepted: 1 February 2024

Abstract

Jasmonic acid (JA) and its derived compound 'jasmonates' are a class of potent phytohormones that play pivotal roles in plants' physio-biochemical processes during growth and development. With the onset of the 21st century, concurrent climate changes all over the world have drastically affected plants' adaptability and survivability against (a)biotic stressors. Such climatic instabilities trigger a variety of pathogens (mainly fungi, bacteria, and viruses) to create disease pressure by affecting plant health and the immune barrier. JA is crucial for plant immune response through their biosynthetic pathways, involvement in signalling cascades and corresponding cross-talk with other phytohormones like salicylic acid (SA), ethylene (ET) and abscisic acid (ABA). In this perspective, the endogenous role of JA lies behind the process of cellular central dogma at the molecular level. The genes and gene products, like transcription factors (TFs) associated with JA biosynthesis and signalling, impart some sort of regulation on plant immune response by activating systemic and localized signalling, pathogenesis-related (PR) genes and proteins, phytoalexins, modulating pathogen effectors, and interacting with host proteins. In addition, the external application of JA can also significantly lead to stimulating the immune circuit of plants through gene expression and protein interaction. In this way, JA can boost a plant's immune system through systemic acquired resistance (SAR) and induced systemic resistance (ISR). This could be an idea to understand the hormonal interplay in plant immune response to various types of pathogens and provide the resistance mechanism against yield and quality losses.

Keywords Crop improvement · Disease resistance · Jasmonic acid · Pathogens · Plant immunity · Signalling

Abbreviations

ABA	Abscisic acid
DAMP	Damage-associated molecular patterns
ET	Ethylene
ETI	Effector-triggered immunity
HAMP	Herbivore-associated molecular patterns
ISR	Induced systemic resistance
JA	Jasmonic acid
JA-Ile	Jasmonyl isoleucine
JAZ	Jasmonate-zim domain
MAMP	Microbe-associated molecular patterns
MeJA	Methyl jasmonate

Handling Editor: M.Iqbal R. Khan.

✉ Rajib Roychowdhury
rajbroychowdhury86@yahoo.com

[1] Department of Plant Pathology and Weed Research, Institute of Plant Protection, Agricultural Research Organization (ARO)-The Volcani Center, 7505101 Rishon LeZiyyon, Israel

[2] Deptment of Entomology and Units of Nematology and Chemistry, Institute of Plant Protection, Agricultural Research Organization (ARO)-The Volcani Center, 7505101 Rishon LeZiyyon, Israel

[3] Department of Botany, Sonarpur Mahavidyalaya, Rajpur, Kolkata, West Bengal 700149, India

[4] Institute for Cereal Crop Improvement, Department of Plant Sciences, Tel Aviv University, 6997801 Tel Aviv, Israel

[5] School of Bioscience, Seacom Skills University, Bolpur, West Bengal 731236, India

[6] Amity Institute of Biotechnology, Amity University, Noida 201313, India

[7] Department of Genetics and Plant Breeding, Faculty of Agriculture, Bangabandhu Sheikh Mujibur Rahman Agricultural University, Gazipur 1706, Bangladesh

Published online: 09 May 2024

PAMP	Pathogen-associated molecular pattern
PRR	Pattern recognition receptors
PTI	PAMP triggered immunity
SA	Salicylic acid
SAR	Systemic acquired resistance

Jasmonic Acid (JA) as an Elicitor for Plants' Defence Response Against Pathogens: An Introduction

Plants, being immobile organisms, encounter various physiological and metabolic adaptations to endure the dynamic natural environment and potential diseases through the invasion of pathogens, such as fungi, bacteria, viruses, viroids, and so forth (Roychowdhury et al. 2020). The continuous phenomenon of global climate change exerts a detrimental impact on plant life, particularly important crops, by increasing the chance of disease pressure, ultimately resulting in crop loss (Chakraborty et al. 2014; Roychowdhury 2014). Such pathogens pose a significant threat to plants, causing a range of detrimental effects that can impede their growth, reduce yield, and even lead to the demise of entire crops. Plant pathogens often penetrate plant tissues, disrupting cellular functions and nutrient transport mechanisms, interfering with crucial biological workflows, such as photosynthesis and water uptake, and compromising the plant's potential to thrive (Selvaraj and Fofana 2012). Additionally, pathogens can produce toxins that further harm plant tissues and trigger defensive responses, leading to visible symptoms like wilting, discolouration, lesions, and stunted growth. The economic and ecological consequences of plant diseases are profound, affecting agricultural productivity, food security, and the delicate balance of ecosystems. In particular, the recent COVID pandemic has served as a valuable lesson, highlighting the significance of all pathogens, and emphasizing the need to not underestimate their potential impact on mankind. In the course of co-evolutionary process, plants developed immunity to cope with biotic stresses induced by pathogens. When a pathogen is encountered, the plant activates its immunity on multiple levels which helps to prevent pathogen invasion in host plants. Pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI) is a general defence mechanism that starts when PAMPs are recognised by pattern recognition receptors (PRRs) of host plants. The effector-triggered immunity (ETI), in contrast, is a more focused defence response that is triggered by the activation of host resistance genes that detect and counteract the effectors released by pathogens, hence inhibiting PTI (Jones and Dangl 2006; Li et al. 2023).

Plant hormones have a significant crucial role in creating a connection between host–pathogen identification, their interaction, and subsequent cellular responses, hence activating defence mechanisms (Kamiya 2010). Jasmonic acid (JA) and its derivatives, including jasmonyl isoleucine (JA-Ile), methyl jasmonate (MeJA), 12-hydroxyjasmonic acid sulphate (12-HSO_4-JA), *cis*-jasmone, JA-glucosyl ester, JA-Ile methyl ester, jasmonoyl-amino acid, 12-carboxy-JA-IIe, 12-*O*-glucosyl-JA-Ile, 12-O-glucosyl-JA, JA-Ile glucosyl ester, are collectively referred to as jasmonates (Ruan et al. 2019; Ghorbel et al. 2021; Wang et al. 2021). In higher plants, JA functions as a stress-responsive phytohormone and is involved in the control of various physiological processes. Development and growth of a plant are controlled by JA in a variety of ways, including embryogenic elongation of the axis, root formation, opening of stomata, leaf senescence, flower development, uptake of phosphate and nitrogen, transportation of organic matter, production of Rubisco. (Huang et al. 2017; Ghorbel et al. 2021). JA is widely present in higher plants and has recently drawn significant interest in the realm of plant stress responses to pathogens and defence mechanisms (Kachroo and Kachroo 2012; Wang et al. 2021).

In general, JA mostly activates plant defence responses against necrotrophic pathogens and herbivores, and amenable for the induction of systemic resistance (ISR; Pandey et al. 2016). The JA mediates plant defence mainly against—(a) insects, such as leaf-eating insects (e.g. beetles, caterpillars), phloem-feeding insects (whiteflies and aphids), herbivorous insects that feed on soft plant tissues, such as leaf miners, as well as insects that suck and pierce plant tissues, such as leaf hoppers, spider mites, thrips, mirid bugs, and fungus gnats (Denno and Kaplan 2007; Sugio et al. 2015; Zhang et al. 2017a; Singh et al. 2021); (b) pathogenic bacteria (e.g. *Plectobacterium atrosepticum*) and fungi (e.g. *Alternaria brassicicola*, *Fusarium oxysporum*, *Plectosphaerella cucumerina*, and *Botrytis cinerea*) (Antico et al. 2012; Zhang et al. 2017a; He et al. 2018; Hou et al. 2019); (c) detritivores crustaceans (Farmer and Dubugnon 2009) and mollusc (Falk et al. 2014; Orrock et al. 2018); and (d) vertebrate herbivores (Machado et al. 2016; Baldwin 2018). Herbivores/pathogens attacks generate various kinds of PAMPs/microbe-associated molecular patterns (MAMPs), such as systemin/systemin-like peptides, elicitors, plant cell-derived oligogalacturonides, flagellin, herbivore-associated molecular patterns (HAMPs), and damage-associated molecular patterns (DAMPs; Abdul Malik et al. 2020; Wang et al. 2021). When plants perceive MAMPs or HAMPs, an increment in the level of JA occurs in the cell followed by the activation of downstream immune responses. A similar response follows on plant's recognition of DAMPs, such as portions of plant cell walls that are degraded during mechanical damage or pathogen attack (Abdul Malik et al. 2020). Plants detect not only wounds but also pressure created by insects' movement, landing, and other mechanical damage, which upregulates the production of JA and its signalling

cascades (Singh et al. 2021). The main active form of JA is JA-Ile, which is conjugated JA with the amino acid isoleucine. Cell surface receptors detect pathogens and abiotic factors as initiating signals. This sets off the production of JA-Ile from plastid lipids, which in turn leads to interactions with transcription factors (TFs). These interactions promote growth, development, and the activation of specialized safeguarding strategies in plants. The JA-mediated signalling pathways might exhibit individual, synergistic, or antagonistic effects depending on the specific invading pathogen (Ghorbel et al. 2021). The immune response mediated by JA is initiated upon the recognition of signals transmitted by HAMPs, MAMPs, and DAMPs. This recognition event ultimately activates specific defensive pathways and subsequent regulation of gene transcription (Lalotra et al. 2020). However, recent studies have shown that the interaction of JA with other defence-responsive hormones like salicylic acid (SA), ethylene (ET) and abscisic acid (ABA), may lead to a complex crosstalk between the two pathways in regulating plant defence responses (Li et al. 2019; Kaya et al. 2023). Numerous studies have demonstrated that the endogenous signalling molecules JA play a crucial role in regulating the immune system of plants. Moreover, it has been observed that the exogenous application of different concentrations of these hormones can augment the plants' resistance against pathogens. Additionally, such phytohormonal priming has been found to stimulate plant growth and development by modulating the expression of immune-responsive genes at both the transcriptional and translational levels (Sun et al. 2023).

In addition, studying the influence of environmental factors on JA signalling and delving into plant–pathogen interactions involving JA will broaden our insights into the adaptability and versatility of JA-mediated defences in plants. Overall, future research endeavours in this field are poised to advance our understanding of plant immunity, offering innovative solutions for crop protection in the face of evolving biotic challenges. In the present context, this review primarily aims to examine the immune defence response in plants against invading pathogens, specifically focusing on the molecular role of JA, its signalling mechanism, molecular components (like genes, TFs, proteins), interactions of JA with other defence responsive phytohormones and JA-mediated exogenous priming for pathogen resistance.

JA Biosynthetic Network in Plants

JA biosynthesis plays a crucial role in plant immunity against various biotic stresses, such as herbivore attacks and pathogen infections (Antico et al. 2012). JA biosynthesis typically begins when a plant perceives external stress signals, such as physical damage from herbivores or recognition of specific PAMPs or effector proteins from pathogens, which triggers a cascade of downstream synthesis (Campos et al. 2014). The biosynthesis of JA takes place in different cellular compartments, mainly the chloroplasts and peroxisomes, and follows the octadecanoid pathway (Fig. 1, Waqas et al. 2018).

In response to stresses, plant's cell membrane releases polyunsaturated fatty acids (PUFAs), typically α-linolenic acid in the chloroplast. These phospholipids are precursors for JA biosynthesis that commences with its oxidation catalysed by phospholipase A2 to form 18:3 fatty acid, which is further converted to 13(S)-hydroperoxy-octadecatrienoic acid (13-HPOT) by lipoxygenase (Wasternack and Hause 2013). JA biosynthesis involves a series of enzymatic conversions to form *cis*-(+)-12-oxo-phytodienoic acid (12-OPDA) mediated by lipoxygenase (13-LOX), allene oxide synthase (AOS) and allene oxide cyclase (AOC) (Aleman et al. 2022). 12-OPDA can be transported from chloroplast to the peroxisomes by ATP-binding cassette transporter, COMATOSE (CTS), and peroxisomal ABC transporter1 (PXA1), where it undergoes further enzymatic conversion to ultimately produce JA. The reactions are mediated by OPDA reductase and involve a series of β-oxidation processes. OPDA reductase (OPR) converts OPDA to OPC8 [3-oxo-2-(2-pentenyl)-cyclopentane-1-octanoic acid], which is subjected to three rounds of oxidation by acyl-CoA oxidase1 (ACX1) to ultimately synthesize JA (Wasternack and Strnad 2019). The produced JA [JA-(+)-7-iso-JA] is then transported to the cytoplasm and undergoes conversion to its bioactive forms—MeJA, JA-Ile and 12-OH-JA. MeJA and JA-Ile are produced by the action of JA carboxyl methyl transferase (JMT) and JA-amino acid synthase1 (JAR1), respectively. JA-Ile is further converted either to 12-COOH-JA-Ile or is responsible for the disease resistance/plant immunity-induced genes' expression by entering the nucleus (Fig. 1; Wasternack and Strnad 2019).

Once JA is produced, it acts as a signalling molecule, triggering a cascade of events involved in plant defence responses (Ruan et al. 2019). This includes the expression of genes involved in the synthesis of defensive compounds, such as protease inhibitors, toxins, and volatile organic compounds, which deter herbivores and inhibit the growth of necrotrophic pathogens (Antico et al. 2012). Furthermore, JA biosynthesis can also be regulated by other phytohormones, such as SA and ET by interacting with JA signalling pathways to modulate the plant's defence responses against biotic stresses. The signal perception of JA in the cells is through receptor complexes, such as coronatine-insensitive1 (COI1) thereby precise perception and signalling of JA leading to the activation of JA responsive genes. The JA-responsive genes further lead to the biosynthesis of a multitude of secondary metabolites, such as terpenoids and alkaloids which further yield resistance against phytopathogens. JA biosynthesis is governed by substrate availability, and tissue specificity, and is regulated

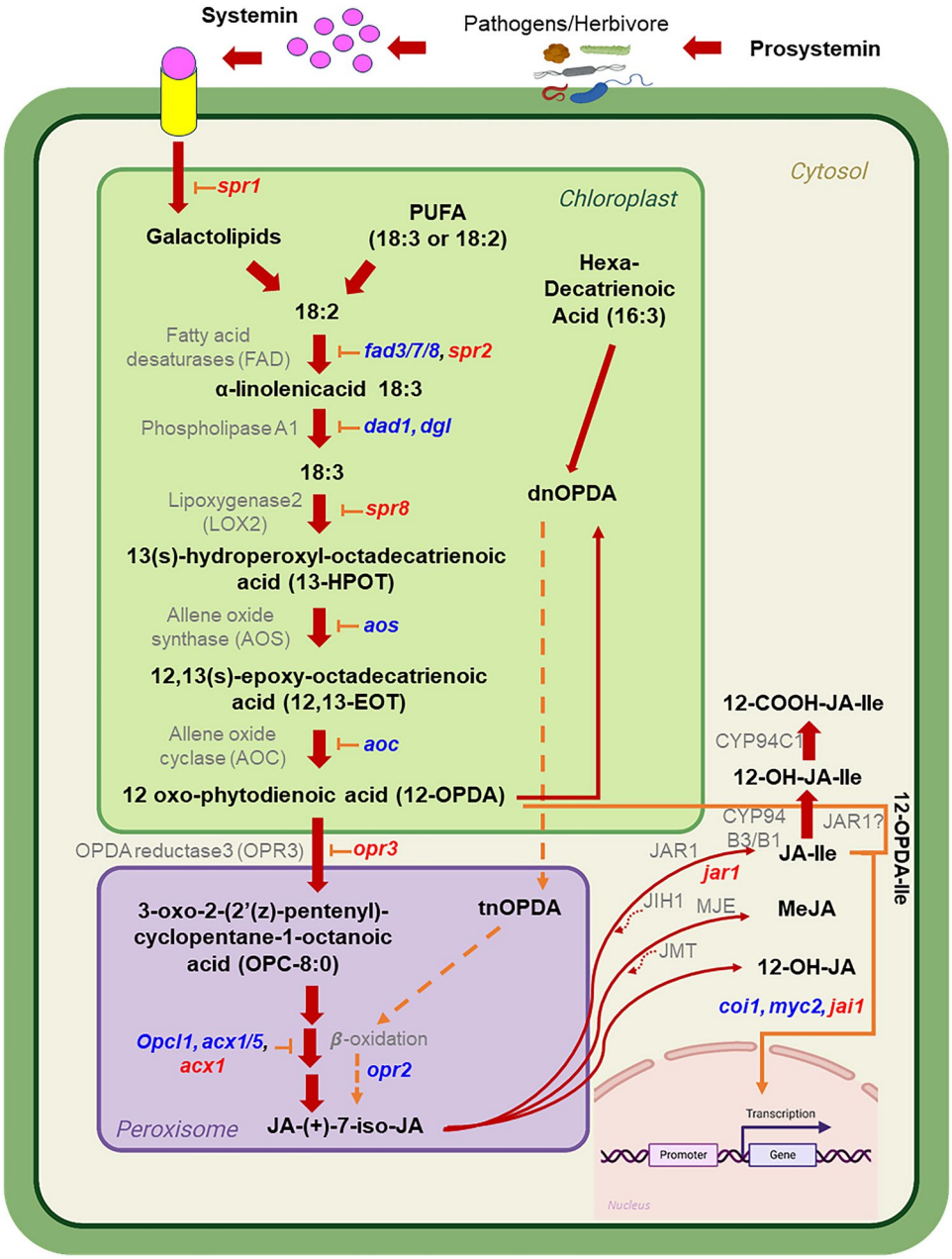

Fig. 1 Jasmonic acid biosynthesis processes inside a plant cell through the invasion of pathogens. Upon insects, pathogens, or mechanical damage happens, systemin is produced from its precursor protein, prosystemin which trigger the JA biosynthesis by interacting with its suggested membrane-bound receptor. JA is synthesized from 18:3, a type of galactolipid. Several enzymes, such as lipoxygenase (LOX), allene oxide synthase (AOS), and allene oxide cyclase (AOC), work in a certain order to make 12-oxo-phytodienoic acid (12-OPDA) in the chloroplast. Then, 12-OPDA is taken to the peroxisome and changed into 3-oxo-2-(2′(Z)-pentenyl)-cyclopentane-1-octanoic acid (OPC-8:0), which further undergoes through three rounds of β-oxidation to produce JA. In the conversion steps, many regulatory genes (check points) and their mutants have been studied in *Arabidopsis* (blue) and tomato (red) so far for restricting JA biosynthesis. Abbreviations are defined in the text (Color figure online)

by the multiple intracellular processes, such as ROS, calcium ions, calcium-dependent protein kinases (CDPKs), and mitogen-activated protein kinase (MAPK) cascades that serve to amplify the signal from local leaves to the systemic parts and from the cytosol to the nuclear region, in turn remodelling the expression of defence related genes (Zhang et al. 2017a; Singh et al. 2021).

JA-Associated Genes, TFs and Proteins in Plant Immune Response

JA's effect on plants manifests as a variety of physiological and molecular responses. The physiological responses in plants frequently involve the activation of antioxidant system through activating enzymatic antioxidants, such as

superoxide dismutase, catalase, and peroxidase that play critical roles during oxidative stress (Maksimov et al. 2011; Yarullina et al. 2011; Karpets et al. 2014). JA also triggers the accumulation of amino acids (isoleucine, proline and methionine), soluble proteins, malondialdehyde, and sugars (Sharma et al. 2019; Pandita 2022). It affects maintaining membrane integrity, and scavenging activity, as well as controlling water absorption capacity by the modulation of stomatal opening and closing (Zamora et al. 2021; Rao et al. 2023). The JA-associated molecular responses pertain to the regulation of the expression of JA-associated genes (*JAZ*, *AOS1*, *AOC*, *LOX2*, and *COI1*) (Wasternack 2014; Wang et al. 2020; Li et al. 2022; Hewedy et al. 2023; Singh et al. 2023), and interactions of JA with other plant hormones such as SA, ET, ABA, brassinosteroid (BR), gibberellic acid (GA), and auxin (IAA). (Shigenaga and Argueso 2016; Yang et al. 2019; Jang et al. 2020; Liu and Timko 2021), and TFs—MYC2 and bHLH148 (Wang et al. 2020, 2021; Hewedy et al. 2023).

The JA signalling starts with the recognition of MAMPs/HAMPs/DAMPs by plant PRRs, which transduce the signal to start the JA biosynthesis. The newly synthesized JA is released into the cytosol, where JA-Ile-conjugate synthetase (JASMONATE RESISTANT, JAR1) brings about the conjugation of JA with isoleucine to form JA-Ile. Mutants defective in the early steps of JA biosynthesis (such as mutants for AOS or COI1) are highly susceptible to infection by *Alternaria brassicicola* (Schilmiller et al. 2007). Similarly, the silencing of the OPR gene in two independent transgenic tomato lines (*SiOPR3-1* and *SiOPR3-2*) increased their susceptibility to the necrotrophic pathogen *B. cinerea* (Scalschi et al. 2015). The plant's susceptibility to the pathogen coincides with the significant decrease in JA-Ile production. Earlier, Bosch et al. (2014) proposed that JA/JA-Ile is requisite for a systemic response in plants as opposed to a local defence response where OPDA can substitute for JA/JA-Ile in the induction of defence gene expression. *LOX* is another JA biosynthetic gene that has been widely studied for its effect during biotic stress response highlighting the prominence of JA-mediated signalling in disease retaliation. LOXs gene has been implicated in the defence response against infestation of aphids in Sorghum (Shrestha et al. 2021). The study reported higher expression of two 13-LOXs (*SbLOX9* and *SbLOX5*) and three 9-LOXs (*SbLOX1*, *SbLOX3*, and *SbLOXo*) during aphid feeding, indicating JA involvement in plant defence response. In another study, *LOX* showed different expression patterns in wild-type and cultivated peanut plants infected with *Aspergillus flavus* which was taken as an explanation for divergent disease resistance amongst different cultivars (Song et al. 2016). In a similar study, gene expression and activity of *LOX* was found to be very low in healthy tissues of tobacco plants but is highly enhanced upon infection by *Phytophthora parasitica nicotianae* (Mène-Saffrané et al. 2003). The transgenic tobacco plants with high expression levels of LOX protein displayed significant resistance to *Phytophthora* infection as well as an increase in survivability of the infected plants.

At the translational level, regulators of JA signalling can be categorised into two groups: the first includes components of SCF^{COI1} complex, whilst the second consists of TFs such as MYC2, which coordinate the expression of JA-regulated genes. As mentioned earlier, the detection of pathogens triggers de novo production of JA-Ile from plastid lipids. The JA receptor protein, COI1 binds to JA-Ile as well as interacts with MAMP or DAMP receptors to modulate downstream signalling pathways (Katsir et al. 2008). The protein encoded by *COI1* contains a degenerate F-box motif that interacts with Skp1 and Cdc53 (cullin) to assemble SCF ubiquitin-ligase (Skp1-Cdc53-F-box protein, SCF^{COI1}) complexes (Xu et al. 2002). In the resting cells, JA-responsive genes are suppressed by the action of repressor protein—JASMONATE ZIM (JAZ), which along with adaptor protein, NOVEL INTERACTOR OF JAZ (NINJA), and co-repressor TOPLESS (TPL) bind to JA TFs (MYC) (Singh et al. 2021; Hou and Tsuda 2022). However, in response to biotic challenges or developmental cues, JA-Ile promulgates recognition of JAZ proteins by the COI1 of SCFCOI1 complexes (Zander et al. 2020). This is followed by the subsequent degradation of JAZ by the ubiquitin-dependent proteolytic pathway by SCFCOI1 and release of TFs such as MYC2, MYC3, MYC4, GLABRA3 (GL3), ENHANCER OF GLABRA3 1 (EGL1), and TRANSPARENT TESTA8 (TT8) involved in various aspects of JA-signalling (Kazan and Manners 2013). The SCFCOI1-JAZ regulatory module depicts a positive feedback loop. Although it is first activated during the expression of JA biosynthetic genes, the subsequent synthesis of JA/JA-Ile, JAZ degradation, and release of TF *MYC2*, further induces the upregulation of JA-responsive promoters involved in their biosynthesis (Chung et al. 2008).

TFs play a pivotal role in the intricate pathways of JA signalling, and are crucial for the activation of defence mechanisms against herbivores, pathogens, and abiotic stressors. The complex interplay between TFs and JA signalling pathways ensures a finely tuned and rapid response to environmental challenges, highlighting their indispensable role in orchestrating the plant's defence strategies. MYC2, in particular, has emerged as a master regulator of JA signalling, integrating a variety of endogenous and exogenous signals (Chung et al. 2008). As stated earlier, the release of MYC2 from JAZ binding up-regulates JA-responsive promoters involved in JA biosynthetic genes (Chung et al. 2008), the defect in binding ability or loss of interaction with JAZ repressors leads to hyperactive TF and activation of a subset of JA responses (Goossens et al. 2015; Major et al. 2017). MYC proteins belong to the basic helix–loop–helix

(bHLH) family (Heim et al. 2003) and act both as an activator and repressor of downstream JA responses. It serves as a molecular switch that regulates the expression of target genes, orchestrating a cascade of events that culminate in the activation of defence responses. MYC2 has been implicated in antagonistically coordinating the defence response against pests and pathogens, as well as regulating the interaction of JA with other phytohormones. MYC2 plays a central role in the JA-mediated induction of ISR in the roots of *Arabidopsis thaliana* infected by the beneficial rhizobacterial strain *Pseudomonas fluorescens* WCS417r (Pozo et al. 2008). The mutants for *myc2* gene failed to depict WCS417r-induced ISR on infection by *Pst* DC3000 and *Hyaloperonospora parasitica*. In addition, JA-mediated systemic immunity that involves a transient increase in JA biosynthetic and associated genes is also moderated by *MYC2* (Truman et al. 2007). The role of *MYC2* in SAR however remain ambiguous with one study stating reduced systemic immunity in *Pst* DC3000 inoculated *myc2* mutants (Truman et al. 2007) whilst other reporting retainment of SAR in *myc2* mutants pre-inoculated with *P. s.* pv. *maculicola* (Attaran et al. 2009). MYC2 negatively regulate JA-mediated defence response against fungal pathogens (Anderson et al. 2004), whilst acting as a positive regulator in resistance against insect pests (Dombrecht et al. 2007; Pauwels and Goossens 2011). The *myc2* mutants showed increased resistance to infection by the fungal pathogen *Fusarium oxysporum*, which was attributed to the reduction in JA sensitivity that may attenuate pathogen-induced lesion development (Anderson et al. 2004; Kazan and Manners 2013). The positive regulation of *MYC2* has been reported in response to wound-responsive genes (such as *VSP1*, *JR1*) and to insect herbivory (Dombrecht et al. 2007). The mutant showed reduced expression of JA responsive genes indicating reduced resistance and an increased susceptibility to the generalist herbivore, *Helicoverpa armigera*.

JA has been reported to regulate the expression and activity of many proteins that are involved in plant defence against herbivores and pathogens. For example, JA can induce the expression of genes encoding for enzymes involved in the biosynthesis of specialized metabolites, such as JAs, glucosinolates, and terpenoids, which deter herbivores and inhibit the growth of pathogens (Wasternack and Strnad 2019). Another way in which JA affects the immune response is by regulating the activity and accumulation of defence-related proteins. JA-responsive genes include a variety of defence-related proteins, including protease inhibitors, proteinase inhibitors, and pathogenesis-related (PR) proteins (Yang et al. 2019). JA signalling activates the activity of proteinase inhibitors (PIs), which are proteins that inhibit the activity of digestive enzymes of herbivores, making the plant less palatable and nutritious (Chen et al. 2005). JA also functionally activates PR proteins having antimicrobial activity and can directly inhibit pathogen growth or induce plant resistance to subsequent infections (Ali et al. 2018). JA signalling induces the expression of several PR proteins, including PR-1, PR-2, and PR-5 (Ali et al. 2018). In addition to its effects on specific proteins, JA also affects general cellular processes that are important for plant immunity. For example, JA has been shown to activate the activity of MAPKs involved in the regulation of defence response signalling and its mediated programmed cell death (PCD; Jagodzik et al. 2018). Agrawal et al. (2003) reported significant expression of JA-induced MAPK gene (*OsBWMK1*) in rice seedlings within 15 min of wounding or treatment with fungal elicitors, including blast infection Overall, JA plays an important role in the protein level of plant immunity, by regulating the expression and activity of many proteins involved in plant defence against herbivores and pathogens.

JA Signalling Pathway: Elicitors, Downstream Signal Amplification, and Pathogen Resistance

The precise regulation of genes associated with the synthesis pathway of JA occurs at various levels both inside and outside the nucleus. As mentioned before, this intricate control system forms a group of genetic and epigenetic regulatory processes, starting from JAs binding to the COI1 receptor located within the nucleus to set off a chain reaction in signal transduction. This leads to the recruitment and successive breakdown of JAZ proteins which are the negative regulators associated with the JA response. Degradation or breakdown of JAZ proteins frees up downstream TFs, triggering the onset of the JA-mediated plant resistance response to influence growth and developmental processes. Prior investigations demonstrate the diminished presence of JA during the periods of plant quiescence accompanied by a substantial buildup of the JA response inhibitor known as the JAZ protein. This JAZ protein tends to form complexes with various co-inhibitors like TPL, HDAs, and PcG, as well as with a sequence of downstream TFs within the JA signalling pathway (Fig. 2a). This complex interaction effectively regulates the activity of the JA pathway, leading to suppression of JA response (Wasternack and Hause 2013). However, the presence of external stimuli, either environmental or developmental, prompts an upsurge in JA synthesis in plants which results in their accumulation of higher JA content.

JA signalling is perceived by the COI1 receptor, *Arabidopsis* serine/threonine kinase 1 (ASK1), CULLIN1 (CUL1) as well as Rbx1 proteins. A pivotal advancement in the JA signalling cascade was achieved through the identification of the F-box protein COI1. This protein acquaintances with SKP1 and CULLIN1 to form the SCF^{COI1} E3 ligase complex, a crucial mediator of JA responses. Upon detection of

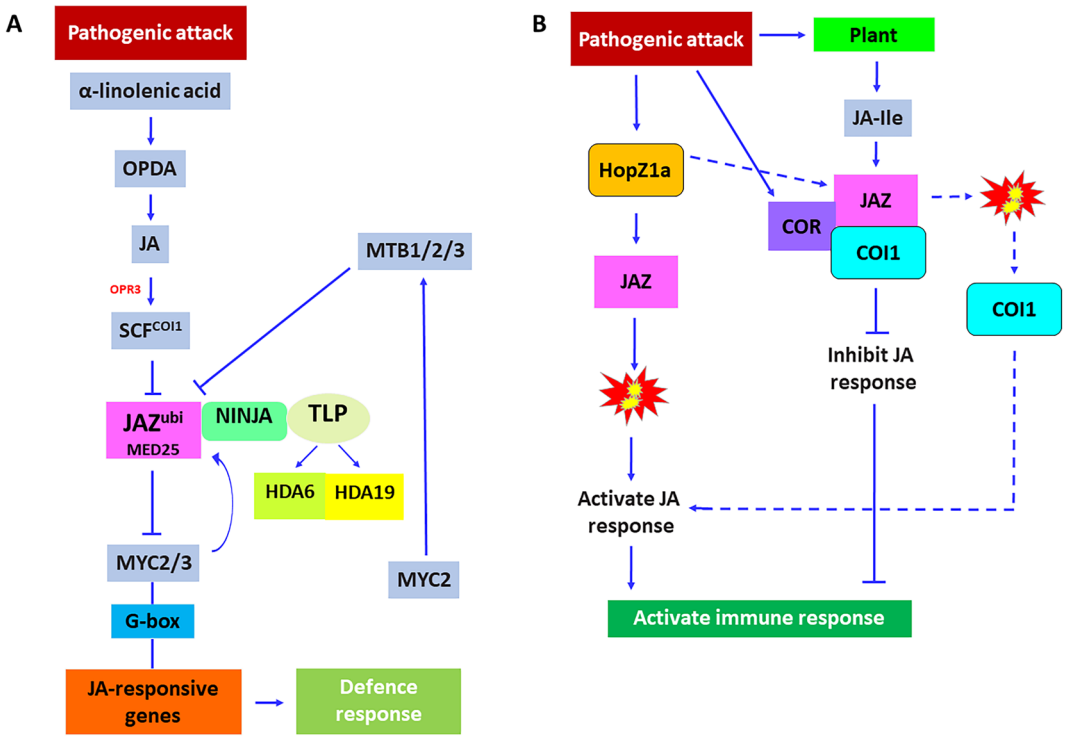

Fig. 2 Schematic representation of jasmonic acid (JA) signal induction and its regulatory mechanism. **A** The JA biosynthesis initiates with the release of α-linolenic acid, which subsequently get converted into *cis*-(+)12-oxo-phytodienoic acid (OPDA) through the sequential action of 13-lipoxygenases (13-LOX). OPDA then undergoes reduction by OPDA reductases 2 and 3 (OPR2 and OPR3) and is subsequently oxidized to JA through two distinct pathways. JA activates the signal transduction cascade by binding to COI1 protein, recruiting the JA inhibitor JAZ protein for degradation, thereby releasing downstream transcription factors (TFs) which initiates JA-regulated plant responses. The perception of JA necessitates JAZ that acts as a bridge between COI1 and JA-Ile. Following JA-Ile binding, the SCF-COI1 complex ubiquitinates JAZ proteins, marking them for proteasomal degradation. This releases MYCs TFs, which were previously sequestered by JAZ proteins, enabling them to activate the JA response. MYC2, in turn, triggers the activation of MYC2-TARGETED BHLH 1 (MTB1), MTB2, and MTB3 proteins that disrupts the interaction between MYC2 and MED25 and engages in competition with itself for binding to its target gene promoter. This competition inhibits the transcriptional activation of MYC2, resulting in negative regulation of the JA signalling response. **B** JA-mediated resistance response against bacterial attack in *Arabidopsis.* HopZ1a effector from pathogen engages in direct interaction with JAZ proteins and triggers its acetylation, activating the JA signalling pathway. Meanwhile, COR induces COI1 to bind to the JAZ proteins, thereby facilitating the activation of the downstream JA response and ensuing plant immune response. Abbreviations are defined in the text

JA signalling, COI1 forms a link with JAZ proteins, initiating their ubiquitination and subsequent breakdown through 26S proteasome pathway. This mechanism entails a competitive interaction leading to the degradation of JAZ repressors. Subsequently, these repressors release downstream TFs like MYCs, which were initially bound by JAZ proteins; which culminate in the activation of JA responses by MYCs (Gimenez-Ibanez et al. 2017; Dubois et al. 2018). COI1 protein, alongside JAZ and MYC, forms the core of JA signal transduction systems and has been validated as a pivotal intersection for various other signal transduction pathways in response to diverse stressors. The interactions between COI1 and JAZ are facilitated by a pair of conserved domains of JAZ protein, namely the ZIM and Jas domains (Thireault et al. 2015). The Jas domain plays a crucial role in facilitating exchanges between JAZ and either COI1 or other TFs (Gimenez-Ibanez et al. 2015). Meanwhile, the ZIM (TIFY) of the JAZ protein is responsible for orchestrating the dimerization of JAZ and its binding to NINJA. This interaction with NINJA further leads to the recruitment of regular transcriptional co-suppressor TPL via the conserved EAR domain. Intriguingly, this process involves a competition with MEDIATOR25 (MED25) for interaction with MYCs (Zhang et al. 2015a). Unlike the positive regulatory role played by JAZ in plants, the MYC TFs (MYC2, MYC3, and MYC4) have a negative impact on gene expression during the cell cycle, consequently hindering plant growth (Campos et al. 2016; Major et al. 2017). As the overall switch of JA signalling pathway, MYC2 is the crucial TF (Gangappa et al. 2013). JAZ proteins, in combination with various TFs, establish distinct JAZ/TF complexes tailored to precisely regulate a multitude of downstream responses (Chini et al. 2016).

Amongst these complexes, the JAZ-MYC module stands out for its role in increasing the amount of defence compounds to initiate a defence response or prevent plant growth in the face of pathogen infection (Havko et al. 2016). Beyond the JAZ-MYC module, various other modules like COI1-JAZ2-MYC2, 3,4-ANAC19,55,72 (Gimenez-Ibanez et al. 2017) and specific JAZ-TF modules have also been elucidated (Jin and Zhu 2017; Mao et al. 2017). The interaction between MYCs and JAZs may extend to encompass other signalling pathways associated with plant hormones. For instance, it could involve ET-mediated cell division, orchestrated by ET response factor (ERF) TFs (Dubois et al. 2018). Recently, Liu et al. (2019b) reported that MYC2 initiates the activation of a cluster of JA-responsive basic helix–loop–helix (bHLH) proteins, named MYC2-TARGETED BHLH 1 (MTB1), MTB2, and MTB3. In this, MYC2 impairs the establishment of its complex with MED25 and, intriguingly, competes with itself to secure binding to the promoter region of its target genes. This dual action serves to inhibit MYC2's transcriptional activation capacity and consequently exerts a dampening effect on JA signalling response. Notably, the interplay between MYC2 and the MTB proteins initiates a self-regulatory negative feedback loop which plays an essential role in orchestrating a meticulous termination of JA signals, ensuring a well-ordered manner for the JA signalling process.

Initiation of the JA signalling pathway in plants as a reaction to pathogenic challenges encompasses the identification of a range of pathogen-derived and endogenous elicitors. These elicitors set off a cascade leading to the accumulation of endogenous JA levels, ultimately instigating a defence response. Understanding the assortment of these elicitors offers insights into the complexities inherent in interactions between plants and pathogens, as well as into the underlying mechanisms governing JA-triggered defence mechanisms. In fungi, chitin forms a prominent constituent of their cell walls, and prompted by pathogen-derived elicitors, plants can identify chitin fragments referred to as chitin oligomers or chito-oligosaccharides (COs) through specialised PRRs, for instance, chitin elicitor receptor kinase 1 (CERK1). Perception of chitin triggers the activation of the JA pathway, ultimately prompting defence responses against fungal adversaries. Similarly, flagellin, a protein present in bacterial flagella, and elongation factor-thermo unstable (EF-Tu), which is found in both bacterial and fungal pathogens, act as elicitors that can be detected by PRRs. The interaction of flagellin and EF-Tu with PRRs initiates a series of signalling events, culminating in the accumulation of JA and thereby contributing to the plant's defence mechanisms against pathogenic intruders. Oligosaccharides sourced from pathogenic entities, like the bacterial elicitors, oligogalacturonides (OGs) and lipopolysaccharides (LPS), can initiate JA-based responses. These elicitors can stimulate the release of JA and its associated derivatives, thereby intensifying the plant's defence signalling. Moreover, an intrinsic peptide hormone known as systemin assumes a pivotal function in both wound signalling and defence reactions. Following tissue damage, the release of systemin ensues, prompting the buildup of JA and the activation of genes responsive to JA. The responses facilitated through the action of systemin are imperative for defence against herbivores and pests. Also, DAMPs emerge as internal entities released amid tissue damage caused by pathogens or physical stress. Identification of DAMPs by PRRs or other receptors can initiate JA accumulation and subsequently initiate the defence responses. Additionally, volatile organic compounds (VOCs) emanating from damaged or infected plant tissues serve as endogenous elicitors. Notably, these VOCs, exemplified by green leaf volatiles (GLVs), possess the capacity to induce JA-dependent defence responses in neighbouring plants, effectively preparing them for potential pathogenic attacks.

The diversity of elicitors and their interactions with the JA pathway highlights the intricate nature of plants responding to pathogenic challenges. Moreover, the signalling pathway governed by JA primarily participates in bolstering the plant's resilience against necrotrophic pathogens or the necrotrophic phase of hemi-biotrophic pathogens (Spoel et al. 2007). Hemi-biotrophic and necrotrophic fungi exhibit broad host spectra, thereby leading to substantial reductions in yields across numerous vital crops (Okada et al. 2015; Pandey et al. 2016). Thus, in terms of pathogen infection, the JA signalling pathway plays a crucial role in enhancing the plants' defence mechanism. In strawberry, grey mould disease caused by *B. cinerea*, JA was found to positively manage the expression of its biosynthetic pathway genes, namely *LOS*, *AOS*, and *OPR* (Chung et al. 2008). Moreover, JA also directed the activation of associated signalling pathway genes, *COI1* and *JMT*. Notably, the infection by *B. cinerea* prompts JA to induce *FaTPS1* expression through *FaMYC2* which orchestrates terpene synthesis within strawberry fruit. The rapid increases in the terpene content, particularly of sesquiterpenoids such as daulene D, improve strawberry's ability to resist infection by *B. cinerea* (Zhang et al. 2022). The bHLH TF gene *SlJIG*, pivotal in the process of JA-triggered terpene biosynthesis, has been observed as a direct target of MYC2 (Huang et al. 2023). It establishes a functional module, MYC2-*SlJIG*, that plays a decisive role in both terpene synthesis and enhancing the plant's resistance to challenges posed by the cotton bollworm and *B. cinerea*. To substantiate this, plants with *SlJIG* knocked out using gene editing exhibited a decrease in terpene content compared to the wild type, attributable to the diminished expression of terpene synthase genes (Cao et al. 2022). Furthermore, the *SlMYC2* gene plays a crucial role in regulating JA-induced disease resistance, and the knockout of *SlMYC2* resulted in a significant decrease in both defensive and

antioxidant enzyme activities (Shu et al. 2020). The plants with the slmyc2 mutation showed a significant reduction in the expression levels of PR genes (*SlPR-1* and *SlPR-STH2*), as well as crucial genes associated with the JA biosynthesis and signalling pathway. These include *allene oxide cyclase* (*SlAOC*), *lipoxygenase D* (*SlLOXD*), *SlMYC2*, and *coronatine insensitive 1* (*SlCOI1*). This was shown to contribute to the exacerbation in the severity of disease symptoms caused by *B. cinerea*.

The orchestration of disease resistance through JAs also entails engagement with epigenetic regulatory mechanisms (Khan et al. 2023). During the infections of hemi-biotrophic fungi *Fusarium brachygibbosum* in tobacco plants, AGO4-dependent miRNAs occupied a pivotal position in regulating both JA biogenesis and signalling (Pradhan et al. 2020). The suppression of *AGO4* led to a pronounced shift in the accumulation patterns of miRNAs, with *AGO4*-silenced (*irAGO4*) plants exhibiting a marked decrease in JA levels as well as a reduction in the transcripts of genes crucial for JA biosynthesis. Silencing *AGO4* (*irAGO4*) in plants heightened the epigenetic susceptibility to pathogens. Consequently, infection by pathogens led to decreased levels of JA and transcripts associated with its biosynthesis, including *LOX3* and *OPR3*. However, when *irAGO4* plants were treated with JA or 12-OPDA, their tolerance levels were reinstated to that of the wild-type plants. The study has also shown the potential role of long non-coding RNA, lncRNA4504 in JA-induced tomato disease resistance (Fu et al. 2022). This potential role may involve facilitating the augmentation of total phenols and flavonoids, intensifying the actions of defensive enzymes, and promoting the upregulation of genes within the JA signalling pathway. In the same study, JA treatment of tomato plants also promoted the transcription of genes associated with JA biosynthesis (*SlAOC*, *SlLOXD* and *SlAOS*) and their signal transduction (SlCOI1 and SlMYC2), concurrent with an increase in endogenous JA levels. Interestingly, the impact of JA on the aforementioned indicators was largely mitigated upon silencing of lncRNA4504, leading to a greater disease incidence and intensity by *B. cinerea* in the lncRNA4504-silenced plants compared to the wild group. In cases of injury or the presence of (pro)-systemin, tomato plants necessitate the initiation of the JA biosynthetic pathway (Li et al. 2002). This activation leads to the production of distant signals that can propagate to distant locations. Importantly, the perception of these signals in remote leaves relies on the functionality of JA signalling. This observation indicates the possibility that JA or a closely associated compound stemming from the octadecane pathway could potentially serve as a transmissible signal in response to wounds. The application of JA leads to the upregulation of antioxidant enzyme genes (*PavSOD*, *PavPOD*, *PavCAT* and *PavPPO*), as well as pivotal genes associated with JA biosynthesis and the signal transduction pathways (*PavOPR3*, *PavAOS*, *PavLOX*, and *PavMYC2*) (Pan et al. 2022). This effect extends to elevating the functions of antioxidant enzymes and enzymes linked to disease resistance (polyphenol oxidase, phenylalanine ammonia-lyase, peroxidase, superoxide dismutase, catalase, chitinase, and -1, 3-glucanase). This concerted enhancement serves the purpose of diminishing the decay rate caused by *Alternaria alternata* in the post-harvest sweet cherries. JA has also exhibited improved resistance to *F. oxysporum* f. sp. *albedinis* infection in date palm roots (Jaiti et al. 2009). This enhancement is achieved by triggering the activation of enzymes associated with defence, including PPO and POX. Also, an array of external stressors such as mechanical damage, insect herbivory, pathogenic invasion, and similar adverse conditions, can prompt the synthesis of JAs, thereby instigating a response governed by the JA pathway. Phytohormones such as ET exert a positive influence on the expression of genes related to JA synthesis, contributing to the swift onset of a burst of JA production (Hu et al. 2021). The comprehension of the wide array of elicitors and their interactions offers valuable insights into the intricate mechanisms that plants employ to fend off pathogens and adapt to the ever-changing challenges presented by their environment.

Pathogens have also evolved sophisticated approaches to suppress plant immune responses, including the manipulation or weakening of the JA signalling pathway. Pathogens can produce effector proteins that directly interfere with components of the JA pathway, disrupting the signalling cascade and compromising the plant's competence to mount an effective defence. Additionally, some pathogens can induce the expression of negative regulators or decoy proteins that divert the plant's resources away from activating the JA pathway. Moreover, the antibiotic biosynthetic monooxygenase (Abm) produced by *Magnaporthe grisea* serves to alter JA into 12-OH-JA, strategically weakening the JA signalling pathway, whilst promoting the colonization of *M. oryzae* by facilitating favourable conditions (Patkar et al. 2015; Zhang et al. 2017a). The rice blast fungus establishes compatibility with rice when it releases Abm to effectively inhibit JA's activity and compromise the host's immune response. Conversely, during incompatible interactions between the rice blast fungus and rice, the Abm secreted by the pathogen undergoes degradation. Consequently, JA accumulates, setting in motion downstream responses as well as an immune response. Notably, this promotion of pathogenesis correlates closely with the expression of *LOB* domain-containing protein 20 (LBD20), which has significant associations with VSP2, THIONIN 2.1 (Thi2.1), and MYC2 (Thireault et al. 2015). Incidentally, numerous strains of hemibiotrophic bacteria, specifically *Pseudomonas syringae*, are capable of producing pathogenic variants encompassing polyketide toxin coronatine (COR), HopZ1a and AvrB. One of the

extensively recognized instances of plant–pathogen interaction influenced by JA is associated with COR regulation. COR, a bioactive compound structurally and functionally akin to JA–Ile, consists of two components: coronamic acid and coronafacic acid (Bender et al. 1999). On one side, COR demonstrates the ability to enhance bacterial infection by impacting PTI, leading to stomatal closure and defence response (Zhang et al. 2015a). Conversely, COR also directly binds to the COI1-JAZ complex, and the ensuing initiation of the COR-mediated JA signalling pathway curbs SA-mediated defence resistance, thus facilitating the success of *P. syringae* infection (Zhang et al. 2015b). In addition, COR possesses toxic attributes encompassing the management of secondary metabolites and the suppression of callose formation. This toxicity operates independently of any antagonistic effects on plant hormones (Millet et al. 2010; Geng et al. 2012). This implies that JA-Ile mimics for instance COR might play an indispensable role in facilitating the infection process of certain bacterial pathogens. COR might also augment the interaction between COI1 and JAZ proteins (Zhou et al. 2015a). AvrB governs the JA signalling pathway in *Arabidopsis* by modulating it in a COI1-dependent manner (He et al. 2004). This process appears to involve the participation of RPM1-interacting protein 4 (RIN4), a protein intrinsic to *Arabidopsis* (Zhou et al. 2015a). AvrB engages with RIN4, consequently triggering the activation of plasma membrane-localized AHA1. Both AvrB and AHA1 contribute to fostering the interaction between COI1 and JAZ proteins. This interaction, in turn, regulates both stomatal opening and the plant's defence response (Fig. 2b). In contrast to the mechanisms involving COR and AvrB, HopZ1a acts directly as an acetyltransferase and forms a direct interaction with JAZ proteins, leading to the acetylation of these proteins serves as the trigger for activating the JA signalling pathway (Jiang et al. 2013).

JA has been extensively studied for their role in defence mechanisms against plant–parasitic nematodes (PPNs) as well; their signalling pathways are crucial in the plant's defence responses to various nematodes, including root-knot nematodes (RKNs) *Meloidogyne chitwoodi* (Vieira dos Santos et al. 2013), *M. graminicola* (Nahar et al. 2011), and the cyst nematode *Heterodera schachtii* (Kammerhofer et al. 2015). It has been reported that the levels of JA or its precursor OPDA play a pivotal role in determining a plant's susceptibility to PPN. In the case of upstream JA biosynthesis mutants like *fad3-2fad7-2fad8* and *dde2-2*, susceptibility to *Meloidogyne hapla* was observed. Conversely, the downstream mutant *acx1/5* and the JA-insensitive mutant *coi1* displayed root galling comparable to the wild-type plants (Gleason et al. 2016). In a recent study, Sikder et al. (2021) demonstrated the majority of JA mutants exhibited a higher relative abundance of *M. hapla* compared to the parental lines, which implies that maintaining an intact hormonal apparatus is essential to prevent the invasion of *M. hapla*. In another study, it was documented that the application of JA resulted in a decrease in the susceptibility of tomato plants to nematode invasion. This effect was attributed to the upregulation of genes associated with protease inhibitors (Zinovieva et al. 2011). Additionally, various investigations demonstrated the utilization of JA to strengthen defence mechanisms against PPNs through the induction of proteinase inhibitors, oxylipins, defensin, thionin, and several PR proteins (Reymond and Farmer 1998; Fan et al. 2015; Naor et al. 2018; Gheysen and Mitchum 2019).

Furthermore, Bhattarai et al. (2008) found that the tomato JA perception mutant, *jai1*, exhibited a notable decrease in *M. incognita* infection, it is noteworthy that *JAI1* in tomato shares homology with *COI1* in *Arabidopsis*. Moreover, maize research has investigated *ZmLOX3* and revealed its role in suppressing the biosynthesis of JA, therefore the mutant *Zmlox3-4* exhibited elevated levels of JA (Gao et al. 2008). In a recent study, Wang et al. (2019) illustrated the transport of endogenous JA from leaves to roots, playing a crucial role in initiating defence responses against RKNs. Besides, the induction of JA and JA biosynthetic genes (such as lipoxygenase, Lox) has been reported during RKN infection in tomato (Shang et al. 2019) and soybean (Ibrahim et al. 2011). These findings imply the essential role of JA pathway initiation in the interactions between plants and RKNs. It will be interesting to observe whether future studies validate the notion that phytohormonal signalling pathways, in general, play a pivotal role in shaping root-associated nematode communities in diverse soils, potentially contributing to a greater diversity of nematode populations.

Exploring CRISPR Studies in JA-Mediated Genes

Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR) become an innovative gene editing technology that allows precise genetic modifications (Roychowdhury et al. 2020). This technology, employing the CRISPR-associated protein 9 (Cas9) and guide RNA (gRNA), enables targeted modifications within plant genomes (Singh et al. 2020). By selectively editing specific genes, CRISPR allows for the development of plants with customizing plant characteristics through the ability to knockout, insert, or gene replacement. It can be used to enhance or suppress the expression of specific genes associated with plant immune responses and depends on a thorough understanding of the underlying molecular mechanisms (Kottakota et al. 2021). CRISPR technology has the potential to be applied for editing of JA biosynthesis pathway, receptor genes, signalling pathways and JA-responsive genes.

By enhancing or inhibiting the expression of JA biosynthesis genes, CRISPR may be able to modulate the JA levels in plants and subsequently affect plant immune responses. Genes encoding JA receptors are also critical components in the JA signalling pathway. CRISPR could be employed to edit JA receptor genes, potentially leading to altered responses to JA and, consequently, changes in plant immunity. Gene editing of two COIs (*OsCOI1* and *OsCOI2*) reveals the JA-responsive function of *OsCOI2* for signalling and functional diversity with COI1 homolog. The *oscoi2* mutants exhibited suppression of MeJA-induced chlorophyll breakdown and accumulation of antimicrobial secondary metabolites to trigger plant immunity (Inagaki et al. 2023). CRISPR makes *OsANN1-Cas9* edited rice plants for calcium binding protein which are more resistant to rice blast disease caused by *M. oryzae*. *OsANN1* interacts with cytochrome P450 monooxygenase (*HAN1*) that catalyze the conversion of bioactive JA-Ile to the inactive 12OH-JA-Ile. As a result, *HAN1* negatively regulates rice immunity by inactivating JA accumulation and transport (Zhao et al. 2023a). CRISPR could be used to modify genes that are directly responsive to JA which may lead to the enhancement or suppression of specific defence mechanisms triggered by JA. For example, *METHYL JASMONATE (MeJA)-INDUCED GENE* (*SlJIG*) in tomato is the direct target of TF MYC2, and its conjugation promotes the JA-mediated induction of *TERPENE SYNTHASE* (*TPS*) genes for terpene biosynthesis. *SlJIG* gene-edited knockout lines show reduced JA responsive genes and JA-mediated induction of TPS that causes the susceptibility for *B. cinerea* and cotton bollworm (Cao et al. 2022). CRISPR is also applied to target genes downstream in the JA signalling pathway. By modifying these genes, gene editing tools may influence the plant's ability to mount an effective defence response. CRISPR-mediated knockout of JAZ2Δjas in tomatoes has an inactive Jas domain that showed resistance towards *P. syringae* and *B. cinerea* by inhibiting the stomatal opening (Ortigosa et al. 2018). The *Gh14-3-3d* gene, which was introduced into cotton plants using genome editing, was stably passed on to subsequent generations. These plants exhibited strong resistance to verticillium wilt, due to the activation of genes associated with JA signalling (Zhang et al. 2018). The mutagenesis of *SlMYC2* using CRISPR-Cas9 negatively affects tomato plant growth and the fruit's resistance to *B. cinerea*, induced by MeJA. Disrupting *SlMYC2* function had a substantial impact on the activity of *Botrytis* resistance and antioxidant enzymes, as well as the expression of PR genes (*SlPR-1*, *SlPR-STH2*) and crucial genes involved in the production and signalling of JA, e.g. *SlAOC*, *SlLOXD*, *SlMYC2*, and *SlCOI1*, and these all together exacerbated the disease symptoms (Shu et al. 2020). The knockout of *SlNPR1* increases the resilience of tomato leaf to *B. cinerea* compared to its wild-type plants via activating the JA signalling pathway and balancing ROS (Li et al. 2020a). In maize, CRISPR-mediated knockout generates two mutants—*coi1a* and *jaz15* in response to Gibberella stalk rot (GSR) caused by *Fusarium graminearum*. The former mutant is more resistant to GSR, whereas the latter is more vulnerable. In addition, the JA-deficient *opr7-5opr8-2* double mutant showed increased resistance to GSR compared to the wild type (Ma et al. 2021). CRISPR validated overexpression of cotton *4-coumarate-CoA ligase 3* (*Gh4CL3*) improved plant resistance to *Verticillium dahliae* by facilitating the activation of JA signalling, which in turn promoted the formation of lignified vascular tissue and increased metabolic flow (Alariqi et al. 2023).

JA-Mediated Localized and Systemic Acquired Resistance (SAR)

Plants possess remarkable capability to enhance their foundational resistance against future attacks by pathogens through a process known as induced resistance (Ziv et al. 2018). This phenomenon can be categorized into various types of induced resistance, which differ in terms of signalling pathways and their range of effectiveness. One form of induced resistance involves the activation of localized defence responses triggered by PAMPs via PRRs. This recognition process results in the release of defence-related compounds and proteins. Moreover, when pathogens manage to breach physical barriers through altering or breaking down the host cell walls, PRRs may detect conserved DAMPs from the plants or PAMPs from the pathogens, instigating a PTI-mediated defence mechanism (Szymański et al. 2020; De Kesel et al. 2021). Pathogens have the capability to release effectors or avr factors that can be identified by proteins possessing nucleotide-binding and leucine-rich repeat domains, known as NB-LRR proteins, as well as other resistance (R) proteins (Li et al. 2023). Subsequently, these avr factors are identified by host-encoded R proteins, that provide more enduring and robust resistance, a phenomenon referred to as ETI and is hypothesized to represent an accelerated and amplified response compared to PTI (Albers et al. 2019; Yuan et al. 2021). ETI is commonly associated with PCD occurring at the infection spot and is part of hypersensitive response (HR). These processes collectively result in the activation of defence genes, trade-offs between growth and defence, and the initiation of defence responses. Recent research has indicated that PRRs are also essential for ETI (Li et al. 2023). Additionally, the intricate and precise immune system formed through the cross-talk between host and pathogen enables beneficial microorganisms to stimulate plant protection by directing key components of the PTI and ETI processes whilst moderating host small RNAs. Inducing local responses in plants requires the transmission of defence signals across the entire plant, resulting in a comprehensive

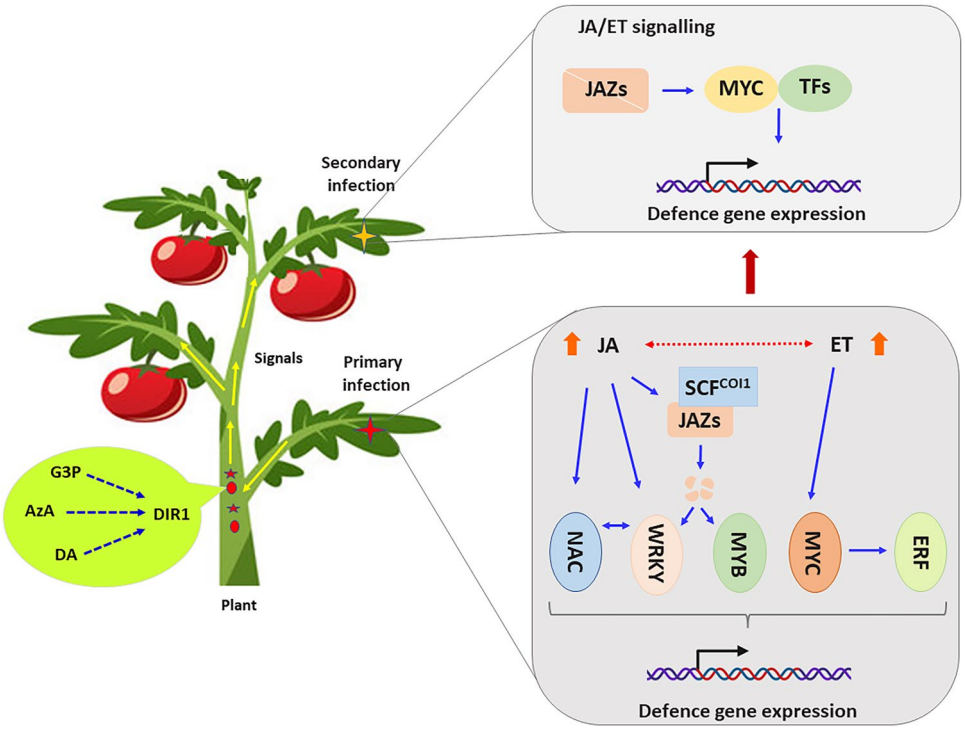

Fig. 3 Schematic overview of the molecular constituents and signalling pathways implicated in systemic acquired resistance (SAR) induced by jasmonic acid (JA) elicitors. Pathogen attack can elevate the levels of internal signalling molecules, initiating the SAR response. Activation of the JA signalling pathway occurs at the sites of infection, subsequently establishing SAR as a defence against further infections. Transcription factors (TFs) play essential roles within the JA signalling pathway, overseeing the regulation of defence genes. The JA receptor, COI1 (coronatine insensitive1), assembles into a SCF^{COI1} complex, which leads to the degradation of jasmonate-ZIM domain (JAZ) repressor proteins. Consequently, this release of related TFs either targets other TFs or directly modulates the transcription of JA-responsive genes. Additionally, ethylene (ET) signalling synergistically interacts with JA signalling, denoted by the red dotted bidirectional arrow, and ethylene response factors (ERFs) primarily function downstream in mediating defence responses. Notable components include azelaic acid (AzA), glycerol-3-phosphate (G3P), abietane diterpenoid dehydroabietinal (DA), and defective in induced resistance 1 (DIR1) (Color figure online)

resistance to secondary infections. This phenomenon is known as SAR, an enduring state of immunity that empowers plants to fend off subsequent pathogenic attacks (Fig. 3; Romanazzi et al. 2016; Yuan et al. 2021).

SAR possesses distinctive qualities that make it particularly appealing, as it holds significant promise for sustainable and enduring crop protection strategies. The initiation of SAR depends on the generation of a mobile signal known as the 'systemic acquired resistance-deficient (SARD)', which is hypothesized to be a methyl ester of JA generated at the primary infection site (Sirisha et al. 2024). This signal acts as a primer for distant tissues, boosting their defence mechanisms, which include the activation of defence-related genes and the accumulation of PR proteins. The effective initiation of SAR also depends on the perception of the mobile signal(s) in distant tissues. Although this feature of SAR remains not fully elucidated, certain factors crucial for signal perception have been recognized. Several factors contribute to this, including the existence of an intact plant cuticle. This cuticle acts as a hydrophobic barrier, consisting of wax and cutin monomers that cover all above-ground surfaces of the plant. Additionally, the crucial plant galactolipid, digalactosyldiacylglycerol (DGDG), and two proteins that localize to plasmodesmata, PDLP1 and PDLP5, play essential roles. It is important to note that plants lacking these components can produce the SAR signal but are unable to detect it (Lim et al. 2016). Whilst the cuticle was initially associated with the perception of SAR signals, subsequent research on cuticle mutants has proposed that perception might be linked to the extent of cuticular damage or potentially other unidentified factors (Harris and Mou 2023). These facets of SAR offer intriguing opportunities to investigate the intersections between SAR and fundamental physiological processes, as well as the distinct factors that determine the initiation of SAR as opposed to normal growth and development. Achieving this goal necessitates a meticulous and well-balanced cross-talk between several phytohormones, metabolites, and proteins.

In general, SAR typically furnishes comprehensive defence against a wide range of pathogenic threats, including oomycetes, fungi, viruses, and bacteria. The immune "memory" established by SAR in plants can endure for extended periods, ranging from weeks to months, potentially spanning the entire growing season (Fu and Dong 2013). In this context, genes associated with the defence that is primed by JA also exhibit heightened transcriptional activity when subjected to subsequent stress, a phenomenon in line with transcriptional memory. However, it is worth noting that the approach governing priming might differ from the mechanism governing the super-induced [+/+] transcriptional memory. To illustrate, prior exposure to biotic stress-induced hormones such as JA or SA (or BTH) did not stimulate transcription from defence genes significantly. However, it notably enhanced their expression upon subsequent attacks (Liu et al. 2023). The increased transcription following JA or SA/BTH treatment has been associated with the quick stimulation of hormone biosynthesis, the accumulation of TFs, and/or the regulation of the activity of kinases/phosphatases (Wasternack and Hause 2013). At the chromatin level, the heightened transcription of primed genes upon subsequent attacks has been associated with an increase in H3K4me3 and acetylated histone marks (H3K9, H4K5, H4K8, H4K12) (Laura et al. 2018). Small RNAs may also potentially contribute to the memory response mediated by JA (Wang and Chekanova 2016). However, the specific mechanisms responsible for the buildup of histone modifications, or whether activated Pol II (modified at Ser5P), is positioned at primed genes ahead of transcription initiation, remain unresolved (Wilkinson et al. 2023). Luna et al. (2012) and Slaughter et al. (2012) illustrated that when parental *Arabidopsis* plants were exposed to *P. syringae* pathovar tomato (*Pst*), *Pst*/avrRpt2, and β-amino butyric acid (BABA), it resulted in a swifter and more vigorous activation of defence genes. This, in turn, led to enhanced resistance against *Pst* and an unrelated pathogen, *Hyaloperonospora arabidopsidis*, in the subsequent generation. In a similar investigation into the transgenerational memory of herbivory resistance mediated by JA, Rasmann et al. (2012) revealed that JA perception through COI1 is necessary in the parental line, but not in the offspring. This suggests the involvement of COI1, and likely NPR1, in the establishment of the primed state. The execution of SAR entails a complex process of transcriptional reprogramming governed by a series of transcriptional events activated by NPR1 that targets the WRKY group of plant-specific TFs (Pandey and Somssich 2009). Certain WRKY TFs are engaged in the feedback regulation of JA synthesis. Previously, Truman et al. (2007) projected JA as the systemic signal governing SAR and observed a significant increase in JA levels 6 h after infection with *Pst* DC3000/AvrRpm1, which returned to baseline levels 11 h post-infection. SAR was found to be compromised in plants with mutations in JA-insensitive mutant sgt1b/jai4, JA-response mutant *jin1* and JA-biosynthesis mutant *opr3* plants. However, contrasting findings emerged, indicating that JA biosynthesis mutants like *dde2* and *opr3*, as well as downstream signalling mutants *coi1*, *jar1*, and *jin1*, maintained their integrity with respect to SAR (Attaran et al. 2009; Fu and Dong 2013). However, it is essential to note that JA was not found in petiole exudates possessing SAR-inducing capabilities and is effective against an extensive range of organisms, which may differ from the parental organism-inducing SAR (Chaturvedi et al. 2008). For instance, in cucumber, an initial inoculation with the fungus *Colletotrichum lagenarium*, which causes anthracnose, triggers SAR against numerous diseases caused by the pathogens (Zhu et al. 2023). In most instances, the initial inoculation results in localized necrosis. In the context of gene-for-gene resistance, a plant is either resistant or susceptible to specific pathogenic races, whereas SAR offers quantitative defence against a broad range of microorganisms (Singla et al. 2023).

JAs are also known to play a beneficial role in enhancing plant resistance against viruses. Geminiviruses, part of a family of plant viruses known for their circular, single-stranded DNA genomes, can invade a diverse range of plant species, resulting in significant yield losses in crucial crops. In *Arabidopsis*, the external application of jasmonates has been shown to decrease susceptibility to geminivirus infections (Prasad et al. 2023). Conversely, in the case of the turnip crinkle virus (TCV), the development of HR and systemic resistance in *Arabidopsis* is dependent on SA, not on JA (Jeong and Jeong 2013). Nevertheless, when a 60 µM JA treatment was followed by a 100 µM SA treatment 24 h later, it resulted in enhanced resistance to tobacco mosaic virus (TMV), cucumber mosaic virus (CMV), and turnip crinkle virus (TCV) in tobacco, *Arabidopsis*, tomato, and hot pepper (Shang et al. 2011). This implies that JA demonstrates cumulative positive impacts on resistance against RNA viruses.

Overall, the molecular mechanisms underpinning JA-mediated local and SAR reveal an intricate network of interactions governing plant defence responses (Sirisha et al. 2024). The capacity to prime plants for heightened defence via JA signalling presents promising avenues for sustainable crop protection strategies. Through a deeper comprehension and manipulation of these mechanisms, researchers and farmers can pave the way for a future in which plants are better equipped to withstand the challenges presented by evolving pathogens and pests. This contribution ultimately bolsters global food security and environmental sustainability.

Crosstalk of JA with Other Plant Defence Hormones

JA antagonistically interacts with other defence-responsive phytohormones—SA, ET, ABA, and others to orchestrate plant immune responses against pathogen attacks (Bürger and Chory 2019; Song et al. 2023). JA along with SA and ET form the backbone of plant immune responses against biotic pathogens which orchestrate effective defence responses to keep the host healthy (Zhu and Lee 2015; Li et al. 2019).

Interaction Between JA and SA

Both JA and SA have signalling pathways consisting of biosynthesizing enzymes, receptors, TFs and downstream immune-responsive genes that are interconnected and form an integrated network to combat pathogen infection and developmental cues (Ghorbel et al. 2021). Resistance conferred by biotrophic pathogens requires SA signalling for defence response (Proietti et al. 2013). The interaction between plant hormones, JA and SA is mutually opposed in most dicots, whilst the interaction may also be synergistic in exceptional cases (Aerts et al. 2021). The status of JA–SA cross-talk has been poorly documented for monocots (Van der Does et al. 2013). Untargeted and targeted metabolic fingerprinting detected several plant metabolites that decreased in phloem exudates of *Plantago lanceolata* during herbivory by *Heliothis virescens* and *Myzus persicae* (Schweiger et al. 2014). However, the survival of each herbivore was found to be reduced by JA and SA priming attributed to plant defence. Administration of JA and SA jointly mitigated the negative impact of at least one herbivore *H. virescens*, suggesting antipathic interaction between JA–SA pathway (Schweiger et al. 2014). In this regard, Wei et al. (2014) illustrated the temporal and dosage effects of cross-talk between JA–SA on host selection and egg depositing site selection behaviour of *Tetranychus urticae* towards *Phaseolus lunatus*. Behavioural alterations of mites in response to single or joint application of JA and SA in plants, resulted in attraction or repellence suggesting antagonistic interaction between JA and SA mediated response. Recent studies have revealed a mechanism wherein, SA binds to *NPR1* to suppress JA-responsive genes in cytoplasm and JA signalling within the nucleus (Nomoto et al. 2021). Also, *NPR1*-mediated *MYC2* inhibition within the nucleus plays a crucial role in mediating *NPR1*-mediated immunity against biotrophic or hemibiotrophic bacterial pathogen *P. synringae* pv. *maculicola* ES4326 (Nomoto et al. 2021). However, the exact molecular mechanism behind *NPR1*-mediated inhibition of *MYC2*-aided *transcriptiona*l activation remains unknown. *MYC2* has been documented to be the target of *ENHANCED DISEASE SUSCEPTIBILITY 1-PHYTOALEXIN DEFICIENT 4 (EDS1-PAD4)* to promote salicylic acid accumulation and signalling to boost immunity against *P. syringae* pv. *tomato* DC3000 (*Pto*) (Lapin et al. 2020). The binding of *NPR1* to promoters associated with JA-responsive genes through the *MYC2*-binding motif and TGA-regulated *WRKY*s suppresses the JA signalling cascade, imparting virulence (Yan et al. 2018).

Although the suppression of JA-responsive genes by SA has been documented, the pathway for inhibition of SA-responsive genes by JA has been scarcely studied. Exogenous application of SA inhibitor [e.g. 1-aminobenzotriazole (ABT)] helped to overcome witches broom symptoms in Chinese jujube by decreasing SA content and increasing amount of JA and MeJA attributing disease resistance after phytoplasma infection (Wang et al. 2022). Hou and Tsuda (2022) suggested that the JA-mediated suppression of SA occurs mainly during its biosynthesis and accumulation rather than hampering the signalling process. JA triggers the expression of *ANAC* genes (*ANAC019*, *ANAC055*, and *ANAC072*) via MYC2 and its homologs which suppresses the expression of SA biosynthesis gene *Isochorismate Synthase 1* (*ICS1*) and *Enhanced Disease Susceptibility 1* (*EDS1*) thereby decreasing accumulation of SA (Gao et al. 2022). The probable interactive mechanism between SA and JA/ET pathway involved in the defence mechanism has been illustrated in Fig. 4.

SA–JA interaction is multifaceted rather than simple mutual antagonism (De Vleesschauwer et al. 2014). Depending on the condition, SA–JA crosstalk may be synergistic or antagonistic. Genome-wide transcriptome analysis revealed additive and synergistic gene regulations by JA and SA in *Arabidopsis* in addition to antagonism (Hickman et al. 2019). Reports on instances of low content of SA and JA inducing synergistic effects whilst at elevated concentrations imparting antagonistic effects have also been referred (Mur et al. 2006). A meta-analysis based on transcriptome data revealed the co-induction of 363 genes by SA and JA under the influence of biotrophic and necrotrophic pathogens (Zhang et al. 2020). SA–JA crosstalk mediates two types of regulation—(i) tuneable and (ii) resilient (Hou and Tsuda 2022). During fine-tuning, SA and JA oppose each other to regulate immune responses within the infected cells. Antagonistic interaction activates either SA or JA signalling in various areas around the site of infection depending upon the necessity. On the other hand, when both SA and JA positively regulate immunity, resilience is imparted. De-repression of JA and loss of SA enhances JA signalling-mediated host immunity. JA induces expression of *EDS5* (SA biosynthesis gene), but simultaneously inhibits *PAD4*

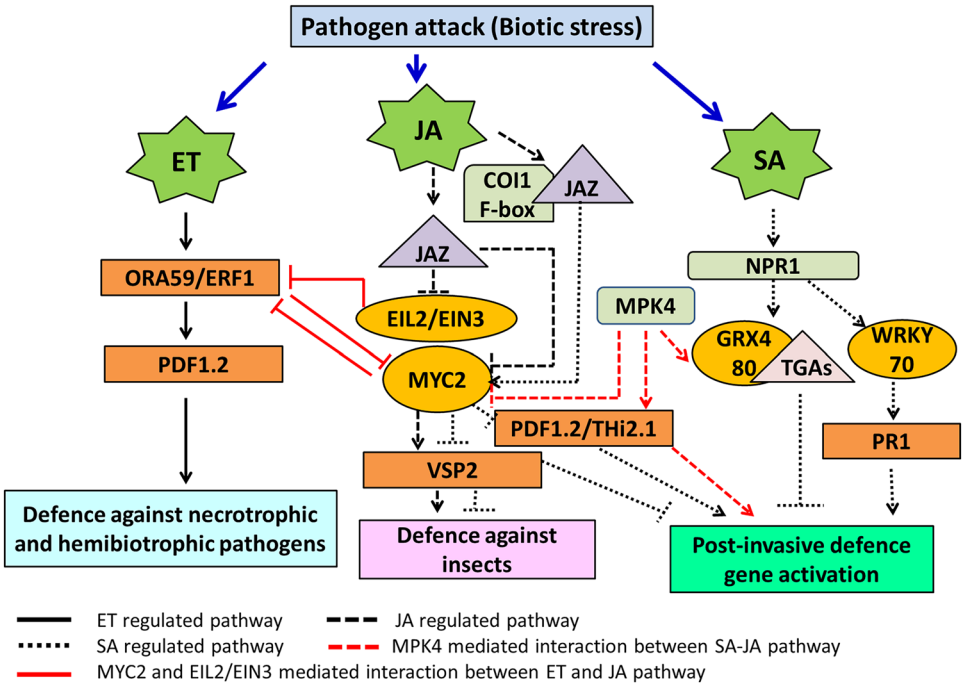

Fig. 4 Illustration of the potential interplay of JA with SA and ET pathways, contributing to the defence against pathogen infection and activation of virulence. MYC2 plays an important role in JA communication and interaction with JAZ repressors to free MYC2 from JAZ repression, allowing JA-mediated defence responses through the activation of MYC2. This is also positively regulated by ABA. In contrast, MYC2 suppresses SA modulation in response to other stresses. The JAZ inhibition of EIN mediates JA and ET signalling synergy in plant resistance, whereas interaction between MYC2 and EIN promotes JA and ET signalling. Abbreviations are defined in the text

expression, a positive regulator of *EDS5*. This inhibits the accumulation of SA within the system. Conversely, when *PAD4* function is disturbed at elevated temperatures or by pathogen attack, JA supports SA accumulation to minimize the negative impact of SA on plant growth and also confer SA-mediated immunity (Mine et al. 2018).

Although JA–SA crosstalk antagonistically promotes immune responses, it has often been exploited by pathogens to manifest infection. For example, pathogens susceptible to SA-mediated immune response alternatively switch on JA signalling to subdue SA-mediated immunity promoting virulence (Berens et al. 2017). Xin et al. (2018) explained the cardinal role of SA-signalling in mediating stomatal and apoplastic immunity as a means of plant defence upon *P. syringae* recognition. On the contrary, *Pto* and other *Pseudomonas* strains synthesize coronatine which structurally mimics JA-Ile to initiate invasion of stomata and virulence by turning on JA signalling. Effectors of *P. syringae* viz. *AvrB*, *HoBB1*, *HopZ1a* and *HoPX1* activate JA signalling resulting in the inhibition of SA signalling and MAPKs that might be beneficial against pathogens (Melotto et al. 2017; Yang et al. 2017). *Botrytis cinerea*, a necrotrophic fungus produces an exopolysaccharide that activates SA signalling and simultaneously inhibits the JA activation cascade in tomato (El Oirdi et al. 2011). JA–SA crosstalk exhibits plasticity and dynamic changes during the infection process by pathogens (Zhang et al. 2017b). Reports on the resistance of *A. thaliana* against various strains of *B. cinerea* were attributed to alternative signalling routes and crosstalk between JA and SA. On the contrary, upon pathogen exposure, when JA and SA signalling inhibit each other, either SA or JA gains ascendancy (Tsuda 2018). Instances of ETI viz., AvrRpt2-triggered and NLR RPS2-mediated ETI have also been documented to be induced by JA-SA signalling. In RPS2-ETI, SA promotes positive regulation of JA signalling contributing to RPS2-mediated ETI. The receptors associated with SA viz., *NPR3* and *NPR4* activate JA signalling by promoting JAZ degradation (Liu et al. 2016). Mutation studies have also revealed the cooperative role of JA and SA signalling during PTI and RPS2-mediated ETI (Hillmer et al. 2017). Microscopic studies aimed at spatial analysis of JA and SA activation revealed that JA–SA signalling is simultaneously turned on upon infection with *P. syringae* carrying AvrRpt2 (Betsuyaku et al. 2018). SA signalling was found to be activated near the site of infection, whilst JA signalling was activated outside the SA-activated area in the infected leaf (Betsuyaku et al. 2018). However, whether antagonistic interactions exist between JA and SA, and their spatial activation remains to be investigated.

Interaction Between JA and ET

The plant hormones such as ethylene exert a positive influence on the expression of JA-biosynthesis genes, contributing to the swift onset of JA production (Hu et al. 2021). Transcriptional activators ERFs15 and ERFs16 play pivotal roles in this process by activating key genes in JA biosynthesis, specifically *TomloxD*, *AOS*, and *OPR3*. Additionally, MYC2, which is activated by JA, along with ERF16, also undertakes the role of a transcriptional activator for

ERF16, leading to a pronounced surge in the expression of *ERF16*. ET-induced ERF15 and ERF16 emerge as potent drivers of transcription, triggering the abrupt escalation in JA levels during herbivore attacks. SA triggers the accumulation of TFs like *Ethylene Insensitive 3* (*EIN3*) in the ET signalling pathway which binds to another protein *ORA59* (*Octadecanoid-responsive Arabidopsis AP2/ERF domain protein 59*) in *Arabidopsis* to mediate proteasomal degradation (He et al. 2017a). JA and ET signalling co-ordinately regulate plant stress responses. *EIN3* and its homologue, *EIN-3 like 1* (*EIL1*) and JAZs-MYC2 of JA signalling cascade are involved in cross-talk between JA and ET (Zhang et al. 2014). Exogenously supplied JA initiates JAZ degradation and MYC2 release to regulate the expression of *ORA59/ERF1* that triggers *VSP2* to resist herbivory (Yang et al. 2019). In contrast, JAZ attenuates transcription of *EIL2/EIN3* within the ET pathway thereby activating downstream *ORA59/ERF1* that initiates expression of *PLANT DEFENSIN 1.2* (*PDF1.2*) and promotes defence against infection of hemibiotrophic pathogens and necrotrophic pathogens (Pieterse et al. 2012). Thus, the evidence shows that JA/ET and SA-mediated defence are mutually antagonistic.

Interaction Between JA and ABA

In recent years, the crosstalk between JA and ABA signalling pathways has been reported to monitor elicitor-induced reprogramming of plant metabolism and growth (Per et al. 2018). JAZs-MYC2 mediated coordination between JA and ABA signalling pathways regulate plant defence responses to insect-feeding herbivores inhibiting plant development (Chen et al. 2011). Pyrabactin resistance 1-like proteins (PYLs) are a family of ABA receptors that regulate metabolic reprogramming via the JA signalling pathway in *Arabidopsis* and tobacco (Yang et al. 2019). Yeast two-hybrid assays revealed that to resist herbivorous insect feeding, a complex is formed between ABA receptor PYL and JAZ to activate the transcription of MYC2, triggering the expression of JA-responsive gene *VSP2*, under the influence of *MED25*. Simultaneous to the said event, MYC2 suppresses the expression of *PTL1* and *PTL2* along with root growth. In addition, ABA induces the degradation of *JAZ12* which plays a crucial role in the interaction between JA and ABA signalling networks (Pauwels et al. 2015). Concomitant with such outcomes, a recent study by Liu et al. (2020) revealed that *VvPYL4* represses the transcriptional activities of *MYC2*, *JAZ* and *JAR1* which attributed to defensive responses to downy mildew in grapevine against *Plasmopara viticola*. Thus, coordinated interactions between JA and ABA signalling especially between JAZ-MYC2 and PYL regulate the balance between plant development and defence response.

Interaction of JA with Brassinosteroid (BR), Gibberellic Acid (GA) and Auxin (IAA)

An intricate balance between JA and BR signalling pathways helps to regulate plant growth and defence response. A low amount of BR induces *OsDI1* and *OsDWARF* expressions during the early and late stages of BR biosynthesis, respectively (Qureshi et al. 2023). At high concentrations of BR, expression of certain downstream genes, *BES1* and *BZR1* mediate abiotic stress tolerance. Nevertheless, high BR concentration also inhibits JA and BR biosynthesis, whilst on the other hand, JA also suppresses the biosynthesis of BR (Choudhary et al. 2012). In plant defence, BR antagonism with the JA pathway inhibits rice defence against root-knot nematodes (Nahar et al. 2013). He et al. (2020) suggested that rice *OsGSK2* [*glycogen synthase kinase 3 (GSK3)-like kinase*] suppresses BR signalling and enhances plant antiviral defences by directly destabilizing *OsJAZ4*, thereby turning on JA signalling. Thus, an intricate balance in the amount of JA and BR regulates plant growth and defence under pathogen attack.

Reports have revealed the relationship between JA and GA under both control and stressful conditions (Nawaz et al. 2023). Herbivore attack in *Camellia sinensis* exhibited JA–GA antagonistic crosstalk, wherein, the activity of defensive proteins, polyphenol oxidases (PPOs) were regulated by JA–GA crosstalk (Zhang et al. 2020). *CsPPO2* and *CsPPO4*-induced PPO activity resulted in elevated levels of JA production.

JA–auxin crosstalk regulates plant growth and development by regulating trade-offs with defence suppression (Gilroy and Breen 2022). Yang et al. (2019) reported that endogenously produced JA induces auxin accumulation due to increased expression of the auxin synthase gene (*ASA1*), whereas, *JAZ1* expression favoured growth of roots. In contrast, Chen et al. (2011) explained that the MYC2 mediated crosstalk between JA and auxin. MYC2 binds to the promoters of auxin-responsive gene PLT (plethora, responsible for stem cell niche maintenance and cell division), causing inhibition of its expression and root meristem activity. COI1 and MYC2 are the important constituents that participate in JA–auxin signalling networks (Wang et al. 2021). JA initiates complex formation between COI1 and JAZ aimed for degradation, thereby activating MYB21/MYB24 to trigger floral induction. ARF6/ARF8 regulates petal expansion and stamen growth in the auxin signalling pathway through modification of endogenous JA content and MYB21/MYB24 in the downstream of JAZ of JA signalling pathway co-ordinately regulate stamen and petal growth (Zhang et al. 2020). Additionally, leaf senescence is determined by JA–auxin cross-talk. Auxin retards leaf senescence, a physiological process induced by JA. Exogenous auxin prevents leaf senescence which is prevented by supplementation of

exogenous JA. JAZ4, JAZ8 and IAA29 are involved in the JA–auxin signalling cascade as negative regulators (Jiang et al. 2014). The functional role of JAZs suppression and WRKY57 negatively regulates JA-induced leaf senescence by competitively interacting with JAZ4, JAZ8 and IAA29 (Jiang et al. 2014). Although ample studies on JA–auxin interaction influencing various physiological statuses of plants have been conducted to date, studies on said interaction upon biotic stress exposure are hardly available to the best of our knowledge.

Exogenous Application of JA for Immune Boosting in Plants

Endogenous regulation of plant immune response by JA has led to the presumption of their application by exogenous means as an alternative mechanism of plant priming, thereby inducing plant defence against the major fungal, bacterial, and viral pathogens (Table 1). It has been observed that PR genes' expression was upregulated upon exogenous application of JA which further contributed to an augmented resistance against a wide range of pathogens. Specifically, MeJA leads to *Ralstonia solanacearum* resistance in legume *Medicago truncatula* (bacterial wilt disease through root colonization) (Yamchi et al. 2018). Exogenous MeJA application can upregulate JA-responsive genes by 25% during biotrophic rust fungus *Melampsora larici-populina* infection in poplar, but counteracting the endogenous SA level was reduced upon SA analogue (BTH) co-treatment (Ullah et al. 2019). However, the external application of MeJA has been doubted in some instances, particularly to control wheat powdery mildew [*Blumeria graminis tritici* (*Bgt*)] and justified with the application of JA biosynthesis inhibitor sodium diethyldithiocarbamate (DIECA) to suppress *Bgt* growth with upregulation of various PR genes (Li et al. 2020b). In addition to the exogenous priming through JA, glycerol application was found to upregulate the hormonal biosynthesis pathway (Ullah et al. 2019; Li et al. 2020b). Li et al. (2020c) claimed that glycerol is upregulating the JA biosynthetic pathway by augmenting lipoxygenase genes as well as the JAZ which confer to express PR genes and PR proteins for the defence against wheat powdery mildew. Similarly, pretreatment of JA can regulate the synthesis of potent plant volatiles like β-Cyclocitral which may confer bacterial blight resistance in rice against *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) through downregulating Abscisic Acid (ABA) biosynthesis (Taniguchi et al. 2023). Foliar application of JA with type-I ribosome-inactivating protein alpha-momorcharin (α-MMC) in tobacco has been proven to manipulate JA–SA crosstalk to govern resistance to tobacco mosaic virus (TMV; Yang et al. 2018). Yang et al. (2020) identified the precise role of JA treatment in rice for rice stripe virus (RSV) resistance, which is activated by the viral coat proteins (CPs) and the downstream interaction of JA-*Argonaute 18* (*AGO18*) regulated by JA-MYB TF. The effect of the exogenous application of JA on the immune system of the host also depends upon the type of plant tissue on which the treatment is applied. In the case of *R. solanacearum* mediated bacterial wild in *Medicago*, MeJA application onto the root tissues not only caused stunting growth of root, shoot and leaf but also an upregulation of *allene oxide synthase 1* (*AOS1*) gene involved in JA biosynthesis which causes a modification in the vessel structure thereby inhibiting bacterial and viral pathogen invasion through aphid attack and their systemic dissemination in distal host parts (Yamchi et al. 2018). A substantial decrease in plant productivity and dry matter was reported after JA foliar application in 3-week-old *Calendula officinalis* seedlings which elevated triterpenoid saponin production up to 86-fold in hairy root cultures (Rogowska et al. 2022). These classic examples provide ample reasons to highlight the differential roles of JA in different host tissues. In addition to the types of plant tissues, age-dependent plant growth stages also play a major role in plant immunity response upon external priming through JA. An earlier work on JA pretreatment in tomato revealed an increase in peroxidase (ROS scavenging enzyme in response to pathogen-infection aided ROS accumulation) activity with the plant age whilst the highest polyphenol oxidase activity was observed in young plant leaves (Cipollini and Redman 1999). Moreover, the exogenous application of JA has been demonstrated to trigger plant defence responses against RKN across various plant species, including tomato, soybean, and *Arabidopsis* (Soriano et al. 2004; Cooper et al. 2005; Fujimoto et al. 2011; Nahar et al. 2011; Zinovieva et al. 2013; Fan et al. 2015; Zhou et al. 2015b; Gleason et al. 2016; Hu et al. 2017). In the case of rice, resistance induced by MeJA was associated with an increased expression of genes involved in JA biosynthesis and defence (Nahar et al. 2011). The application of MeJA appears to render the nematode less capable of efficiently suppressing or counteracting plant defences (Fan et al. 2015). In rice, the application of external methyl JA led to an upregulation of the *OsPR1a* and *OsPR1b* genes during the early phase of *M. graminicola* infection, in turn, played a positive role in enhancing the systemic defence of rice against nematode parasitism (Nahar et al. 2011).

Conclusion and Future Prospects

This review has explored the complex molecular processes and complicated networking associated with JA-mediated immunity, providing insight into its crucial role in promoting disease resistance in plants against fungal, bacterial, viral, and nematode pathogens. Examining the

Table 1 JA-mediated priming of plant parts for improved immunity through upregulating genes' expression

Plant	Disease	Pathogen	Pathogen type	Conc. of JA	Plant parts treated	Effect on genes	Major functions	References
Kiwifruit (*Actinidia deliciosa*)	Bacterial canker	*Pseudomonas syringae* pv. *actinidiae*	Bacteria	1 mM	Whole plant	Superoxide dismutase, lipoxygenase	Defence and JA biosynthesis genes	da Silva et al. (2021)
Barrelclover (*Medicago truncatula*)	Bacterial wilt	*Ralstonia solanacearum*	Bacteria	0.1 mM	Roots, stem and leaves	AOS1, lipoxygenase, Kunitz proteinase inhibitor	JA biosynthesis	Yamchi et al. (2018)
Holy Basil (*Ocimum sanctum*), *Arabidopsis*	Necrotic lesions	*Pseudomonas syringae* pv. *tomato* PstDC3000	Bacteria	0.2 mM	–	*OscWRKY*	Phenylalanine ammonia lyase pathway	Joshi et al. (2022)
Rice (*Oryza sativa*)	Bacterial blight	*Xanthomonas oryzae pv. oryzae* (Xoo)	Bacteria	0.1 mM	Roots	Linalool synthase	JA biosynthesis	Taniguchi et al. (2014)
Rice (*Oryza sativa*)	Bacterial blight	*Xanthomonas oryzae pv. oryzae*	Bacteria	0.002–0.01 mM	Seeds	*OsGLP3-7*	JA pathway	Sun et al. (2023)
Wild banana (*Musa acuminate* ssp. *malaccensis*)	Sigatoka disease	*Pseudocercospora fijiensis*	Bacteria	0.1 mM	Leaf discs	*MaWRKY18*, *MaWRKY45*, *MaWRKY60*, *MaWRKY70*	Role in JA signalling	García-Laynes et al. (2022)
Alfalfa (*Medicago sativa*)	Root rot	*Fusarium oxysporum*	Fungus	100 mM	Roots	Superoxide dismutase, phenylalanine ammonia-lyase	Defence genes	Zhou et al. (2019)
Bread wheat (*Triticum aestivum* L.)	Spot blotch	*Bipolaris sorokiniana*	Fungus	3.3 mM	Seedlings	Catalase, peroxidase, phenylalanine ammonia-lyase, ascorbate peroxidase	Defence genes	Singh et al. (2019)
Kiwifruit (*Actinidia deliciosa* cv. *Jinkui*)	Soft rot	*Botryosphaeria dothidea*	Fungus	0.1 mM	fruit	Peroxidase, Superoxide dismutase, chitinase and B 1,3, glucanase	Defence genes	Pan et al. (2020)
Panax notoginseng	Root rot	*Fusarium solani*	Fungus	0.1 mM	Roots	Linoleate 13S-lipoxygenase, *AOC*, *AOS*, *TIFY*, defencin, and *PR*s	JA signalling pathway	Liu et al. (2019a)
Peach (*Prunus persica* L. *Batsch*)	Soft rot	*Rhizopus stolonifera*	Fungus	0.01 mM	Fruit	Phenylalanine ammonia-lyase (PAL) and 4-coumarateCoA ligase (4CL)	Defence genes	Ji et al. (2021)
Pine (*Pinus sylvestris* L.)	Root and butt rot	*Heterobasidion annosum*	Fungus	10 mM	Seedlings	Thaumatin like proteins (TLPs)	Pathogenesis related (PR) protein (members of PR-5 family)	Šņepste et al. (2021)

Table 1 (continued)

Plant	Disease	Pathogen	Pathogen type	Conc. of JA	Plant parts treated	Effect on genes	Major functions	References
Pine (*Pinus sylvestris* L.)	Needle cast	*Lophodermium seditiosum*	Fungus	10 mM	Seedlings	Thaumatin like proteins (TLPs)	Pathogenesis related (PR) protein (members of PR-5 family)	Šņepste et al. (2021)
Potato (*Solanum tuberosum*)	Late blight	*Phytophthora infestans* CN152	Oomycetes	1 mM	Whole plant	*AOS*, *lipoxygenase*, acyl-CoA oxidase ACX and Acyl-CoA, thioesterase (*ACOT*) genes, phytoalexin-deficient 4–2 protein (*PAD4*)	JA biosynthesis genes, hypersensitive response (HR) related genes	Yang et al. (2022)
Rice (*Oryza sativa*)	Rice blast	*Magnaporthe oryzae*	Fungus	0.1 mM	Seedlings	*OsOPR1a*, *OsOPR1b; OsJAZ8*, *OsJAZ9*, *OsJAZ10*; *OsAOS1, OsAOS2, OsAOC*	Defence, JA signalling, jasmonate biosynthesis genes	Ma et al. (2022)
Rose (*Rosa chinensis*) 'Old blush'	Grey mould	*Botrytis cinerea*	Fungus	100 mM	Leaf	*RcJAZ1*, *RcMYBX84, RcMYB123*	JAZ mediated signalling	Ren et al. (2020)
Tomato (*Solanum lycopersicum*)	Root-knot	*Meloidogyne incognita*	Nematode	100 µM	Leaf	*SlMYB2, SlMYB57, SlMYB78, SlMYB108, SlMYB112*	JA signalling pathways	Zhao et al. (2023b)
Arabidopsis	Root-knot	*Meloidogyne hapla*	Nematode	50 µM	Seedlings	*VSP2*	JA-responses and storage protein	Gleason et al. (2016)
Soybean (*Glycine max*)	Root-knot	*Meloidogyne hapla*	Nematode	500 µM	Leaf	*GmAOS1, GmAOS2, GmCHIB, GmLOX7*	JA biosynthesis genes	Hu et al. (2017)
Tomato (*Solanum lycopersicum*)	Root-knot	*Meloidogyne incognita*	Nematode	0.1 mM	Root	Proteinase inhibitor II (*PI II*) and Multicystatin (*MC*)	Plant defense mechanisms	Martínez-Medina et al. (2017)
Tomato (*Solanum lycopersicum*)	Root-knot	*Meloidogyne incognita*	Nematode	100 nM	Seeds	*SOD, CAT, POD, GST,* and *GPOX*	Defence-related responses	Bali et al. (2020)
Rice (*Oryza sativa*)	Ufra	*Ditylenchus angustus*	Nematode	100 µM	Shoots	*OsAOS2, OsJMT1, OsJAmyb*	Defence genes	Khanam et al. (2018)
Rice (*Oryza sativa*)	Root-gall	*Meloidogyne graminicola*	Nematode	100 µM	Seedlings	*OsPR1a, OsPR1b*	Defence genes	Nahar et al. (2011)
Rice (*Oryza sativa*)	Black streaked disease	Rice black streaked dwarf virus	Virus	0.05 mM	Entire seedlings	*OsLOXs, OsAOS2, OsJMT1*	JA biosynthesis genes	He et al. (2017b)

Table 1 (continued)

Plant	Disease	Pathogen	Pathogen type	Conc. of JA	Plant parts treated	Effect on genes	Major functions	References
Rice (*Oryza sativa*)	Stripe disease	Rice stripe virus (RSV)	Virus	0.01 and 0.1 mM	Entire seedlings	AGO18 promoter sequence and JAMYB transcription factor regulating AGO18	JA pathway	Yang et al. (2020)
Tobacco (*N. tabacum*) cv. *Samsun NN*	Mosaic disease	Tobacco mosaic virus (TMV)	Virus	0.3 mM	Leaf	*COI1*, *PDF1.2*	Induction of JA pathway, Pathogenesis related proteins	Yang et al. (2020)
Tomato (*Solanum lycopersicum*)	Yellow leaf curl disease	Yellow leaf curl virus	Virus	0.1 mM	Entire plants	*SlMAPK3*	JA pathway for enhanced defence	Li et al. (2017)

mechanisms of JA signalling, including the spatial and temporal features and interactions with other defence-responsive phytohormonal pathways, emphasises the intricate regulatory networks that contribute to a more comprehensive understanding of how plants adjust their defensive responses. As our comprehension of JA-mediated immunity becomes more profound, next investigations should prioritise the revelation of supplementary intricacies, such as the discovery of new JA-responsive genes, study of the dynamics of JA signalling in various plant species and CRISPR-mediated gene editing for those responsive genes' functional validation and knockout studies. Moreover, investigating the interaction between JA and other hormone pathways and the epigenetic control of JA responses might provide useful knowledge for the development of comprehensive approaches to enhance plant immunity. The use of omics technology and genetic techniques to boost JA responsiveness in crops has the potential to greatly transform agricultural practices, leading to more sustainable and resilient crop output. Furthermore, investigating the impact of environmental conditions on JA signalling and exploring plant–microbe interactions related to JA would enhance our understanding of the flexibility and variety of JA-mediated defences. In summary, further research in this area is expected to enhance our comprehension of plant immunity, providing novel strategies for safeguarding crops against changing biotic threats.

Here are some key questions for future JA research:

- How does plastid and peroxisome effectively deliver the synthesized JA to downstream effectors and cellular compartments?
- How can the JA-binding affinity to cell receptors be improved molecularly?
- Are there any new JA-responsive genes in plants and their functional validation through CRISPR-mediated editing?
- How to establish JA-mediated immunity research in field crops (in cereals, legumes, etc.) for in vivo application?
- What is the precise way by which JA modulates SAR and ISR?

Acknowledgements The authors are thankful to the Agricultural Research Organization (ARO-Volcani Center, Israel) for research support. The authors are also thankful to the editors and anonymous reviewers for their critical comments to improve the manuscript. We apologize to those researchers whose relevant research publications are not cited in this manuscript due to the space limitation in the present form.

Author Contributions RR conceptualized and supervised the work; RR, AH, SB, SM, MRP, SPD and SR contributed in writing to make the draft manuscript; AK, US and SM—reviewed and edited the manuscript; RR, AH, and SB made the figure illustrations; All authors have read the final version of the manuscript and agreed to its submission in the journal for possible publication.

Declarations

Conflict of interest The authors declare no conflict of interest.

References

Abdul Malik NA, Kumar IS, Nadarajah K (2020) Elicitor and receptor molecules: orchestrators of plant defense and immunity. Int J Mol Sci 21:963

Aerts N, Pereira Mendes M, Van Wees SCM (2021) Multiple levels of crosstalk in hormone networks regulating plant defense. Plant J 105:489–504

Agrawal GK, Tamogami S, Iwahashi H et al (2003) Transient regulation of jasmonic acid-inducible rice MAP kinase gene (*OsBWMK1*) by diverse biotic and abiotic stresses. Plant Physiol Biochem 41:355–361

Alariqi M, Ramadan M, Wang Q et al (2023) Cotton *4-coumarate-CoA ligase 3* enhanced plant resistance to *Verticillium dahliae* by promoting jasmonic acid signaling-mediated vascular lignification and metabolic flux. Plant J 115:190–204

Albers P, Üstün S, Witzel K et al (2019) A remorin from *Nicotiana benthamiana* interacts with the *Pseudomonas* type-III effector protein HopZ1a and is phosphorylated by the immune-related kinase PBS1. Mol Plant-Microbe Interact 32:1229–1242

Aleman GHJ, Thirumalaikumar VP, Jander G et al (2022) OPDA, more than just a jasmonate precursor. Phytochemistry 204:113432

Ali S, Ganai BA, Kamili AN et al (2018) Pathogenesis-related proteins and peptides as promising tools for engineering plants with multiple stress tolerance. Microbiol Res 212:29–37

Anderson JP, Badruzsaufari E, Schenk PM et al (2004) Antagonistic interaction between abscisic acid and jasmonate-ethylene signaling pathways modulates defense gene expression and disease resistance in *Arabidopsis*. Plant Cell 16:3460–3479

Antico CJ, Colon C, Banks T, Ramonell KM (2012) Insights into the role of jasmonic acid-mediated defenses against necrotrophic and biotrophic fungal pathogens. Front Biol (beijing) 7:48–56

Attaran E, Zeier TE, Griebel T, Zeier J (2009) Methyl salicylate production and jasmonate signaling are not essential for systemic acquired resistance in *Arabidopsis*. Plant Cell 21:954–971

Baldwin IT (2018) The jasmonate cascade and the complexity of induced defence against herbivore attack. Annu Plant Rev Online 158–188

Bali S, Kaur P, Jamwal VL et al (2020) Seed priming with jasmonic acid counteracts root knot nematode infection in tomato by modulating the activity and expression of antioxidative enzymes. Biomolecules 10(1):98

Bender CL, Alarcón-Chaidez F, Gross DC (1999) *Pseudomonas syringae* phytotoxins: mode of action, regulation, and biosynthesis by peptide and polyketide synthetases. Microbiol Mol Biol Rev 63:266–292

Berens ML, Berry HM, Mine A et al (2017) Evolution of hormone signaling networks in plant defense. Annu Rev Phytopathol 55:401–425

Betsuyaku S, Katou S, Takebayashi Y et al (2018) Salicylic acid and jasmonic acid pathways are activated in spatially different domains around the infection site during effector-triggered immunity in *Arabidopsis thaliana*. Plant Cell Physiol 59:8–16

Bhattarai KK, Xie QG, Mantelin S et al (2008) Tomato susceptibility to root-knot nematodes requires an intact jasmonic acid signaling pathway. Mol Plant-Microbe Interact 21:1205–1214

Bosch M, Wright LP, Gershenzon J et al (2014) Jasmonic acid and its precursor 12-oxophytodienoic acid control different aspects of constitutive and induced herbivore defenses in tomato. Plant Physiol 166:396–410

Bürger M, Chory J (2019) Stressed out about hormones: how plants orchestrate immunity. Cell Host Microbe 26:163–172

Campos ML, Kang J-H, Howe GA (2014) Jasmonate-triggered plant immunity. J Chem Ecol 40:657–675

Campos ML, Yoshida Y, Major IT et al (2016) Rewiring of jasmonate and phytochrome B signalling uncouples plant growth–defense tradeoffs. Nat Commun 7:12570

Cao Y, Liu L, Ma K et al (2022) The jasmonate-induced bHLH gene SlJIG functions in terpene biosynthesis and resistance to insects and fungus. J Integr Plant Biol 64:1102–1115

Chakraborty S, Pattanayak A, Mandal S et al (2014) An overview of climate change: causes, trends and implications. In: Crop improvement in the era of climate change. IK International Publishing House, New Delhi, pp 1–29

Chaturvedi R, Krothapalli K, Makandar R et al (2008) Plastid ω3-fatty acid desaturase-dependent accumulation of a systemic acquired resistance inducing activity in petiole exudates of *Arabidopsis thaliana* is independent of jasmonic acid. Plant J 54:106–117

Chen H, Wilkerson CG, Kuchar JA et al (2005) Jasmonate-inducible plant enzymes degrade essential amino acids in the herbivore midgut. Proc Natl Acad Sci USA 102:19237–19242

Chen Q, Sun J, Zhai Q et al (2011) The basic helix–loop–helix transcription factor MYC2 directly represses *PLETHORA* expression during jasmonate-mediated modulation of the root stem cell niche in *Arabidopsis*. Plant Cell 23:3335–3352

Chini A, Gimenez-Ibanez S, Goossens A, Solano R (2016) Redundancy and specificity in jasmonate signalling. Curr Opin Plant Biol 33:147–156

Choudhary SP, Yu J-Q, Yamaguchi-Shinozaki K et al (2012) Benefits of brassinosteroid crosstalk. Trends Plant Sci 17:594–605

Chung HS, Koo AJK, Gao X et al (2008) Regulation and function of *Arabidopsis JASMONATE ZIM-domain* genes in response to wounding and herbivory. Plant Physiol 146:952–964

Cipollini DF, Redman AM (1999) Age-dependent effects of jasmonic acid treatment and wind exposure on foliar oxidase activity and insect resistance in tomato. J Chem Ecol 25:271–281

Cooper WR, Jia L, Goggin L (2005) Effects of jasmonate-induced defenses on root-knot nematode infection of resistant and susceptible tomato cultivars. J Chem Ecol 31:1953–1967

da Silva MN, Vasconcelos MW, Pinto V et al (2021) Role of methyl jasmonate and salicylic acid in kiwifruit plants further subjected to *Psa* infection: biochemical and genetic responses. Plant Physiol Biochem 162:258–266

De Kesel J, Conrath U, Flors V et al (2021) The induced resistance lexicon: do's and don'ts. Trends Plant Sci 26:685–691

De Vleesschauwer D, Xu J, Höfte M (2014) Making sense of hormone-mediated defense networking: from rice to *Arabidopsis*. Front Plant Sci 5:611

Denno RF, Kaplan I (2007) Plant-mediated interactions in herbivorous insects: mechanisms, symmetry, and challenging the paradigms of competition past. In: Ecological communities: plant mediation in indirect interaction webs. Cambridge University Press, Cambridge, pp 19–50

Dombrecht B, Xue GP, Sprague SJ et al (2007) MYC2 differentially modulates diverse jasmonate-dependent functions in *Arabidopsis*. Plant Cell 19:2225–2245

Dubois M, Van den Broeck L, Inzé D (2018) The pivotal role of ethylene in plant growth. Trends Plant Sci 23:311–323

El Oirdi M, El Rahman TA, Rigano L et al (2011) *Botrytis cinerea* manipulates the antagonistic effects between immune pathways to promote disease development in tomato. Plant Cell 23:2405–2421

Falk KL, Kästner J, Bodenhausen N et al (2014) The role of glucosinolates and the jasmonic acid pathway in resistance of

Arabidopsis thaliana against molluscan herbivores. Mol Ecol 23:1188–1203

Fan JW, Hu CL, Zhang LN et al (2015) Jasmonic acid mediates tomato's response to root knot nematodes. J Plant Growth Regul 34:196–205

Farmer EE, Dubugnon L (2009) Detritivorous crustaceans become herbivores on jasmonate-deficient plants. Proc Natl Acad Sci USA 106:935–940

Fu ZQ, Dong X (2013) Systemic acquired resistance: turning local infection into global defense. Annu Rev Plant Biol 64:839–863

Fu X, Li J, Min D et al (2022) LncRNA4504 involved in methyl jasmonate-induced resistance to *Botrytis cinerea* in postharvest tomato fruit. Sci Hortic (amst) 305:111381

Fujimoto T, Tomitaka Y, Abe H et al (2011) Expression profile of jasmonic acid-induced genes and the induced resistance against the root-knot nematode (*Meloidogyne incognita*) in tomato plants (*Solanum lycopersicum*) after foliar treatment with methyl jasmonate. Plant Physiol 168:1084–1097

Gangappa SN, Srivastava AK, Maurya JP et al (2013) Z-box binding transcription factors (ZBFs): a new class of transcription factors in *Arabidopsis* seedling development. Mol Plant 6:1758–1768

Gao X, Starr J, Gobel C et al (2008) Maize 9-lipoxygenase *ZmLOX3* controls development, root-specific expression of defense genes, and resistance to root-knot nematodes. Mol Plant-Microbe Interact 21:98–109

Gao Y, Li Z, Yang C et al (2022) *Pseudomonas syringae* activates *ZAT18* to inhibit salicylic acid accumulation by repressing *EDS1* transcription for bacterial infection. N Phytol 233:1274–1288

García-Laynes S, Herrera-Valencia VA, Tamayo-Torres LG et al (2022) The banana *MaWRKY18*, *MaWRKY45*, *MaWRKY60* and *MaWRKY70* genes encode functional transcription factors and display differential expression in response to defense phytohormones. Genes (Basel) 13:1891

Geng X, Cheng J, Gangadharan A, Mackey D (2012) The coronatine toxin of *Pseudomonas syringae* is a multifunctional suppressor of *Arabidopsis* defense. Plant Cell 24:4763–4774

Gheysen G, Mitchum MG (2019) Phytoparasitic nematode control of plant hormone pathways. Plant Physiol 179:1212–1226

Ghorbel M, Brini F, Sharma A, Landi M (2021) Role of jasmonic acid in plants: the molecular point of view. Plant Cell Rep 40:1471–1494

Gilroy E, Breen S (2022) Interplay between phytohormone signalling pathways in plant defence—other than salicylic acid and jasmonic acid. Essays Biochem 66:657–671

Gimenez-Ibanez S, Boter M, Solano R (2015) Novel players fine-tune plant trade-offs. Essays Biochem 58:83–100

Gimenez-Ibanez S, Boter M, Ortigosa A et al (2017) JAZ 2 controls stomata dynamics during bacterial invasion. N Phytol 213:1378–1392

Gleason C, Leelarasamee N, Meldau D, Feussner I (2016) OPDA has key role in regulating plant susceptibility to the root-knot nematode *Meloidogyne hapla* in *Arabidopsis*. Front Plant Sci 7:e1565

Goossens J, Swinnen G, Vanden Bossche R et al (2015) Change of a conserved amino acid in the MYC 2 and MYC 3 transcription factors leads to release of *JAZ* repression and increased activity. N Phytol 206:1229–1237

Harris FM, Mou Z (2023) Damage-associated molecular patterns and systemic signaling. Phytopathology 114(2):308–327

Havko NE, Major IT, Jewell JB et al (2016) Control of carbon assimilation and partitioning by jasmonate: an accounting of growth–defense tradeoffs. Plants 5:7

He P, Chintamanani S, Chen Z et al (2004) Activation of a COI1-dependent pathway in *Arabidopsis* by *Pseudomonas syringae* type III effectors and coronatine. Plant J 37:589–602

He X, Jiang J, Wang C, Dehesh K (2017a) *ORA59* and *EIN3* interaction couples jasmonate-ethylene synergistic action to antagonistic salicylic acid regulation of *PDF* expression. J Integr Plant Biol 59:275–287

He Y, Zhang H, Sun Z et al (2017b) Jasmonic acid-mediated defense suppresses brassinosteroid-mediated susceptibility to Rice black streaked dwarf virus infection in rice. N Phytol 214:388–399

He Y, Han J, Liu R et al (2018) Integrated transcriptomic and metabolomic analyses of a wax deficient citrus mutant exhibiting jasmonic acid-mediated defense against fungal pathogens. Hortic Res 5:43

He Y, Hong G, Zhang H et al (2020) The *OsGSK2* kinase integrates brassinosteroid and jasmonic acid signaling by interacting with *OsJAZ4*. Plant Cell 32:2806–2822

Heim MA, Jakoby M, Werber M et al (2003) The basic helix–loop–helix transcription factor family in plants: a genome-wide study of protein structure and functional diversity. Mol Biol Evol 20:735–747

Hewedy OA, Elsheery NI, Karkour AM et al (2023) Jasmonic acid regulates plant development and orchestrates stress response during tough times. Environ Exp Bot 208:105260

Hickman R, Mendes MP, Van Verk MC et al (2019) Transcriptional dynamics of the salicylic acid response and its interplay with the jasmonic acid pathway. BioRxiv 742742

Hillmer RA, Tsuda K, Rallapalli G et al (2017) The highly buffered *Arabidopsis* immune signaling network conceals the functions of its components. PLoS Genet 13:e1006639

Hou S, Tsuda K (2022) Salicylic acid and jasmonic acid crosstalk in plant immunity. Essays Biochem 66:647–656

Hou Y, Wang Y, Tang L et al (2019) SAPK10-mediated phosphorylation on *WRKY72* releases its suppression on jasmonic acid biosynthesis and bacterial blight resistance. Iscience 16:499–510

Hu YF, You J, Li CJH et al (2017) Exogenous application of methyl jasmonate induces defence against *Meloidogyne hapla* in soybean. Nematology 19:293–304

Hu C, Wei C, Ma Q et al (2021) Ethylene response factors 15 and 16 trigger jasmonate biosynthesis in tomato during herbivore resistance. Plant Physiol 185:1182–1197

Huang H, Liu B, Liu L, Song S (2017) Jasmonate action in plant growth and development. J Exp Bot 68:1349–1359

Huang Z, Xue Z, Zhao X et al (2023) Transcription factors, potential regulatory targets in fruit defense responses to pathogens. Postharvest Biol Technol 206:112589

Ibrahim HM, Hosseini P, Alkharouf NW et al (2011). Analysis of gene expression in soybean (*Glycine max*) roots in response to the root knot nematode *Meloidogyne incognita* using microarrays and KEGG pathways. BMC Genom 12:1–6

Inagaki H, Hayashi K, Takaoka Y et al (2023) Genome editing reveals both the crucial role of *OsCOI2* in jasmonate signaling and the functional diversity of *COI1* homologs in rice. Plant Cell Physiol 64(4):405–421

Jagodzik P, Tajdel-Zielinska M, Ciesla A et al (2018) Mitogen-activated protein kinase cascades in plant hormone signaling. Front Plant Sci 9:1387

Jaiti F, Verdeil JL, El Hadrami I (2009) Effect of jasmonic acid on the induction of polyphenoloxidase and peroxidase activities in relation to date palm resistance against *Fusarium oxysporum* f. sp. *albedinis*. Physiol Mol Plant Pathol 74:84–90

Jang G, Yoon Y, Do CY (2020) Crosstalk with jasmonic acid integrates multiple responses in plant development. Int J Mol Sci 21:305

Jeong M-A, Jeong R-D (2013) Resistance protein-mediated defense signalling in response to Turnip Crinkle Virus in *Arabidopsis*: recent advances. J Plant Dis Prot 120:97–104

Ji N, Wang J, Li Y et al (2021) Involvement of *PpWRKY70* in the methyl jasmonate primed disease resistance against *Rhizopus stolonifer* of peaches via activating phenylpropanoid pathway. Postharvest Biol Technol 174:111466

Jiang S, Yao J, Ma K-W et al (2013) Bacterial effector activates jasmonate signaling by directly targeting JAZ transcriptional repressors. PLoS Pathog 9:e1003715

Jiang Y, Liang G, Yang S, Yu D (2014) *Arabidopsis WRKY57* functions as a node of convergence for jasmonic acid- and auxin-mediated signaling in jasmonic acid-induced leaf senescence. Plant Cell 26:230–245

Jin H, Zhu Z (2017) Temporal and spatial view of jasmonate signaling. Trends Plant Sci 22:451–454

Jones JDG, Dangl JL (2006) The plant immune system. Nature 444:323–329

Joshi A, Jeena GS, Shikha et al (2022) *Ocimum sanctum*, *OscWRKY1*, regulates phenylpropanoid pathway genes and promotes resistance to pathogen infection in *Arabidopsis*. Plant Mol Biol 110:235–251

Kachroo P, Kachroo A (2012) The roles of salicylic acid and jasmonic acid in plant immunity. In: Molecular plant immunity. Wiley, New York, pp 55–79

Kamiya Y (2010) Plant hormones: versatile regulators of plant growth and development. Annu Rev Plant Biol. https://doi.org/10.1146/annurev.arplant.61.031110.100001

Kammerhofer N, Radakovic Z, Regis JMA et al (2015) Role of stress-related hormones in plant defence during early infection of the cyst nematode *Heterodera schachtii* in *Arabidopsis*. N Phytol 207:778–789

Karpets YV, Kolupaev YE, Lugovaya AA, Oboznyi AI (2014) Effect of jasmonic acid on the pro-/antioxidant system of wheat coleoptiles as related to hyperthermia tolerance. Russ J Plant Physiol 61:339–346

Katsir L, Schilmiller AL, Staswick PE et al (2008) COI1 is a critical component of a receptor for jasmonate and the bacterial virulence factor coronatine. Proc Natl Acad Sci USA 105:7100–7105

Kaya C, Ugurlar F, Ashraf M, Ahmad P (2023) Salicylic acid interacts with other plant growth regulators and signal molecules in response to stressful environments in plants. Plant Physiol Biochem 196:431–443

Kazan K, Manners JM (2013) MYC2: the master in action. Mol Plant 6:686–703

Khan MIR, Singh A, Poor P (2023) Plant hormones in crop improvement. Elsevier, Amsterdam

Khanam S, Bauters L, Singh RR et al (2018) Mechanisms of resistance in the rice cultivar Manikpukhat the rice stem nematode *Ditylenchus angustus*. Mol Plant Pathol 19:1391–1402

Kottakota C, Pradhan B, Roychowdhury R, Dubey VK (2021) Improvement of wheat (*Triticum* spp.) through genetic manipulation. In: Genetically modified crops: current status, prospects and challenges, vol 1. Springer, Singapore, pp 33–66

Lalotra S, Hemantaranjan A, Yashu BR et al (2020) Jasmonates: an emerging approach in biotic and abiotic stress tolerance. In: Plant science—structure, anatomy and physiology in plants cultured in vivo in vitro. IntechOpen, London

Lapin D, Bhandari DD, Parker JE (2020) Origins and immunity networking functions of EDS1 family proteins. Annu Rev Phytopathol 58:253–276

Laura B, Silvia P, Francesca F et al (2018) Epigenetic control of defense genes following MeJA-induced priming in rice (*O. sativa*). J Plant Physiol 228:166–177

Li L, Li C, Lee GI, Howe GA (2002) Distinct roles for jasmonate synthesis and action in the systemic wound response of tomato. Proc Natl Acad Sci USA 99:6416–6421

Li Y, Qin L, Zhao J et al (2017) SlMAPK3 enhances tolerance to tomato yellow leaf curl virus (TYLCV) by regulating salicylic acid and jasmonic acid signaling in tomato (*Solanum lycopersicum*). PLoS ONE 12:e0172466

Li N, Han X, Feng D et al (2019) Signaling crosstalk between salicylic acid and ethylene/jasmonate in plant defense: do we understand what they are whispering? Int J Mol Sci 20:671

Li R, Wang L, Li Y et al (2020a) Knockout of *SlNPR1* enhances tomato plants resistance against *Botrytis cinerea* by modulating ROS homeostasis and JA/ET signaling pathways. Physiol Plant 170(4):569–579

Li Y, Qiu L, Liu X et al (2020b) Glycerol-induced powdery mildew resistance in wheat by regulating plant fatty acid metabolism, plant hormones cross-talk, and pathogenesis-related genes. Int J Mol Sci 21:673

Li Y, Qiu L, Zhang Q et al (2020c) Exogenous sodium diethyldithiocarbamate, a Jasmonic acid biosynthesis inhibitor, induced resistance to powdery mildew in wheat. Plant Direct 4:e00212

Li C, Xu M, Cai X et al (2022) Jasmonate signaling pathway modulates plant defense, growth, and their trade-offs. Int J Mol Sci 23:3945

Li Y, Roychowdhury R, Govta L et al (2023) Intracellular reactive oxygen species-aided localized cell death contributing to immune responses against wheat powdery mildew pathogen. Phytopathology 113(5):884–892

Lim G-H, Shine MB, de Lorenzo L et al (2016) Plasmodesmata localizing proteins regulate transport and signaling during systemic acquired immunity in plants. Cell Host Microbe 19:541–549

Liu H, Timko MP (2021) Jasmonic acid signaling and molecular crosstalk with other phytohormones. Int J Mol Sci 22:2914

Liu L, Sonbol F-M, Huot B et al (2016) Salicylic acid receptors activate jasmonic acid signalling through a non-canonical pathway to promote effector-triggered immunity. Nat Commun 7:13099

Liu D, Zhao Q, Cui X et al (2019a) A transcriptome analysis uncovers *Panax notoginseng* resistance to *Fusarium solani* induced by methyl jasmonate. Genes Genomics 41:1383–1396

Liu Y, Du M, Deng L et al (2019b) MYC2 regulates the termination of jasmonate signaling via an autoregulatory negative feedback loop. Plant Cell 31:106–127

Liu L, Liu C-Y, Wang H et al (2020) The abscisic acid receptor gene *VvPYL4* positively regulates grapevine resistance to *Plasmopara viticola*. Plant Cell Tissue Organ Cult 142:483–492

Liu Y, Yu Y, Fei S et al (2023) Overexpression of *Sly-miR398b* compromises disease resistance against *Botrytis cinerea* through regulating ROS homeostasis and JA-related defense genes in tomato. Plants 12:2572

Luna E, Bruce TJA, Roberts MR et al (2012) Next-generation systemic acquired resistance. Plant Physiol 158:844–853

Ma L, Sun Y, Ruan X et al (2021) Genome-wide characterization of jasmonates signaling components reveals the essential role of *ZmCOI1a-ZmJAZ15* action module in regulating maize immunity to gibberella stalk rot. Int J Mol Sci 22(2):870

Ma J, Morel J-B, Riemann M, Nick P (2022) Jasmonic acid contributes to rice resistance against *Magnaporthe oryzae*. BMC Plant Biol 22:601

Machado RAR, McClure M, Herve MR et al (2016) Benefits of jasmonate-dependent defenses against vertebrate herbivores in nature. Elife 5:e13720

Major IT, Yoshida Y, Campos ML et al (2017) Regulation of growth–defense balance by the JASMONATE ZIM-DOMAIN (JAZ)-MYC transcriptional module. N Phytol 215:1533–1547

Maksimov IV, Sorokan AV, Chereoanova EA et al (2011) Effects of salicylic and jasmonic acids on the components of pro/antioxidant system in potato plants infected with late blight. Russ J Plant Physiol 58:299–306

Mao Y-B, Liu Y-Q, Chen D-Y et al (2017) Jasmonate response decay and defense metabolite accumulation contributes to age-regulated dynamics of plant insect resistance. Nat Commun 8:13925

Martínez-Medina A, Fernandez I, Lok GB et al (2017) Shifting from priming of salicylic acid-to jasmonic acid-regulated defences

by *Trichoderma* protects tomato against the root knot nematode *Meloidogyne incognita*. N Phytol 213(3):1363–1377
Melotto M, Zhang L, Oblessuc PR, He SY (2017) Stomatal defense a decade later. Plant Physiol 174:561–571
Mène-Saffrané L, Esquerré-Tugayé M-T, Fournier J (2003) Constitutive expression of an inducible lipoxygenase in transgenic tobacco decreases susceptibility to *Phytophthora parasitica* var. *nicotianae*. Mol Breed 12:271–282
Millet YA, Danna CH, Clay NK et al (2010) Innate immune responses activated in *Arabidopsis* roots by microbe-associated molecular patterns. Plant Cell 22:973–990
Mine A, Seyfferth C, Kracher B et al (2018) The defense phytohormone signaling network enables rapid, high-amplitude transcriptional reprogramming during effector-triggered immunity. Plant Cell 30:1199–1219
Mur LAJ, Kenton P, Atzorn R et al (2006) The outcomes of concentration-specific interactions between salicylate and jasmonate signaling include synergy, antagonism, and oxidative stress leading to cell death. Plant Physiol 140:249–262
Nahar K, Kyndt T, De Vleesschauwer D et al (2011) The jasmonate pathway is a key player in systemically induced defense against root knot nematodes in rice. Plant Physiol 157:305–316
Nahar K, Kyndt T, Hause B et al (2013) Brassinosteroids suppress rice defense against root-knot nematodes through antagonism with the jasmonate pathway. Mol Plant-Microbe Interact 26:106–115
Naor N, Gurung FB, Ozalvo R et al (2018) Tight regulation of *allene oxide synthase* (*AOS*) and *allene oxide cyclase-3* (*AOC3*) promote *Arabidopsis* susceptibility to the root-knot nematode *Meloidogyne javanica*. Eur J Plant Pathol 150:149–165
Nawaz H, Irum A, Nasim W et al (2023) Hormonal cross-talk mechanisms and plant immunity or defense: an overview. In: Hormonal cross-talk, plant defense and development. Elsevier, Amsterdam, pp 1–12
Nomoto M, Skelly MJ, Itaya T et al (2021) Suppression of MYC transcription activators by the immune cofactor NPR1 fine-tunes plant immune responses. Cell Rep 37(11):110125
Okada K, Abe H, Arimura G (2015) Jasmonates induce both defense responses and communication in monocotyledonous and dicotyledonous plants. Plant Cell Physiol 56:16–27
Orrock JL, Connolly BM, Choi W-G et al (2018) Plants eavesdrop on cues produced by snails and induce costly defenses that affect insect herbivores. Oecologia 186:703–710
Ortigosa A, Gimenez-Ibanez S, Leonhardt N et al (2018) Design of a bacterial speck resistant tomato by CRISPR/Cas9-mediated editing of *SlJAZ2*. Plant Biotechnol J 17:665–673
Pan L, Zhao X, Chen M et al (2020) Effect of exogenous methyl jasmonate treatment on disease resistance of postharvest kiwifruit. Food Chem 305:125483
Pan L, Chen X, Xu W et al (2022) Methyl jasmonate induces postharvest disease resistance to decay caused by *Alternaria alternata* in sweet cherry fruit. Sci Hortic (amst) 292:110624
Pandey SP, Somssich IE (2009) The role of WRKY transcription factors in plant immunity. Plant Physiol 150:1648–1655
Pandey D, Rajendran SRCK, Gaur M et al (2016) Plant defense signaling and responses against necrotrophic fungal pathogens. J Plant Growth Regul 35:1159–1174
Pandita D (2022) Jasmonates: key players in plant stress tolerance. In: Emerging plant growth regulators in agriculture. Elsevier, Amsterdam, pp 165–192
Patkar RN, Benke PI, Qu Z et al (2015) A fungal monooxygenase-derived jasmonate attenuates host innate immunity. Nat Chem Biol 11:733–740
Pauwels L, Goossens A (2011) The JAZ proteins: a crucial interface in the jasmonate signaling cascade. Plant Cell 23:3089–3100
Pauwels L, Ritter A, Goossens J et al (2015) The ring e3 ligase keep on going modulates jasmonate zim-domain12 stability. Plant Physiol 169:1405–1417
Per TS, Khan MIR, Anjum NA et al (2018) Jasmonates in plants under abiotic stresses: crosstalk with other phytohormones matters. Environ Exp Bot 145:104–120
Pieterse CMJ, Van der Does D, Zamioudis C et al (2012) Hormonal modulation of plant immunity. Annu Rev Cell Dev Biol 28:489–521
Pozo MJ, Van Der Ent S, Van Loon LC, Pieterse CMJ (2008) Transcription factor MYC2 is involved in priming for enhanced defense during rhizobacteria-induced systemic resistance in *Arabidopsis thaliana*. N Phytol 180:511–523
Pradhan M, Pandey P, Baldwin IT, Pandey SP (2020) Argonaute4 modulates resistance to *Fusarium brachygibbosum* infection by regulating jasmonic acid signaling. Plant Physiol 184:1128–1152
Prasad A, Sharma S, Prasad M (2023) Post translational modifications at the verge of plant–geminivirus interaction. Biochim Biophys Acta Gene Regul Mech 1866:94983
Proietti S, Bertini L, Timperio AM et al (2013) Crosstalk between salicylic acid and jasmonate in *Arabidopsis* investigated by an integrated proteomic and transcriptomic approach. Mol Biosyst 9:1169–1187
Qureshi FF, Rasheed R, Hussain I, Ashraf MA (2023) Signaling crosstalk between brassinosteroids and jasmonates in plant defense, growth, and development. In: Hormonal cross-talk, plant defense and development. Elsevier, Amsterdam, pp 123–148
Rao S, Tian Y, Zhang C et al (2023) The JASMONATE ZIM-domain-OPEN STOMATA1 cascade integrates jasmonic acid and abscisic acid signaling to regulate drought tolerance by mediating stomatal closure in poplar. J Exp Bot 74:443–457
Rasmann S, De Vos M, Casteel CL et al (2012) Herbivory in the previous generation primes plants for enhanced insect resistance. Plant Physiol 158:854–863
Ren H, Bai M, Sun J et al (2020) *RcMYB84* and *RcMYB123* mediate jasmonate-induced defense responses against *Botrytis cinerea* in rose (*Rosa chinensis*). Plant J 103:1839–1849
Reymond P, Farmer EE (1998) Jasmonate and salicylate as global signals for defense gene expression. Curr Opin Plant Biol 1:404–411
Rogowska A, Stpiczyńska M, Pączkowski C, Szakiel A (2022) The influence of exogenous jasmonic acid on the biosynthesis of steroids and triterpenoids in *Calendula officinalis* plants and hairy root culture. Int J Mol Sci 23:12173
Romanazzi G, Sanzani SM, Bi Y et al (2016) Induced resistance to control postharvest decay of fruit and vegetables. Postharvest Biol Technol 122:82–94
Roychowdhury R (2014) Crop improvement in the era of climate change. IK International Publishers, New Delhi
Roychowdhury R, Choudhury S, Hasanuzzaman M, Srivastava S (2020) Sustainable agriculture in the era of climate change. Springer, Cham
Ruan J, Zhou Y, Zhou M et al (2019) Jasmonic acid signaling pathway in plants. Int J Mol Sci 20:2479
Scalschi L, Sanmartín M, Camañes G et al (2015) Silencing of *OPR3* in tomato reveals the role of *OPDA* in callose deposition during the activation of defense responses against *Botrytis cinerea*. Plant J 81:304–315
Schilmiller AL, Koo AJK, Howe GA (2007) Functional diversification of acyl-coenzyme A oxidases in jasmonic acid biosynthesis and action. Plant Physiol 143:812–824
Schweiger R, Heise A, Persicke M, Müller C (2014) Interactions between the jasmonic and salicylic acid pathway modulate the plant metabolome and affect herbivores of different feeding types. Plant Cell Environ 37:1574–1585

Selvaraj K, Fofana B (2012) An overview of plant photosynthesis modulation by pathogen attacks. In: Advances in photosynthesis—fundamental aspects, vol 22. IntechOpen, London, pp 466–484

Shang J, Xi D-H, Xu F et al (2011) A broad-spectrum, efficient and nontransgenic approach to control plant viruses by application of salicylic acid and jasmonic acid. Planta 233:299–308

Shang Y, Wang K, Sun S et al (2019) COP9 signalosome CSN4 and CSN5 subunits are involved in jasmonate-dependent defense against root-knot nematode in tomato. Front Plant Sci 10:1223

Sharma A, Shahzad B, Kumar V et al (2019) Phytohormones regulate accumulation of osmolytes under abiotic stress. Biomolecules 9:285

Shigenaga AM, Argueso CT (2016) No hormone to rule them all: interactions of plant hormones during the responses of plants to pathogens. In: Seminars in cell and developmental biology. Elsevier, Amsterdam, pp 174–189

Shrestha K, Pant S, Huang Y (2021) Genome-wide identification and classification of *Lipoxygenase* gene family and their roles in sorghum-aphid interaction. Plant Mol Biol 105:527–541

Shu P, Li Z, Min D et al (2020) CRISPR/Cas9-mediated *SlMYC2* mutagenesis adverse to tomato plant growth and MeJA-induced fruit resistance to *Botrytis cinerea*. J Agric Food Chem 68:5529–5538

Sikder MM, Vestergård M, Kyndt T et al (2021) Phytohormones selectively affect plant parasitic nematodes associated with *Arabidopsis* roots. N Phytol 232(3):1272–1285

Singh UB, Malviya D, Singh S et al (2019) *Trichoderma harzianum*- and methyl jasmonate-induced resistance to *Bipolaris sorokiniana* through enhanced phenylpropanoid activities in bread wheat (*Triticum aestivum* L.). Front Microbiol 10:1697

Singh A, Roychowdhury R, Singh T et al (2020) Improvement of crop's stress tolerance by gene editing CRISPR/CAS9 system. In: Sustainable agriculture in the era of climate change. Springer, Cham, pp 557–587

Singh P, Arif Y, Siddiqui H, Hayat S (2021) Jasmonate: a versatile messenger in plants. In: Jasmonates and salicylates signaling in plants. Springer, Cham, pp 129–158

Singh AD, Kour J, Kumar P et al (2023) Role of jasmonates in regulating physiological and molecular aspects of plants under abiotic stress. In: The role of growth regulators and phytohormones in overcoming environmental stress. Elsevier, Amsterdam, pp 137–173

Singla P, Bhardwaj RD, Sharma S, Sunidhi (2023) Plant–fungus interaction: a stimulus–response theory. J Plant Growth Regul 43:1–13

Sirisha VL, Mitra S, Suprasanna P (2024) Plant immune system: mechanisms and resilience. In: Stress: immunology and inflammation. Elsevier, Amsterdam, pp 9–21

Slaughter A, Daniel X, Flors V et al (2012) Descendants of primed *Arabidopsis* plants exhibit resistance to biotic stress. Plant Physiol 158:835–843

Šņepste I, Krivmane B, Šķipars V et al (2021) Induction of defense responses in *Pinus sylvestris* seedlings by methyl jasmonate and response to *Heterobasidion annosum* and *Lophodermium seditiosum* inoculation. Forests 12:628

Song H, Wang P, Li C et al (2016) Identification of *lipoxygenase* (*LOX*) genes from legumes and their responses in wild type and cultivated peanut upon *Aspergillus flavus* infection. Sci Rep 6:35245

Song W, Shao H, Zheng A et al (2023) Advances in roles of salicylic acid in plant tolerance responses to biotic and abiotic stresses. Plants 12:3475

Soriano IR, Asenstorfer RE, Schmidt O, Riley IT (2004) Inducible flavone in oats (*Avena sativa*) is a novel defense against plant parasitic nematodes. Phytopathology 94:1207–1214

Spoel SH, Johnson JS, Dong X (2007) Regulation of tradeoffs between plant defenses against pathogens with different lifestyles. Proc Natl Acad Sci USA 104:18842–18847

Sugio A, Dubreuil G, Giron D, Simon J-C (2015) Plant–insect interactions under bacterial influence: ecological implications and underlying mechanisms. J Exp Bot 66:467–478

Sun B, Li W, Ma Y et al (2023) *OsGLP3-7* positively regulates rice immune response by activating hydrogen peroxide, jasmonic acid, and phytoalexin metabolic pathways. Mol Plant Pathol 24:248–261

Szymański J, Bocobza S, Panda S et al (2020) Analysis of wild tomato introgression lines elucidates the genetic basis of transcriptome and metabolome variation underlying fruit traits and pathogen response. Nat Genet 52:1111–1121

Taniguchi S, Hosokawa-Shinonaga Y, Tamaoki D et al (2014) Jasmonate induction of the monoterpene linalool confers resistance to rice bacterial blight and its biosynthesis is regulated by JAZ protein in rice. Plant Cell Environ 37:451–461

Taniguchi S, Takeda A, Kiryu M, Gomi K (2023) Jasmonic acid-induced β-cyclocitral confers resistance to bacterial blight and negatively affects abscisic acid biosynthesis in rice. Int J Mol Sci 24:1704

Thireault C, Shyu C, Yoshida Y et al (2015) Repression of jasmonate signaling by a non-TIFY JAZ protein in *Arabidopsis*. Plant J 82:669–679

Truman W, Bennett MH, Kubigsteltig I et al (2007) *Arabidopsis* systemic immunity uses conserved defense signaling pathways and is mediated by jasmonates. Proc Natl Acad Sci USA 104:1075–1080

Tsuda K (2018) Division of tasks: defense by the spatial separation of antagonistic hormone activities. Plant Cell Physiol 59:3–4

Ullah C, Tsai C, Unsicker SB et al (2019) Salicylic acid activates poplar defense against the biotrophic rust fungus *Melampsora larici-populina* via increased biosynthesis of catechin and proanthocyanidins. N Phytol 221:960–975

Van der Does D, Leon-Reyes A, Koornneef A et al (2013) Salicylic acid suppresses jasmonic acid signaling downstream of *SCF-COI1-JAZ* by targeting GCC promoter motifs via transcription factor ORA59. Plant Cell 25:744–761

Vieira dos Santos MC, Curtis RHC, Abrantes I (2013) Effect of plant elicitors on the reproduction of the root-knot nematode *Meloidogyne chitwoodi* on susceptible hosts. Eur J Plant Pathol 136:193–202

Wang HV, Chekanova JA (2016) Small RNAs: essential regulators of gene expression and defenses against environmental stresses in plants. Wiley Interdiscip Rev RNA 7:356–381

Wang G, Hu C, Zhou J et al (2019) Systemic root–shoot signaling drives jasmonate-based root defense against nematodes. Curr Biol 29:3430–3438

Wang J, Song L, Gong X et al (2020) Functions of jasmonic acid in plant regulation and response to abiotic stress. Int J Mol Sci 21:1446

Wang Y, Mostafa S, Zeng W, Jin B (2021) Function and mechanism of jasmonic acid in plant responses to abiotic and biotic stresses. Int J Mol Sci 22:8568

Wang L, Liu S, Gao M et al (2022) The crosstalk of the salicylic acid and jasmonic acid signaling pathways contributed to different resistance to phytoplasma infection between the two genotypes in Chinese jujube. Front Microbiol 13:800762

Waqas M, Shahzad R, Hamayun M et al (2018) Biochar amendment changes jasmonic acid levels in two rice varieties and alters their resistance to herbivory. PLoS ONE 13:e0191296

Wasternack C (2014) Jasmonates in plant growth and stress responses. In: Phytohormones: a window to metabolism, signaling and biotechnological applications. Springer, New York, pp 221–263

Wasternack C, Hause B (2013) Jasmonates: biosynthesis, perception, signal transduction and action in plant stress response, growth and development. An update to the 2007 review in Annals of Botany. Ann Bot 111:1021–1058

Wasternack C, Strnad M (2019) Jasmonates are signals in the biosynthesis of secondary metabolites—pathways, transcription factors and applied aspects—a brief review. Nat Biotechnol 48:1–11

Wei J, van Loon JJA, Gols R et al (2014) Reciprocal crosstalk between jasmonate and salicylate defence-signalling pathways modulates plant volatile emission and herbivore host-selection behaviour. J Exp Bot 65:3289–3298

Wilkinson SW, Hannan Parker A, Muench A et al (2023) Long-lasting memory of jasmonic acid-dependent immunity requires DNA demethylation and ARGONAUTE1. Nat Plants 9:81–95

Xin X-F, Kvitko B, He SY (2018) *Pseudomonas syringae*: what it takes to be a pathogen. Nat Rev Microbiol 16:316–328

Xu L, Liu F, Lechner E et al (2002) The SCFCOI1 ubiquitin-ligase complexes are required for jasmonate response in *Arabidopsis*. Plant Cell 14:1919–1935

Yamchi A, Ben C, Rossignol M et al (2018) Proteomics analysis of *Medicago truncatula* response to infection by the phytopathogenic bacterium *Ralstonia solanacearum* points to jasmonate and salicylate defence pathways. Cell Microbiol 20:e12796

Yan C, Fan M, Yang M et al (2018) Injury activates Ca^{2+}/calmodulin-dependent phosphorylation of JAV1-JAZ8-WRKY51 complex for jasmonate biosynthesis. Mol Cell 70:136–149

Yang L, Teixeira PJPL, Biswas S et al (2017) *Pseudomonas syringae* type III effector HopBB1 promotes host transcriptional repressor degradation to regulate phytohormone responses and virulence. Cell Host Microbe 21:156–168

Yang T, Zhu L, Meng Y et al (2018) Alpha-momorcharin enhances Tobacco mosaic virus resistance in tobacco^{NN} by manipulating jasmonic acid-salicylic acid crosstalk. J Plant Physiol 223:116–126

Yang J, Duan G, Li C et al (2019) The crosstalks between jasmonic acid and other plant hormone signaling highlight the involvement of jasmonic acid as a core component in plant response to biotic and abiotic stresses. Front Plant Sci 10:1349

Yang Z, Huang Y, Yang J et al (2020) Jasmonate signaling enhances RNA silencing and antiviral defense in rice. Cell Host Microbe 28:89–103

Yang Y, Yang X, Guo X et al (2022) Exogenously applied methyl Jasmonate induces early defense related genes in response to *Phytophthora infestans* infection in potato plants. Hortic Plant J 8:511–526

Yarullina LG, Troshina NB, Cherepanova EA et al (2011) Salicylic and Jasmonic acids in regulation of the proantioxidant state in wheat leaves infected by *Septoria nodorum* Berk. Appl Biochem Microbiol 47:549–555

Yuan M, Jiang Z, Bi G et al (2021) Pattern-recognition receptors are required for NLR-mediated plant immunity. Nature 592:105–109

Zamora O, Schulze S, Azoulay-Shemer T et al (2021) Jasmonic acid and salicylic acid play minor roles in stomatal regulation by CO_2, abscisic acid, darkness, vapor pressure deficit and ozone. Plant J 108:134–150

Zander M, Lewsey MG, Clark NM et al (2020) Integrated multi-omics framework of the plant response to jasmonic acid. Nat Plants 6:290–302

Zhang X, Zhu Z, An F et al (2014) Jasmonate-activated MYC2 represses *ETHYLENE INSENSITIVE3* activity to antagonize ethylene-promoted apical hook formation in *Arabidopsis*. Plant Cell 26:1105–1111

Zhang F, Yao J, Ke J et al (2015a) Structural basis of JAZ repression of MYC transcription factors in jasmonate signalling. Nature 525:269–273

Zhang L, Yao J, Withers J et al (2015b) Host target modification as a strategy to counter pathogen hijacking of the jasmonate hormone receptor. Proc Natl Acad Sci USA 112:14354–14359

Zhang L, Zhang F, Melotto M et al (2017a) Jasmonate signaling and manipulation by pathogens and insects. J Exp Bot 68:1371–1385

Zhang W, Corwin JA, Copeland D et al (2017b) Plastic transcriptomes stabilize immunity to pathogen diversity: the jasmonic acid and salicylic acid networks within the *Arabidopsis*/*Botrytis* pathosystem. Plant Cell 29:2727–2752

Zhang Z, Ge X, Luo X et al (2018) Simultaneous editing of two copies of *GH14-3-3D* confers enhanced transgene-clean plant defense against *Verticillium dahliae* in allotetraploid upland cotton. Front Plant Sci 9:842

Zhang R, Min Y, Holappa LD et al (2020) A role for the Auxin Response Factors *ARF6* and *ARF8* homologs in petal spur elongation and nectary maturation in Aquilegia. N Phytol 227:1392–1405

Zhang Z, Lu S, Yu W et al (2022) Jasmonate increases terpene synthase expression, leading to strawberry resistance to *Botrytis cinerea* infection. Plant Cell Rep 41(5):1243–1260

Zhao Q, Liu R, Zhou Q et al (2023a) Calcium-binding protein OsANN1 regulates rice blast disease resistance by inactivating jasmonic acid signaling. Plant Physiol 192(2):1621–1637

Zhao W, Liang J, Huang H et al (2023b) Tomato defence against *Meloidogyne incognita* by jasmonic acid-mediated fine-tuning of kaempferol homeostasis. N Phytol 238(4):1651–1670

Zhou Z, Wu Y, Yang Y et al (2015a) An *Arabidopsis* plasma membrane proton ATPase modulates JA signaling and is exploited by the *Pseudomonas syringae* effector protein AvrB for stomatal invasion. Plant Cell 27:2032–2041

Zhou J, Jia F, Shao S et al (2015b) Involvement of nitric oxide in the jasmonate-dependent basal defense against root-knot nematode in tomato plants. Front Plant Sci 6:193

Zhou W, Guo Z, Niu J et al (2019) Effect of methyl jasmonate on resistance of alfalfa root rot caused by *Fusarium oxysporum*. Acta Phytopathol Sin 49:379–390

Zhu Z, Lee B (2015) Friends or foes: new insights in jasmonate and ethylene co-actions. Plant Cell Physiol 56:414–420

Zhu F, Cao M-Y, Zhang Q-P et al (2023) Join the green team: inducers of plant immunity in the plant disease sustainable control toolbox. J Adv Res 57:15–42

Zinovieva SV, Vasyukova NI, Udalova ZV et al (2011) Involvement of salicylic acid in induction of nematode resistance in plants. Biol Bull 38:453–458

Zinovieva SV, Vasyukova NI, Udalova ZV, Gerasimova NG (2013) The participation of salicylic and jasmonic acids in genetic and induced resistance of tomato to *Meloidogyne incognita* (Kofoid and White, 1919). Biol Bull 40:297–303

Ziv C, Zhao Z, Gao YG, Xia Y (2018) Multifunctional roles of plant cuticle during plant–pathogen interactions. Front Plant Sci 9:1088