Plant Archives Vol. 24, No. 2, 2024 pp. 2601-2612 e-ISSN:2581-6063 (online), ISSN:0972-5210

Plant Archives

Journal homepage: http://www.plantarchives.org

DOI Url : https://doi.org/10.51470/PLANTARCHIVES.2024.v24.no.2.372

HARNESSING NATURE’S SIGNAL: THE TRANSFORMATIVE ROLE OF JASMONIC ACID IN PLANT DEFENSE AND DEVELOPMENT

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(Date of Receiving-13-04-2024; Date of Acceptance-02-07-2024)

ABSTRACT

The plant hormones jasmonic acid (JA) and its derivative, jasmonoylisoleucine (JA-Ile), serve as crucial signaling molecules regulating both cell defense and development in plants. This review highlights the increasing research interest in JA since the 1990s. JA has been identified as a stress hormone orchestrating plant responses to various stressors, including biotic factors (pathogens and herbivores) and abiotic factors (wounding, UV, salt, drought, heavy metals, micronutrient toxicity, low temperature). As signaling molecules, JAs play a vital role in activating defense mechanisms and modulating the expression of numerous genes in response to abiotic stress. The association between plant resistance to abiotic stress and jasmonic acid is emphasized, with JA influencing physiological and molecular reactions. Key responses include the activation of the antioxidant system, accumulation of soluble sugars and amino acids (such as methionine and isoleucine), and control over stomatal opening and closing. This comprehensive overview underscores the multifaceted role of JA in mediating plant responses to diverse stress conditions.

Key words : Jasmonic acid, Abiotic stress, Plant resistance and Environment.

Introduction

Jasmonic acid (JA), a plant hormone and its derivative jasmonoylisoleucine (JA-Ile), the amino acid conjugate signal molecules that control cell defense and development in plants. It was discovered to be a stress hormone that regulates plant responses to both biotic (caused by pathogens and herbivores) and abiotic (caused by wounding and UV) stress (Gomi, 2020). It directly affects many physiological processes, including root growth, senescence, and anther growth. Jasmonates (JA) are a class of oxidized lipids known as oxylipins. These are fatty acids derived from cyclopentanones (Wasternack *et al.*, 2018). These oxylipins are physiologically active signaling molecules, either non-enzymatically through the autoxidation of polyunsaturated fatty acids or enzymatically through lipoxygenases or alpha-dioxygenases (Gobel *et al.*, 2009). In higher plant species, the JAs are widely distributed with high concentrations in reproductive tissues and flowers and very low concentrations in mature leaves and roots (Dar *et al.*, 2015). It regulates many important plant growth and development processes, such as vegetative growth, cell cycle regulation, anthocyanin biosynthesis, anther and trichome development, fruit ripening, senescence, inhibitionof rubisco biosynthesis, stomatal opening, nitrogen and phosphorus absorption and glucose transport (Parthier *et al.*, 1991). As signaling molecules, they support particular defense mechanisms and control the expression of numerous genes in response to abiotic stress such as salt, drought, heavy metals, micronutrient toxicity, low temperature, etc (Li *et al.*, 2018). 3-oxo-2-2'-cis-pentenylcyclopentane-1-acetic acid is the fundamental component of the chemical structure of JA. It is an endogenous signaling molecule involved in multiple developmental processes and initially believed to be a stress-related hormone in higher plants (Llanes *et al.*, 2016). In soybeans, jasmonic acid controls the

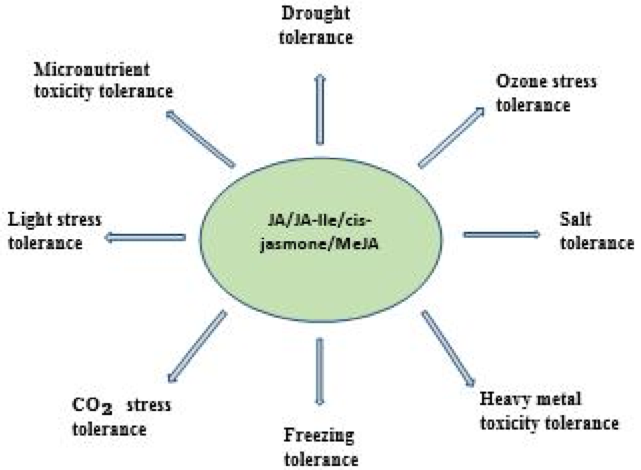

Fig. 1 : Types of abiotic stress response by jasmonic acid.

morphogenesis of the leaves and roots. The first source of jasmonic acid isolation was fungus cultures. In methyl jasmonate, *Lasiodiplodia theobromae* was extracted from the essential oils of *Rosmarinus officinalis L*. and *Jasminum grandiforum* L. All higher plants contain jasmonates, with the concentration being highest in flowers and reproductive tissues, as well as in certain roots and fully grown leaves. Jasmonic acid has several derivatives, most of which are hydroxylated forms like tuberone acid, succinic acid and other conjugates of amino acids. (Hamberg *et al.*, 1992).

Biosynthesis and metabolism during abiotic stress

JA is synthesized biologically with the help of sequential enzyme action occurring in the cell cytoplasm, peroxisome and plastid (Feussner *et al*., 2002). Linolenic acid occurring as a precursor is oxygenated with the help of lipoxygenase (LOX), allene oxide synthase (AOS), and allene oxide cyclase (AOC) enzymes resulting in the production of 12-oxo-phytodienoic acid (12-oxo-PDA). Further with three cycles of beta-oxidation and the activity of 12-oxo-phytodienoic acid reductase (OPR), JA is synthesized from 12-oxo-PDA. Therefore, the jasmonic acid is synthesized following a regular action and interaction of different enzymes; hence the process of synthesis is regarded as an octadecanoid pathway (Ruan *et al*., 2019).

In the chloroplast, JA biosynthesis begins with the chloroplast membrane release of linolenic acid, which is finally converted to 12-oxo-PDA. Upon transport of 12-oxo-PDA into the peroxisome, a series of enzymes work to convert it to JA, which is then exported to the cytoplasm. In the cytosol, the synthesis of jasmonic acid results in the conversion of phytohormone into 30 different active and inactive derivatives based on the chemical changes in the pentanone ring (carboxylic acid group) or pentenyl side chain such as JA, jasmonic acid; JA-Ile, jasmonylisoleucine; MeJA, methyl jasmonate; 12-HSO$_4$ -JA, 12-hydroxy jasmonic acid sulfate; 12-oxo-PDA, 12-oxo-phytodienoic acid (Fig. 1) (Wasternack *et al.*, 2016). Among the different groups of metabolites, free JA, cis-jasmone, MeJA and JA-Ile are regarded as the key to bioactive JA in plants (Wasternack *et al*., 2013).

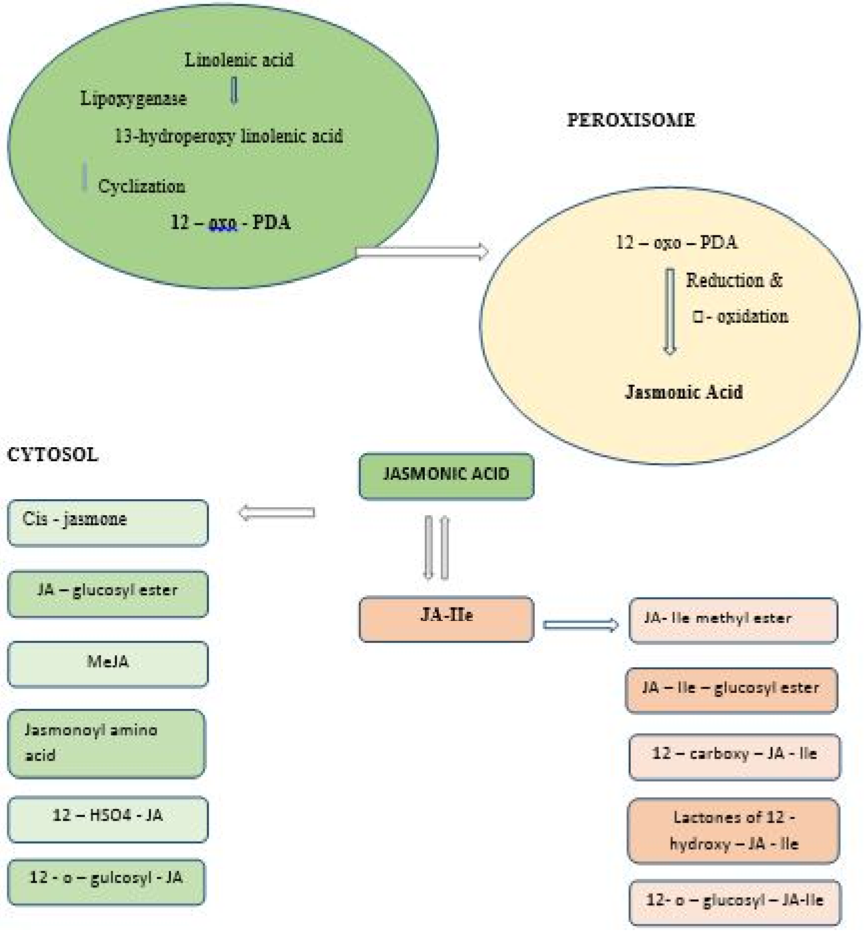

Fig. 2 : Diagram showing the biosynthesis and metabolism of Jasmonic acid in response to abiotic stress.

Signaling pathway for jasmonic acid

The signaling pathway for jasmonic acid (JA) is a crucial mechanism in plants for responding to various environmental stresses. When a plant is subjected to stress, it triggers the release or activation of jasmonic acid (JA) or its derivatives, primarily jasmonoyl-isoleucine (JA-Ile). When JA-Ile levels are low or absent, JAZ proteins repress transcription factors, which stops them from activating the

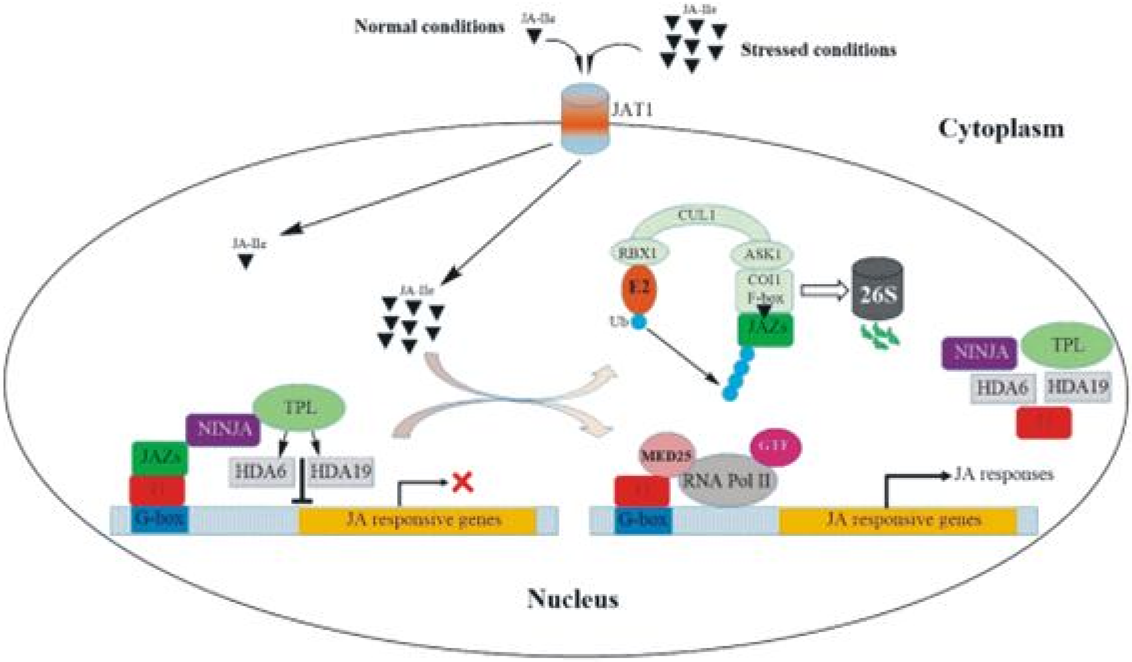

Fig. 3 : Jasmonic acid perception and signal transduction during abiotic stress (Sarafat *et al*., 2020). JA-Ile, jasmonyl isoleucine; JAT1, jasmonic acid transfer protein 1; TF, transcription factor; JAZ, jasmonate ZIM domain; NINJA, novel interactor of JAZ; TPL, topless; HDA6, HDA19, histone deacetylase 6, 19; Ub, ubiquitin; E2, ubiquitin-conjugating enzymes; RBX1, ring box 1; CUL1, cullin 1; ASK1, Arabidopsis SKP1 homolog 1; COI1, coronatine insensitive 1; MED25, mediator 25; RNA Pol II, RNA polymerase II; GTF, general transcription factor).

promoters of genes that respond to jasmonate. In order to create an active transcriptional repression complex that inhibits JA responses, JAZ proteins recruit TPL and the adaptor protein NINJA. This complex then changes from an open to a closed state by recruiting HDA6 and HDA19. The synthesis of JA is elevated by abiotic stresses and easily epimerizes into JA-Ile. After that, the JAT1 transporter moves the latter to the nucleus. JAZ is degraded by proteases when it interacts with the F-box protein COI1 in the SCF complex, which is facilitated by JA-Ile. The expression of jasmonate-responsive genes is caused by the recruitment of MED25, RNA Pol II, and GTF when the derepressed TF binds to the G-box element (Fig. 2) (Sarafat *et al*., 2020). This signaling pathway allows plants to mount an effective defense response against various stresses, contributing to their survival and adaptation in challenging environments.

Impact of jasmonic acid on abiotic stress

Cold stress

Low-temperature stress, such as freezing (below 0°C) and chilling (0–15°C), is referred to as cold stress (Wang *et al*., 2016). The expression of JA biosynthesis-related genes was upregulated in cold-stressed plants, suggesting that cold stress triggers JA production by inducing the expression of JA biosynthetic genes (Du *et al*., 2013). JA treatment increased the adaptability of orange (*Citrus sinensis*) plants to cold stress. Oranges treated with JA had lower H_2O_2 levels and higher antioxidant gene expression when compared with untreated control plants (Habibi *et al*., 2019). Additionally, research examining how JA affects bananas' (*Musa acuminata*) ability to withstand cold stress observed that, in comparison to fruit that was not treated, JA treatment increased the expression of genes related to this ability and strengthened the fruit's resistance to cold stress (Zhao *et al*., 2013). When the temperature falls below -2ºC, apples are more vulnerable to frost damage and are susceptible to chilling. According to Yoshikawa *et al*. (2007), applying PDJ (n-propyl dihydrojasmonate) can reduce frost damage by 14–10%. It can also raise UFGluT transcription levels in apple fruit skin, which activates the enzyme involved in the synthesis of anthocyanins (Kondo, 2010). Chilling damage can be minimized by reducing the quantity of methyl jasmonate used to chill grapefruit and avocado storage (Meir *et al*., 1996). Treatments with salicylic acid and methyl jasmonate build up reactive oxygen species (ROS), which strengthen resistance against chilling injury (Siboza and Bertling, 2015). Additionally, discovered that methyl jasmonate treatments in guava fruit reduce the ion leakage percentage as well as the chilling injury index. Moreover, MJ had no effect on total phenols, vitamin C, or chlorophyll. It increased the levels of lipoxygenase (LOX), phenylalanine-ammonia lyase (PAL) and sugar content. The result was that MJ reduces chilling injury and activates the fruit defense response according to the behavior of total phenols and the increase in sugar content, PAL and LOX activities (González *et al.*, 2004).

Drought Stress

The primary abiotic stress influencing plant growth and productivity is drought. Due to stomata closing, which reduces photosynthetic efficiency; it can alter the integrity of membranes, reduce turgor pressure, and hinder gas exchange (Seleiman *et al.*, 2021). Recent research suggests that treatment with exogenous acetate elevates drought tolerance and leads to dynamic metabolic changes that produce acetate, which triggers JA biosynthesis and response (Kim *et al.*, 2017). After 12 hours of drought exposure, tomato (*Solanum lycopersicum*) roots exhibited JA levels that were approximately ten times greater than those of untreated roots. Tomato leaves also displayed an increase in cellular JA levels (Zhang *et al.*, 2013). Additional evidence of JA's involvement in the plant drought response was obtained by analyzing the effect of exogenous JA on drought tolerance. The soybean (*Glycine max*) treated with JA exhibited greater resistance to drought stress when compared to the untreated control plants (Riemann *et al.*, 2015). Osmotic imbalance, ROS accumulation, and cellular dehydration are caused by drought. The build-up of osmoprotectants and the elimination of ROS are critical components of the plant defense mechanism against drought stress. This was partially supported by the fact that, in the presence of abiotic stress, the osmoprotectant proline regulates ROS homeostasis (Mahmood *et al.*, 2020). According to Fugate *et al.* (2018), plants exposed to JA showed reduced ROS levels and increased proline accumulation. The impact of JA on proline and ROS levels was supported by findings showing that JA regulates the transcriptional expression of genes involved in proline metabolism and the ROS scavenging system (Anjum *et al.*, 2013).

Salt Stress

Salt stress, which is brought on by high salt concentrations in the soil or water supply, disrupts the osmotic balance and reduces water absorption. As a result, there may be an accumulation of reactive oxygen species (ROS), inhibition of photosynthetic processes, membrane leakage, and growth suppression, among other physiological and developmental disorders (Li *et al.*, 2020). In almond (*Prunus dulcis*) plants, exogenous JA treatment reduced the negative effects of salt stress on plant growth and chlorophyll levels, indicating that JA increases plant tolerance to salt stress (Tavallali *et al.*, 2019).Using JA biosynthesis and signaling mutants, genetic methods were also used to investigate the function of JA in the salt response and tolerance. The production of JA is governed by LOX3 in Arabidopsis. Under typical growth conditions, the LOX3 knock-out mutant (*lox3*) and the wild type had comparable germination rates (Ding *et al.*, 2016). However, the *lox3* mutant's germination rate under salt stress was 2.5 times lower than that of plants of the wild type. Furthermore, under salt stress conditions, the lox3 mutant plants exhibited roughly three times lower levels of JA than the wild-type plants, suggesting that JA controls plant tolerance to salt stress. Comparably, def-1, a tomato mutant lacking in JA, exhibited reduced resistance to salt stress. Under salt stress, the def-1 mutant's nitrogen accumulation was considerably lower than that of the wild-type plants (Abouelsaad *et al.*, 2018).

Heavy metals stress

Heavy metal stress is caused by an excessive accumulation and absorption of heavy metals, which includes metals and metalloids that may be toxic or ecotoxic (Ojuederie *et al.*, 2017). *Suaeda glauca* and *A. thaliana* exposed to high concentrations of lead (Pb), nickel (Ni), cadmium (Cd) and manganese (Mn) showed a decrease in fresh weight and concentrations of photosynthetic pigment. In plants, many of these metals serve no useful purpose and in very small amounts, may even be detrimental. (Ali *et al.*, 2012).

There is mounting evidence that plants use JA as a response to heavy metal stress. When copper (Cu) or cadmium (Cd) were applied to Arabidopsis cells, the amount of JA increased quickly (Ojuederie *et al.*, 2017). Research has shown that exposure to heavy metals increases the amount of JA in the body and activates genes that are either JA-responsive or JA-biosynthetic. Numerous studies examining the effects of exogenous JA on plants exposed to heavy metals have also shown the involvement of JA in heavy metal stress. In pepper, cadmium decreased root growth and chlorophyll b levels; however, exogenous JA treatment mitigated these effects (Yan *et al.*, 2013). In *Wolffia arrhiza*, lead (Pb) reduced the levels of carotenoids and chlorophyll a. However, Pb-induced reductions in carotenoids and chlorophyll a were reversed by an exogenous JA treatment that decreased Pb absorption and accumulation (Bali *et al.*, 2019). Another study determined that JA inhibited lipid peroxidase activity by activating ascorbate or glutathione's antioxidant machinery. Moreover, the significant increase in HO-1 antioxidant enzyme activity they observed during heavy metal stress may have been solely regulated by ROS homeostasis. The results obtained imply that JAs regulate plants' antioxidant systems, which in turn regulates how the plants respond to heavy metal stress (Carvalho *et al*., 2013).

Light stress

Light is essential for regulating both JA biosynthesis and signal transduction (Kazan *et al*., 2011). In wheat seedlings, the study found that JA pretreatment significantly reduced the negative effects of UV-B on photosystem II function by increasing the effective photosystem II quantum yield, the photosynthetic electron transport rate, the rate of nonphotochemical quenching, and the reaction center's capture efficiency of excitation energy. The results show that exogenous JA could lessen the harmful effects of UV-B stress on wheat seedlings. (Liu *et al.,* 2012). JA signaling pathways in *Nicotiana* and *Brassica* species were activated by the JA biosynthesis caused by UVB treatment, which enhanced the plants' defense mechanisms, according to several studies (Svyatyna *et al*., 2012). Blue light-mediated light morphogenesis in *A. thaliana* and tomato (*L. esculentum*) and red light/far-red light-mediated photomorphogenesis in *A. thaliana* and rice (*O. sativa*) were both influenced by JA signaling (Cerrudo *et al*., 2012; Mewis *et al*., 2021).

Ozone stress

Plants experience lesions and programmed cell death as a result of ROS produced by ozone. Following ozone treatment, it was discovered that the JA content in wild-type Arabidopsis increased noticeably (Rao *et al*., 2000). An external MeJA treatment could stop the spread of ozone-induced programmed cell death (Overmyer *et al*., 2000). Furthermore, following exogenous MeJA treatment, the hybrid poplar (*Populus maximowizii* × *P. trichocarpa*) and tomato (*L. esculentum*) displayed decreased ozone sensitivity (Koch *et al*., 2000). In tomato plants, elevated ozone triggered the JA pathway, which dramatically increased the rates at which volatile compounds were emitted to shield the plants from natural enemies (Cui *et al*., 2016). However, recent research on *Gossypium hirsutum*, the cotton plant, has revealed that MeJA only inhibits the diffusion of ozone damage at high concentrations of ozone, along with ethylene antagonistic effects (Grantz*et al*., 2012).

CO_2 stress

MeJA and cis-JA were released into the environment at higher CO_2 concentrations in lima beans (*P. lunatus*) (Ballhorn *et al*., 2011). Tomato plants' defense mechanism against nematodes was strengthened by an elevated CO_2 level, which activated the JA- and SA-signaling pathways (Sun *et al*., 2011). In tobacco, the elevated CO_2 level also increased the levels of JA and major defense-related metabolites, whereas in rice, they decreased (Lu *et al*., 2018).

Micronutrient toxicity

According to several reports, JAs may be able to shield plants from the damaging effects of micronutrient toxicity. It has been observed that a high boron concentration inhibits plant growth and development (Karabal *et al* ., 2003). The study examined the impact of 2.50 mg B l^{-1} excess boron in greenhouse conditions on grafted sour orange and 'Navelina' orange plants that were derived from Swingle citrumelo rootstocks. Based on the results, plants grafted on Swingle citrumelo showed reduced leaf thickness and chloroplast size because of their decreased sensitivity to excess B (Papadakis *et al.,* 2004). Overexposure of apple rootstock EM 9 to boron (B) altered the functions of antioxidant and nucleolytic enzymes and resulted in oxidative stress. Overexposure to B lowered catalase activity and increased H_2O_2 accumulation, lipid peroxidation, and lipoxygenases. The study found a correlation between B toxicity and oxidative damage (Molassiotis *et al.,* 2003). Exogenous MeJA treatment may be able to mitigate the boron toxicity in plants by preventing lipid peroxidation and stimulating the antioxidative defense enzymes (CAT, POD and SOD) (Aftab *et al*., 2011). Furthermore, JAs are necessary for the defense mechanisms that plants have against lead (Pb) stress. Plant growth was enhanced and Pb uptake was reduced when JA was applied to prime tomato seeds (Bali *et al*., 2019).

Effects on seed germination

It has been demonstrated that phytohormones like JA, IAA, and ABA (abscisic acid) stimulate seed germination (Xiao *et al*., 2018). Both ABA and JA inhibit seed germination, but it is unclear how they interact with one another during this process (Tang *et al*., 2020). According to Dave *et al*. (2011), JA inhibition takes place in Arabidopsis without the COI1 co-receptor's assistance. JA promotes cold-stimulated germination because cold-stimulated seed germination leads to an increase in endogenous JA following the overexpression of genes related to JA biosynthesis in *Triticum aestivum* plants (Xu *et al*., 2015b; Avramova, 2017). According to Wang *et al*. (2020), a novel rice pathway known as SAPK10-bZIP72-AOC has been observed. In this pathway, ABA encourages the synthesis of JA and when combined with other inhibitors suppress the germination of rice seeds.

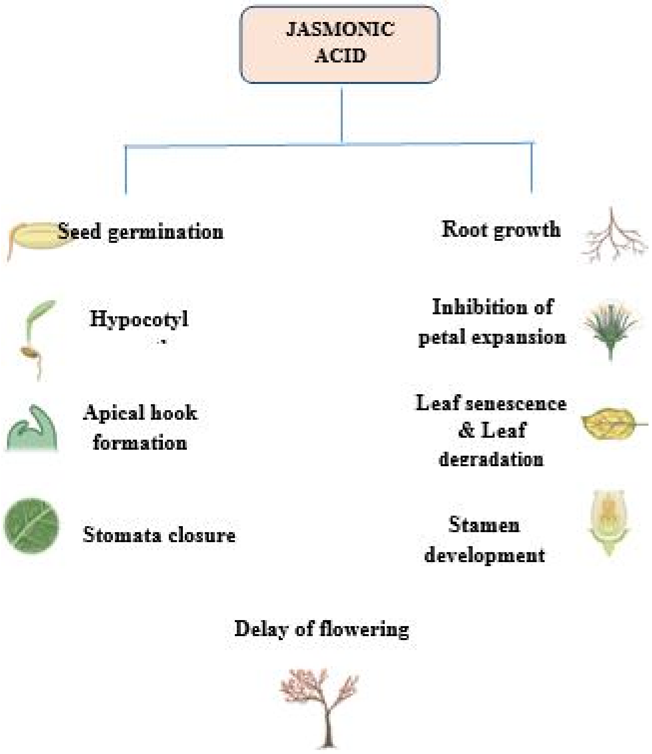

Fig. 4 : Impact of jasmonic acid on plant growth and development.

When high JA concentrations are present, autophosphorylation triggers SAPK10 (serine/threonine protein kinase), which strengthens the bZIP72 transcription factor (TF; basic leucine zipper) and enhances its binding to the G-box cis-element within the AOC (assimilable organic carbon) promoter. It's interesting to consider that blocking JA biosynthesis decreased ABA sensitivity (Wang *et al.*, 2020).

Delay of flowering

In Arabidopsis, jasmonate prevents the plant from reaching reproductive maturity. The interaction between COI1 and JAZ hinders flowering. Plants that had been transgenic for JAZ13A and coi1 mutants exhibited early flowering. Moreover, TARGET OF EAT TFs (TOE1 and TOE2), APETALA2/ETHYLENE RESPONSE FACTOR (AP2/ERF) domain TFs, regulate plant flowering. Indeed, their binding to JAZ proteins inhibits blooming by deactivating transcription of FLOWERING LOCUS T. Conversely, the *coi1* early-blooming phenotype is inhibited by overexpression of TOE1 and TOE2 (Zhai *et al.*, 2015).

Inhibition of Root Growth

Plants resistant to JAs' inhibition of primary root maturation have mutations in the JA-Ile (Jasmonoyl-isoleucine) COI1 (Coronatine-insensitive 1) co-receptor (Yan *et al.*, 2009). By enhancing COI1–JAZ9 (Jasmonate Zim Domain) interactions, isositolpentakisphosphate (InsP5) eliminates the impact of JAs on root elongation and maturation (Mosblech *et al.*, 2011). By preventing COI1–JAZs interactions, coronatine-O-methyloxime, a competitive JA antagonist, inhibits the coronatine inhibitory effect on primitive root growth (Monte *et al.*, 2014). Most JAZ proteins in Arabidopsis lack the Ethylene-responsive element-binding factor (ERF)-associated amphiphilic repression (EAR) domain. They should interact with TPL (TOPLESS) and TPL-related proteins (TPRs) by using the EAR of NINJA proteins to suppress JA responses. JAZ8 and JAZ13, two non-canonical JAZ proteins, use their single EAR domain directly (Thireault *et al.*, 2015; Chini *et al.*, 2016). The inhibitory effect of JA on primary root development was reduced by overexpressing NINJA proteins or modified JAZ proteins; however, the inhibitory effect was increased by NINJA/TPL or combination mutations in JAZ7, JAZ8, JAZ10 and JAZ13 (Thireault *et al.*, 2015; Thatcher *et al.*, 2016). By interacting with JAZ proteins, the bHLH (basic helix– loop–helix) transcription factors of Arabidopsis, MYC2 and its homologs, prevent primary root growth at the primary root apex (Qi *et al.*, 2015; Gasperini *et al.*, 2015). Furthermore, MYC2 restricts PLETHORA (PLT1 and PLT2) gene expression, which impacts root meristematic cells and mitigates their activity as well as growth. By interacting with MED25 (MEDIATOR25, MYC2 inhibits RNA polymerase II and simplifies the inhibitory effect of the enzyme (Chen *et al.*, 2011). JAZ9 interferes with MYC3-MED25 communication (Gimenez-Ibanez *et al.*, 2014). In the involvement of mitogen-activated protein kinase 3/6, PLANT U-BOX PROTEIN10 reduces JA-mediated root maturation inhibition by hindering MYC2 ubiquitination and phosphorylation (Chico *et al.*, 2014; Sethi *et al.*, 2014).

Inhibition of petal expansion

Using the COI1 pathway, jasmonate inhibits the growth of petals in Arabidopsis, leading to coi1 mutants to exhibit larger petals at anthesis and JA-deficient mutants to have similar patterns in *aos* and *opr3* genes of Arabidopsis (Brioudes *et al.*, 2009; Reeves *et al.*, 2012). JA interferes with the growth of petals by inhibiting the MYB21(Myeloblastosis 21)and MYB24 proteins during the development of sexual organs. They are essential for the growth of petals. Grand petals and continuous petal expansion are a consequence of enhanced MYB21 expression in aos and coi1 plants (Reeves *et al.*, 2012). Higher expression of bHLH TF BIGPETALp in *opr3* plants controls post-mitotic cell growth, which results in enlarged petals and cells

(Brioudes *et al*., 2009).

Induction of leaf senescence and leaf degradation

Arabidopsis uses the interaction of JA with COI1 and JAZ7 to stimulate senescence in the leaves (Qi *et al*., 2015; Yu *etal*., 2016). Chlorophyll degradation is enhanced by other NAC TF family members, including NAC019, NAC055, and NAC072, which function downstream of MYC2/3/4 (Melotto *et al*., 2006). Subgroup IIIdbHLH TFs competitively bind to their specific promoters to avoid leaf senescence (Qi *et al*., 2015). By physically interacting with JAZ4/8 when JA is present, WRKY57 functions as an adverse regulator of leaf senescence (Jiang *et al*., 2014). Additionally, YABBY1 and YABBY3 proteins interact with JAZs to facilitate the degradation of chlorophyll (Boter *et al*., 2015).

Stamen development

It has been discovered that JA-deficient mutants, such as coi1, lox3 (Lipoxygenases), lox4, aos, opr3, fad3 (Acyl-lipid omega-3 desaturase), fad7, fad8, dad1 (Defective in anther dehiscence1), JAZ13A, JAZ10.4 and CYP94B3(Jasmonoyl- isoleucine-12-hydroxylase) overexpression lines have been identified in male sterile Arabidopsis plants. The development of stamen is compromised in these mutants (Song *et al*., 2013). Exogenous JA administration can restore stamen growth in plants lacking JA production, rather than in JA signaling mutants. Re-expressing COI1 in various tissues, including filament epidermis or anthers, can reestablish anther dehiscence, filament elongation and pollen maturation (Jewell and Browse, 2016). In response to interactions with JAZs, the R2R3-MYB TFs MYB21, MYB24 and MYB57 produce short filaments, non-viable pollen grains, and delayed anther dehiscence. Stamen formation has been restored in *coi1-1* plants through overexpression of MYB21 proteins (Song *et al*., 2011). By physically binding to MYC2, MYC3, MYC4, and MYC5, MYB21, and MYB24 control the development of stamens, whereas overexpressing MYC5 and MYC3 increases stamen maturation and plant productivity (Qi *et al*., 2015).

Inhibition of apical hook formation

In plants grown in darkness, the COI1-JAZs-MYC2/3/4 cascade prevents the development of apical hooks (Song *et al*., 2014). Transcription factors MYC2, MYC3, and MYC4 are stimulated by JA during dark stress, which affects the transcription of EIN3/EIL1 (Ethylene-insensitive3/ETHYLENE-INSENSITIVE3-like 1). By inhibiting the *HOOKLESS1* gene and preventing apical hook curvature, this regulates the formation of apical hooks (Song *et al*., 2014; Zhang *et al*., 2014). Furthermore, MYC2 encourages EIN3 BINDING F-BOX PROTIEN1, leading to EIN3 degradation (Zhang *et al*., 2014).

Regulation of stomatal closure

Plant stomata control water loss, gas exchange and resistance to phytopathogens. By triggering an H^+-ATPase in the plasma membrane and resulting in an H^+ and Ca_2^+ influx, JA contributes to stomatal closure (Yin *et al*., 2016). Methyl-JA/COI1 stimulates Cl^- and K^+ channels, which in turn causes reactive oxygen species generation, K^+ efflux and Cl^- efflux, which in turn causes the loss of turgor guard cells and stomatal closure (Yan *et al*., 2015). In response to drought and salt stress, plants close their stomata; in Arabidopsis, stomatal closure is modulated by OPEN STOMATA1 protein kinase activation via the JA and ABA pathways (Yin *et al*., 2016). In drought conditions, plants produce OPDA, which is more useful than JA. ABA and OPDA work together to enhance stomatal closure e (Savchenko *et al*., 2014).

Inhibition of hypocotyl growth

In Arabidopsis, jasmonate prevents hypocotyl elongation in response to a variety of light stressors, including bluewavelengths *via* COI1 and far-red light. Under far-red light, the JA-deficient mutant JAR1 plants have extended hypocotyls. Longer hypocotyls are a feature of *coi*1 mutants in far-red, red, or low R/FR light conditions (Robson *et al.,* 2010; Chen *et al.,* 2013). With a low R/FR light ratio and shorter hypocotyls in far-red light, the MYC2/JIN1 mutant exhibits longer hypocotyls in blue light. Under far-red light, jasmonate-insensitive mutant 1 plants have shorter hypocotyls and a lower R/FR light ratio (Robson *et al.,* 2010; Yadav *et al*., 2005). In *Zea mays*, JA inhibits ear shoot maturity, but it does not affect coleoptile maturity in rice or plant growth (Yan *et al*., 2012; Yang *et al*., 2012).

Impact of jasmonic acid on fruit formation and fruit quality

Jasmonic acid (JA), a naturally occurring lipid is involved in the development of fruit, ripening, abscission, senescence of the leaves and regulation of plant growth (Zheng *et al*., 2017). It promotes dormant seed germination in a range of fruits by causing lipid peroxidation and membrane damage during seed germination. In apples, JA derivatives stimulate lipase to promote dormant embryo germination (Dave *et al*., 2011; Ranjan and Lewak, 1992). Early stages of the fruit's development, grape-seeded berries have higher concentrations of JA and MeJA and JA causes grapevine tendril coiling (Kondo and Fukuda, 2001; Malabarba *et al*., 2018). MeJA concentrations in apple seeds are lower than those of trans- and cis-JA when cherry fruits, are harvested (Kondo *et al.*, 2000). The effects of exogenous jasmonates vary based on their concentration and may be related to the concentrations of endogenous jasmonates (Fan *et al.*, 1998b). Firmness and color are improved in apple fruit when PDJ (n-propyl dihydrojasmonate) is applied 20–30 days before harvest (Kondo, 2010). Fruit firmness is increased by applying MeJA at the last stage of fruit development, post-harvest in various cherry varieties (Saracoglu *et al.*, 2017). While MJ on "Kumagai" guavas affects fruit ripening, endogenous jasmonates accumulate in the early ripening stages of sweet cherries and apples (Silva *et al.*, 2017; (Kondo *et al.*, 2000). MeJA treatment of "Kensington Pride" mangoes at the climacteric stage resulted to increased fatty acids, skin color, total aroma volatile, and enhanced fruit ripening through ethylene biosynthesis; additionally, it decreased n-tetradecane production (Lalel *et al.*, 2015). Nevertheless, MeJA fumigation at low temperatures raised the â-carotene concentration without increasing the color change, stem-end rot, or anthracnose (Boonyaritthongchai *et al.*, 2017). Mango fruit quality can be enhanced by applying MeJA, an exogenous agent, without affecting ripening (Gonzalez-Aguilar *et al.*, 2000) Applying JA to papayas before harvest extends their shelf life and improves their quality after harvest. Devarakonda *et al*. (2020) found that applying 150 µM of JA to papaya fruit improves fruit firmness, reduces lipid peroxidation, and reduces weight loss. Additionally, it prolongs the shelf life of strawberries by delaying the deterioration brought on by fungi (Ayala *et al.*, 2005). It has been demonstrated that JA is a substitute degreening agent for mandarin fruits in the citrus industry (Gómez *et al.*, 2017). Antioxidants were increased by a pre-harvest methyl jasmonate spray without compromising lemon fruit quality or yield metrics. Numerous processes are regulated by jasmonate derivatives (Serna-Escolano *et al.*, 2019).

Conclusion

The significant role of jasmonic acid (JA) and its derivatives in modern fruit production, serves as natural hormones that enhance defensive mechanisms against various biotic and abiotic stresses. These compounds, applied as pre- and post-harvest treatments, contribute to the acceleration of defensive responses, protection against chilling injury, and post-harvest management. Moreover, JA promotes the production of essential secondary metabolites, including antioxidants, phenolic acids, flavonoids and anthocyanins. The versatile applications of JA extend to protection against post-harvest fungal diseases, frost, insect attacks and other stressors in fruit production. The urgency for further research on jasmonates and their derivatives, either independently or in combination with other growth regulators, is emphasized. Future studies are anticipated to delve into the intricate molecular mechanisms of JA action, including its movement through transporters, resource allocation, and interactions with other hormonal signaling pathways, thereby expanding our understanding of how JAs contribute to plant resilience against both biotic and abiotic stresses.

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