The Role of Salicylic Acid, Jasmonic Acid and Ethylene in Plant Defense

Firoozeh Chamandoosti

Iranian Research Institute of Plant Protection, Agricultural Research, Education and Extension Organization (AREEO), Tehran, Iran PhD of Cellular and Developmental Biology, Assistant Professor of Iranian Research Institute of Plant Protection Department of Plant Diseases

Abstract— *A complex network of cross – talk between the salicylic acid (SA) and jasmonic acid (JA) pathways further fine tunes plant defense responses. SA can be formed from cinnamate via o – coumarate or benzoate depending on whether the hydroxylation of the aromatic ring takes place before or after the chain – shortening reactions. SA not only functions against biotrophs, but also activates plant resistance against the below – ground disease such as root – knot nematodes. The synthesis of jasmonates and many other oxylipins is initiated by lipoxygenases (LOXs), which catalyze the regio – and stereoselective dioxygenation of polyunsaturated fatty acids. JA activates plant immune responses to necrotrophic pathogens, some phloem – feeding insects and chewing herbivores. Also JA is also involved in other aspects of plant – pathogen interactions, including systemic acquired resistance (SAR). The role of ethylene (ET) in plant diseases resistance is dramatically different duo to type of pathogene and plant species. There are many evidence that show ethylene response is linked to gene for gene resistance. It is proven that there are a strong connection between different pathways related to SA, JA and ET for plant diseases resistance. So that SA – dependent and JA/ethylene – dependent pathwa ys induce expression of different sets of PR genes and result in plant resistance to different pathogens.*

Keywords— Jasmonic acid, Plant Resistance, Salicylic acid.

I. INTRODUCTION

Plant resistance to pathogenic agents usually operates through a complex defense mechanism network. Compounds such as salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) regulate plant defense pathways to trigger appropriate responses to different pathogens [4]. Whereas the SA signaling pathway is mainly activated against biotrophic pathogens, the JA/ET signaling pathway is activated against necrotrophic pathogens [28]. So SA rely on living plant tissue for nutrients [34]; [79] and [13]. In contrast, plants produce jasmonic acid (JA) in response to wounding caused by insects and to necrotrophic microbes which obtain nutrients from dead host cells [13]; [118] and [102]. It can be said that the SA – mediated defenses have a major role in the basal resistance to the bacterial and oomycete pathogens, *Pseudomonas syringae* and *Peronospora parasitica*, respectively, turnip crinkle virus (TCV) and cucumber mosaic virus (CMV) [10]; [86]; [116]; [20] and [38]. In contrast, JA signaling has an important role in the basal resistance to the fungal pathogen *Botrytis cinerea* [13]. ET is a critical third player from the perspective of understanding how plants prioritize and tailor their responses to diverse attackers [1]. Also ET modulates SA related plant defense signaling both positively and negatively.

At the end of this section, it is important to point out that a complex network of cross – talk between the SA and JA pathways further fine tunes plant defense responses [10] and [11]. Most studies have identified antagonistic interactions between the SA – and JA – mediated signaling pathways [112]. But the relationship between SA and JA is not always antagonistic [101]. The following examples that are presented sequential help to clear this concept. The application of JA depressed the activation of the genes for the acidic PR proteins, [Pathogenesis – related protein(s)] which are SA dependent [112]. In rice is demonstrated that JA signalling positively regulates plant resistance to the biotrophic pathogen, *Xanthomonas oryzae* pv. *oryzae* (Xoo), [101**]** possibly due to a common defence system activated by both SA and JA [24].

II. BIOSYNTHESIS OF SALICYLIC ACID

As mentioned above Salicylic acid (SA) is an important signal molecule in plants.

Biochemical studies using isotope feeding have suggested that plants synthesize SA from cinnamate produced by phenylalanine ammonia lyase (PAL). PAL is a key regulator of the phenylpropanoid pathway and is induced under a variety of biotic and abiotic stress conditions. SA can be formed from cinnamate via *o* – coumarate or benzoate depending on whether the hydroxylation of the aromatic ring takes place before or after the chain – shortening reactions. In sunflower,

potato and pea, isotope feeding indicated that SA was formed from benzoate, which is synthesized by cinnamate chain shortening reactions most likely through a β – oxidation process analogous to fatty acid β – oxidation. [35]. Feeding of 14 – labeled phenylalanine and cinnamate to young *Primula acaulis* and *Gaultheria procumbens* leaf segments indicated that SA was formed via *o* – coumarat [106]. In the same plants, labeled SA was also formed after treatment with 14C – labeled benzoate, [106] suggesting that these plants may use both pathways for SA synthesis. Likewise, in young tomato seedlings, SA appeared to be formed mostly from cinnamate via benzoate but after infection with *Agrobacterium tumefaciens*, 2 – hydroxylation of cinnamate to *o* – coumarate was favored [68]. In tobacco and rice, several lines of evidence suggest that SA is synthesized from cinnamate via benzoate [50]; [88] and [84]. First, infiltration of healthy tobacco leaf discs with 0.1 mM benzoate increased total SA level 14 fold after 18 hours [84]. Second, in TMV – infected tobacco, large increases in the levels of benzoate and SA were detected [84]. Third, in both TMV – infected tobacco leaves and rice seedlings, labeled benzoate and SA, but not *o* – coumarate, were detected after feeding with ^{14}C – labeled cinnamate [88] and [84] More label was incorporated into SA when ^{14}C – labeled benzoate was fed than when ^{14}C – labeled cinnamate was used, consistent with benzoic acid being the immediate precursor of [88] and [84]. Similar results were also obtained from the labeling experiments in potato and cucumber [87] and [57]. Furthermore, a benzoic acid 2 – hydroxylase (BA2H) activity was detected in plants including tobacco and rice. In tobacco, the BA2H activity was induced by TMV infection and was partially purified as a soluble 160 kDa protein that could be immunoprecipitated by antibodies against the soluble SU2 cytochrome P450 from *Streptomyces griseolus* [50]. Despite the extensive biochemical and molecular evidence, none of the enzymes required for the conversion of SA from cinnamate in the PAL pathway has been isolated from plants. Although partial purification and immunoprecipitation of a tobacco BA2H activity were reported in 1995, [50], there has been no further report on its purification or isolation of the corresponding gene(s).

III. BIOSYNTHESIS OF JASMONIC ACID

The synthesis of jasmonates and many other oxylipins[1] is initiated by lipoxygenases (LOXs), which catalyze the regio – and stereoselective dioxygenation of polyunsaturated fatty acids [26]; [44]; [33]; [29] and [15]. Linoleic acid (18:2) and linolenic acid (18:3) are oxygenated by specific LOXs at C9 or C13 to result in the corresponding (9S) – or (13S) – hydroperoxy – octadecadi(tri)enoic acids, which feed into at least seven alternative pathways resulting in the formation of a large variety of oxylipins [26] and [44]. The first committed step in the two parallel pathways for JA biosynthesis i.e. the octadecanoid pathway from 18:3 and the hexadecanoid pathway from 16:3 [41], is performed by allene oxide[2] synthase (AOS), an unusual cytochrome P450 which uses its hydroperoxide substrate as source for reducing equivalents and as oxygen donor, and is thus independent of molecular oxygen and NAD(P)H. AOS catalyzes the dehydration of 13(S) – hydroperoxy – octadecatrienoic acid (13 – HPOT) to form an unstable allene oxide, 12,13(S) – epoxy – octadecatrienoic acid (12,13 – EOT). In aqueous media, 12,13 – EOT rapidly decomposes to – and – ketols, or undergoes cyclization to form 12 – oxo – phytodienoic acid (OPDA). As opposed to spontaneous cyclization which results in a racemic mixture of OPDA enantiomers, allene oxide cyclase (AOC) ensures the formation of the optically pure 9S,13S enantiomer. Dinor – OPDA (dnOPDA) is generated in the parallel pathway from 16:3 The short half – life of 12,13 – EOT in aqueous media (26 s at 0◦C and pH 6.7, [81]; [60] and the optical purity of endogenous OPDA [22] suggest tight coupling of the AOS and AOC reactions in vivo. However, physical contact of AOS and AOC in an enzyme complex does not seem to be required for stereochemical control of the cyclization reaction [90]. Only 9S,13S – OPDA, i.e. one out of four possible OPDA stereoisomers, is a percursor for biologically active JA. AOC is thus crucially important to establish the enantiomeric structure of the cyclopentenone ring. The crystal structure of *Arabidopsis* AOC2 has recently been solved shedding light on how the enzyme exerts stereochemical control on the cyclization reaction [27]. Considering the fact that cyclization occurs spontaneously in aqueous solution, AOC2 does not

[1]- constitute a family of oxygenated natural products which are formed from fatty acids by pathways involving at least one step of dioxygen-dependent [15].
Oxylipins are derived from polyunsaturated fatty acids (PUFAs) by COX enzymes (cyclooxygenases), by LOX enzymes (lipoxygenases), or by cytochrome P450 epoxygenase [115].

[2] - In organic chemistry, an allene (is a compound in which one carbon atom has double bonds with each of its two adjacent carbon centres.) oxide is an epoxide of an allene. The parent allene oxide is $CH_2=C(O)CH_2$, a rare and reactive species of only theoretical interest. Typical allene oxides require steric protection for their isolation. Certain derivatives can be prepared by epoxidation of the allenes with peracetic acid. Allene oxides tend to rearrange to cyclopropanones [114].
Despite the esoteric character of synthetic allene oxides, allene oxides occur naturally. They are intermediates in the chemical defense of some plants against attack by herbivores. Specifically, a hydroperoxide of linolenic acid is the substrate for the enzyme allene-oxide synthase. The resulting allene oxide in turn is converted by allene oxide cyclase to jasmonic acid [2].

need to be much of a catalyst in terms of lowering the activation energy barrier. Indeed, binding of the substrate or the transition state does not involve any induced fit mechanism. The hydrophobic protein environment and very few ionic interactions with a glutamate residue (Glu23) and a tightly bound water molecule, ensure binding and correct positioning of the substrate 12,13 – EOT. Steric restrictions imposed by the protein environment enforce the necessary conformational changes of the substrate's hydrocarbon tail resulting in the absolute stereoselectivity of the AOC2 – mediated as opposed to the chemical cyclization reaction [27].

IV. BIOSYNTHESIS OF ETHYLENE

The biochemistry of ethylene biosynthesis has been a subject of intensive study in plant hormone physiology [36]. The discovery of ethylene as a plant growth regulator can be attributed to the work of the Russian scientist [23]. He reported that dark – grown pea seedlings showed a reduced hypocotyl growth in combination with an exaggerated hypocotyl bending when exposed to illumination gas [23]. Neljubov 1901 [23] could pinpoint ethylene gas as the active component that caused dark – grown pea seedlings to bend, by flowing the illumination gas over several filters prior to exposing the seedlings. This typical ethylene response of dark – grown seedlings was later defined as the triple response: (1) shortening of the hypocotyl and roots, (2) radial swelling of the hypocotyl, and (3) the exaggeration of the apical hook[3] [71].

Ethylene is formed from methionine via S – adenosyl – L – methionine (AdoMet) and the cyclic non – protein amino acid 1 – aminocyclopropane – 1 – carboxylic acid (ACC). ACC is formed from AdoMet by the action of ACC synthase (ACS) and the conversion of ACC to ethylene is carried out by ACC oxidase (ACO) [36]. In addition to ACC, ACS produces 5' methylthioadenosine, which is utilized for the synthesis of new methionine via a modified methionine cycle. This salvage pathway preserves the methylthio group through every revolution of the cycle at the cost of one molecule of ATP. Thus high rates of ethylene biosynthesis can be maintained even when the pool of free methionine is small [70].

Analysis of ACS gene induction in mutant fruit with disrupted ethylene signalling has been used to identify which ACS gene is ethylene – regulated. The Never ripe (Nr) mutant cannot perceive ethylene due to a mutation in the ethylene – binding domain of the NR ethylene receptor [73] and [53]. Fruit from the ripening inhibitor (rin) mutant do not show autocatalytic ethylene production [94]. and cannot transmit the ethylene signal downstream to ripening genes due to a mutation in the RIN transcription factor [52]. Nr and rin mutant fruit have shown that LEACS2 expression requires ethylene whereas LEACS1A and LEACS4 exhibited only slightly delayed expression in Nr indicating that ethylene is not responsible for regulation of these genes [19]. All four fruit ACS genes showed the same expression patterns in rin fruit as in mature green wild – type fruit, but did not show any ripening – related changes of expression [19]. Therefore, it has been proposed that LEACS1A and LEACS6 are involved in the production of system 1 ethylene in green fruit [19]. System 1 continues throughout fruit development until a competence to ripen is attained, where upon a transition period is reached, during which LEACS1A expression increases and LEACS4 is induced. During this transition period, system 2 ethylene synthesis (autocatalysis) is initiated and maintained by ethylene – dependent induction of LEACS2 [19]. Antisense inhibition of LEACS2, which also down – regulated LEACS4, reduced ripening – related synthesis of ethylene to 0.1% of control fruit. The antisense fruit displayed an abnormal pattern of ripening such as reduced lycopene accumulation, delayed softening and a much reduced climacteric peak [91].

V. THE ROLE OF SALICYLIC ACID IN PLANT DEFENSE

Salicyclic acid plays crucial role in various plant – pathogen interactions by activating defense responses. It is typically involved in defense against microbial biotrophs such as the bacteria *Pseudomonas syringae* [7]. SA biosynthetic pathway has been well characterized in plants. It includes two distinct enzymatic pathways, isochorismate synthase (ICS/SID2) – mediated isochorismate pathway and phenylalanine ammonia lyase (PAL) – mediated phenylalanine pathway, which has been reported to be required for both systemic and local acquired resistance as well as PCD [76]. Thus, SA is involved in inducing systemic acquired resistance (SAR) [108] and [67].

Stimulation of defense responses occurs not only at the recognition site of microbes, but also in distal parts of plants to protect undamaged tissues from subsequent microbial pathogen invasion for a long – lasting protection, commonly known as SAR [77]. Evidences from grafting experiments suggest that methyl salicylate (MeSA) is the critical mobile signal for SAR in tobacco plant [105]. SA signaling is activated to suppress viral, bacterial, and fungal pathogens invasion in many plant

[3] - Hook – like structure which develops at the apical part of the hypocotyl in dark – grown seedlings in dicots.

species through up – regulation of pathogenesis – related (*PR*) genes expression and the development of SAR [58]. The role of SA signaling in disease resistance was first illustrated by White and coworkers, who demonstrated that treatment of tobacco with SA, or its derivative aspirin significantly stimulated PR protein accumulation and enhanced plant resistance to tobacco mosaic virus (TMV) infection [97]. Increasing progress revealed that SA played an essential role in resistance against multiple pathogens [40]; [85] and [72]. A study conducted on wheat demonstrated that SA exerted direct and significant impact on *Fusarium graminearum*, the major causal agent of fusarium head blight in wheat; conidia germination and mycelial growth were remarkably inhibited at higher SA concentrations [85]. Exogenous application of SA induces *PR1*, *PR3* (chitinase), *PR5* (thaumatin – like), and *PR9* (peroxidase) gene expression as well as the resistance against bacterial pathogen *Pseudomonas syringae* pv. *syringae* in barley, which is characterized by a concomitant increase in endogenous SA levels [72]. Additionally, foliar application of SA can induce resistance against Fusarium wilt caused by *Fusarium oxysporum* f. sp. *Phaseoli* in common bean which is accompanied with elevated contents of endogenous free and conjugated SA as well as activities of PAL and peroxidases [96]. In tomato, SA treatment induces expession of PR2 and PR3 proteins, activity of PAL, concentration of antioxidants as well as photosynthesis, thereby reducing the infections caused by *Potato virus X* [109]. Over – expression of SA biosynthetic genes or mutation in SA – related genes also affects plant disease resistance [76] and [75]. Over – expression of two bacterial SA biosynthetic genes in transgenic tobacco conferred highly increased SA accumulation accompanied with upregulated defense genes expression particularly those encoding PR proteins, and thus enhancing plant resistance to viral and fungal infection [75]. Transgenic tobacco plants with low or no salicylic acid biosynthesis capacity are defective to induce SAR [108]. Interestingly, exogenous SA application could establish SAR and enhance disease resistance to pathogens even in the SA biosynthetic mutants [76]. SA and benzothiadiazole (BTH, a SA analogue) treatments on perennial ryegrass (*Lolium perenne* L.) enhance expression levels of *PR* genes and deposition of callose, and thereby minimizing the incidence as well as severity of gray leaf spot disease (causal agent: *Magnaporthe oryzae*) [3]. However, plant responses to SA treatment are strictly dependent on developmental stage for a given pathogen. For instance, exogenous SA induces resistance to *Magnaporthe grisea* in mature rice plants, but not in young rice plants, which is accompanied with increased accumulation of SA and PR proteins, resulting in the formation of hypersensitive reaction lesions [110]. Environmental factors also stimulate SA accumulation and thus influence plant resistance. Cucumber plants exhibit increased resistance against *Sphaerotheca fuligineathan* under red light and this resistance is associated with enhanced SA – dependent signaling [40]. SA not only functions against biotrophs, but also activates plant resistance against the below – ground disease such as root – knot nematodes (RKN) [14] and [37]. RKN are sedentary endoparasite which feed cells for their nutrition and keep its sedentary life cycle [32]. The yield loss in agriculture caused by nematodes is devastating, which accounts for 5% to 12% of annual crop loss worldwide [69]. Branch *et al.* [14] demonstrated that SA was involved in *Mi – 1* – mediated defense response to RKN (*Meloidogyne incognita*) in tomato. SA induces glutathione status in tomato plant and imparts resistance against RKN *M. incognita* [42]. Foliar application of SA is able to trigger SAR response to RKN in tomato roots [99]. To date, numerous scientific studies indicate that activation of SA pathway is important for induced defense in plants against biotrophic pathogens, such as *Pseudomonas syringae* and *Golovinomyces cichoracearum* [103] and [31]. However, there are some exceptions that conflict with the current model. For instance, Novakova *et al.* [82] showed that SA was involved in the resistance against necrotrophic pathogen *Sclerotinia sclerotiorum (Lib.)* de *Bary*, which was previously known to be functioned by JA and ET signaling pathways. In spite of contradictory evidences, it is well accepted that SA is an important endogenous marker and crucial for plant disease resistance against biotrophic as well as necrotrophic pathogens. A number of studies demonstrate that invading pathogens or insects release effectors proteins that interfere or block the plant immune system for the benefit of its own establishment to cause disease development in the plant tissue [25]; [63] and [80]. Virulent *Pseudomonas syringae* produces the bacterial effector proteins virulence factor coronatine (COR), which is a phytotoxin that structurally and functionally mimic methyl jasmonate and suppresses SA – dependent immune defenses, thus the pathogen utilizes the hormone – regulated defense signaling network to promote susceptibility of the host [25] and [63]. Meanwhile, the fungal pathogen *Sclerotinia sclerotiorum* participates actively in the degradation of SA, and the presence of low levels of SA affect growth or oxalate production by *Sclerotinia sclerotiorum* [17]. *Hyaloperonospora arabidopsidis* (Hpa) causes downy mildew in *Arabidopsis thaliana*. During their interaction, Hpa effector suppresses SA – induced interaction and attenuates responses to SA [98]. Interestingly, mollusks secrete phytohormone – like substances into their mucus which contain significant amounts of SA and induce SA – responsive gene *PR1* expression in wounded leaves of *Arabidopsis thaliana*, suggesting the potential of mucus in regulating plant defense [49]. It is now clear that SA plays important role in activating plant defense mechanism and therefore application of SA could be considered as a feasible strategy to minimize biotic stress induced yield reduction in commercial agriculture.

VI. THE ROLE OF JASMONIC ACID IN PLANT DEFENSE

As previously mentioned Jasmonic acid and its metabolites, including methyl jasmonate (MeJA), are lipid – derived compounds originating from plastid membrane a – linolenic acid. They act as signals to mediate plant growth and developmental processes, as well as plant responses to biotic and abiotic stresses [2]. Numerous reports indicate that JA activates plant immune responses to necrotrophic pathogens, some phloem – feeding insects and chewing herbivores [48]. JA is also involved in other aspects of plant – pathogen interactions, including SAR. Application of JA to rice plants induces resistance against necrotrophs pathogen *Rhizoctonia solanivia* through the activation of phenylpropanoid pathway [89] Predominantly, JA responses are mediated through the coronatine insensitive 1 (COI1 Fbox protein and *coi1* mutants possess elevated SA levels and exert more resistance to bacterial pathogens [9]. On the contrary, *coi1* plants show greater susceptibility to *Botrytis cinerea* and *Alternaria brassicicola* [13]. Stotz *et al.* [39] unveiled JA – dependent and *COI1* – independent defense responses against *Sclerotinia sclerotiorum* in *Arabidopsis thaliana*; however, AUXIN RESPONSE FACTOR 2 (ARF2) acts as a negative regulator of defense responses against this pathogen. The environmental factors such as light quality can influence plant resistance via JA pathway. Low red/far – red ratios reduce *Arabidopsis* resistance to *Botrytis cinerea* and JA responses via a COI1 – JAZ10 – dependent and SA – independent mechanism [43]. Increasing evidences have proved that JA is critical for plant resistance against plant disease RKN [65] and [61]. After the foliar treatment with MeJA, the expression profiles of JA genes were induced resulting in highly enhanced resistance against RKN (*Meloidogyne incognita*) in tomato plants [111]. Transcript profiles from microarray analyses indicate that an intact JA signaling pathway is required for tomato susceptibility to RKN [66]. Several studies demonstrated that other hormones might function to suppress RKN through the crosstalk with JA pathway. Nahar *et al.* [62] reported that BRs suppressed the JA biosynthesis and signaling pathway under RKN infestation in rice and thus determined the outcome of the rice – RKN interaction through crosstalk between BR and JA pathway. Exogenous application of MeJA to the rice shoots also induces a strong systemic defense response in the roots, making the plant more resistant to nematodes infection, while ET activates the JA biosynthesis and signaling pathway in a facultative manner, indicating a pivotal role of JA pathway in systemically induced resistance against RKN in the root [61]. Environmental factors also play remarkable role in determining severity of RKN infection through the activation of JA pathway. Elevated CO2 changes the interactions between nematode and tomato genotypes differing in the JA pathway. CO2 enrichment possibly impairs the allocation of plant resources between growth and defense by affecting contents of secondary metabolites, volatile organic compounds and C: N ratio that eventually reduces plant resistance to RKN in tomato [121] and [122]. In addition to well established role in resistance against necrotrophic pathogens in various plant species, JA has also been reported to induce plant resistance against biotrophic pathogens [124] and [5]. Exogenous MeJA significantly induced nine PRs gene expression and enhanced the powdery mildew resistance in the susceptible varieties of wheat, suggesting that JAs play important role in defense against wheat powdery mildew infection and manipulation of JA pathway through breeding may improve powdery mildew resistance in wheat [124]. Exogenous application of JA can also enhance resistance to the bacterial blight and blast caused by *Xanthomonas oryzae* pv. *oryzae* [101] and *Magnaporthe oryzae*, respectively in rice [83]. It's worth mentioning that rice plants constitutively expressing the pathogen – responsive WRKY30 gene showed enhanced resistance against fungal pathogens *Rhizoctonia solani* and blast fungus *Magnaporthe grisea*, concomitantly with increased endogenous JA accumulation and induction of JA biosynthesis related genes expression [119].

VII. THE ROLE OF ETHYLENE IN PLANT DEFENSE

Plants have evolved sophisticated detection and defense systems to protect themselves from pathogen invasion. Ethylene seems to play an important role in various plant disease resistance pathways. However, depending on the type of pathogen and plant species, the role of ethylene can be dramatically different. Plants deficient in ethylene signaling may show either increased susceptibility or increased resistance. For example, in soybean, mutants with reducedethylene sensitivity produce less severe chlorotic symptoms when challenged with the virulent strains *Pseudomonas syringae* pv *glycinea* and *Phytophthora sojae*, whereas virulentstrains of the fungi *Septoria glycines* and *Rhizoctonia solani* cause more severe symptoms [113]. Simiarly, in *Arabidopsis*, the ethylene – insensitive mutant *ein2* develops only minimal disease symptoms as the result of enhanced disease tolerance when infected by virulent *P. syringae* pv *tomato* or *Xanthomonas campestris* pv *campestris* [8]. However, the *ein2* mutant also displays enhanced susceptibility to the necrotrophic fungus *Botrytis cinerea* [12]. On the basis of these observations, ethylene seems to inhibit symptom development in necrotrophic pathogen infection but enhances the cell death caused by other type of pathogen infection. In fact, *Arabidopsis* protoplasts isolated from the *etr1 – 1* mutant display reduced cell death from the fungal toxin fumonisin B1 [107 and presence of the *ein2* mutation reduces cell

death in the accelerated *cell death 5* (*acd5*) mutant [59] supporting a role for ethylene in the regulation of programmed cell death.

Upon pathogen infection, the avirulence signal (*avr*) carried by pathogens is recognized by a specific plant resistance (*R*) gene product [64]. This avr/R interaction is called gene – for – gene resistance and often triggers a strong defense mechanism that includes the programmed cell death of plant cells at the site of infection (known as the hypersensitive response), resulting in efficient containment of the pathogen. In tomato, it has been demonstrated that a direct interaction between the *R* gene *Pto* and the avirulence gene *avrPto* in the *P. s. tomato* strain determines gene – for – gene specificity in this plant – pathogen interaction [104] and [95]. Recently, a transcription factor, Pti4, has been identified on the basis of its specific interaction with Pto [123]. Interestingly, this Pti4 protein shares extensive similarity with the amino acid sequences of EREBPs and can specifically bind the GCC – box cis element present in the promoter of many ethylene – regulated pathogen – related (*PR*) genes. Expression of *Pti4* in tomato leaves is rapidly induced by ethylene, and this induction precedes expression of GCC – box – containing *PR* genes. Moreover, phosphorylation of Pti4 by the Pto kinase enhances its binding to the GCC box. These results provide evidence that the ethylene response is linked to gene – for – gene resistance in tomato.

VIII. INTERACTION AMONG THE SALICYLIC ACID, JASMONIC ACID AND ETHYLENE IN PLANT DEFENSE

Activation of the hypersensitive response triggers a longlasting response known as systemic acquired resistance, which provides immunity against subsequent infections caused by a broad spectrum of pathogens [51]. In many cases, systemic acquired resistance is characterized by an increase in endogenous salicylic acid (SA) levels and expression of a subset of *PR* genes, as well as enhanced resistance to a broad spectrum of virulent pathogens. However, some pathogens can induce plant defense responses via activation of the ethylene and JA signal transduction pathways. *Arabidopsis* plants with defects in ethylene perception (*ein2*) or JA signaling (*coi1*) fail to induce a subset of *PR* gene expression, including the plant defensin gene *PDF1.2*, a basic chitinase (*PR-3*), and an acidic hevein – like protein (a lectin – like protein from *Hevea brasiliensis*) (*PR-4*), resulting in enhanced susceptibility toward certain pathogens [46]. Interestingly, the induction of *PDF1.2* requires both intact JA and ethylene signaling, whereas the majority of other responses mediated by these hormones are specific to only one of the signals. This suggests that the ethylene and JA pathways interact with each other, co – regulating expression of some genes involved in plant defense. Because only a small subset of genes is affected by both signals, the interaction between these two pathways is likely to be downstream, possibly at the level of the specific defense gene promoters. Nevertheless, ethylene and JA signaling may also function independently to regulate distinct processes in defense response. A recent study has shown that pathogen – or elicitor – induced accumulation of the defense compound 3 – indolylmethylglucosinolate is mediated by JA but not by ethylene or SA [30] indicating that ethylene and JA pathways may have different roles in disease resistance. Although SA – dependent and JA/ethylene – dependent pathways induce expression of different sets of *PR* genes and result in plant resistance to different pathogens, there appear to be considerable interactions between these two pathways in systemic acquired resistance. Here, use of the word "cross – talk" is reserved for communications between two separate, linear signal transduction pathways that are simultaneously activated in the same cell. Therefore, the components of the two signaling pathways have to be (1) shown to be expressed in the same cell and (2) demonstrated to physically interact under normal physiological conditions [100]. A recent survey of changes in the expression levels of 2375 selected genes upon pathogen infection or SA, JA, and ethylene treatment had revealed that although some genes are affected by one signal or another, many respond to two or more defense signals [92]. These results indicate the existence of a substantial network of regulatory interaction and coordination among different plant defense pathways. For example, two *Arabidopsis* mutants that constitutively express *PR* genes, *cpr5* and *cpr6*, express both *PR – 1* and *PDF1.2* genes in the absence of pathogen infection. Although the constitutive expression of *PR – 1* is dependent on SA, it is only partially suppressed by the *npr1* (for non – expressor of *PR – 1*) mutation, a gene that is required downstream of SA to activate PR – 1 gene expression, indicating the existence of a SA – mediated, NPR1 – independent response [55]. Only when ethylene signaling is also blocked by *ein2* in addition to *npr1* mutation in *cpr5* and *cpr6* mutants is *PR – 1* gene expression abolished completely. Furthermore, *ein2* potentiates SA accumulation in *cpr5* and dampens SA accumulation in *cpr6*. These results suggest the existence of interactions between ethylene – and SA – dependent signaling through an NPR1 – independent pathway. Interestingly, a suppressor of *npr1*, *ssi1*, which completely bypasses NPR1 function, constitutively expresses the JA/ethylene – dependent marker *PDF1.2* gene in an SA dependent manner, suggesting that SSI1, together with CPR5 and CPR6, may participate in the interactions between the SA – and JA/ethylene – dependent pathways. Recently, a null mutation in the *EDR1* gene has been shown to enhance resistance to *P. syringae* and *Erisyphe cichoracearum*, and causes rapid activation of defenserelated genes such as *PR – 1* [16]. This enhanced disease resistance depends on the SA – induced defense response pathway and is independent of the JA/ ethylene pathway. However, *PR – 1* gene expression, which

is SA – dependent, is highly induced by ethylene treatment in*edr1* mutant plants, whereas it is almost undetectable in wild – type plants. This again suggests that there is significant interaction between the ethylene and SA – dependent pathways. In this case, ethylene potentiates SA – mediated *PR – 1* gene expression, and EDR1 negatively regulates this process. Removal of EDR1 produces a dramatic effect of ethylene on SA – dependent responses, resulting in enhanced disease resistance in *edr1* mutant plants. *EDR1* encodes aputative MAPKKK similar to CTR1, but unlike the *ctr1* mutant, *edr1* does not display ethylene response phenotypes. There are many other examples of similar interaction between the SA and JA/ethylene pathways. Perturbations in SA – dependent signaling have been reported to affect JA/ethylene-dependent signaling represented by *PDF1.2* expression [45] ; [46]; [54]; [55]; [47]; [117] and [79]. It has been noticed that there is a correlation between a decrease in SA levels and increased *PDF1.2* expression, indicating that SA may have an inhibitory effect on JA/ethylene biosynthesis or signaling [21]. Consistent with this observation, *PDF1.2* mRNA accumulates at higher levels in mutants defective in SA signaling compared with levels in the wild type after *B. cinerea* infections [56]. This may also explain why mutants that disrupt SA – mediated responses become sensitized for activation of the JA/ethylene pathway [54] ; [55] and [117]. On the other hand, JA/ethylene can also repress the expression of SA – induced genes by inhibiting SA accumulation. For example, the *mpk4* (for MAP kinase 4) mutant, which has elevated SA levels and constitutive activation of SA – dependent signaling, failed to induce the expression of *PDF1.2* gene upon JA treatment [78]. This could result from high SA levels antagonizing JA/ethylene signaling as described above. However, when the *mpk4* mutant is crossed to plants carrying the *nahG* transgene, which encodes an enzyme that degrades SA, activation of *PDF1.2* expression is still blocked in the *nahG mpk4* double mutant. These results suggest that block in JA/ethylene signaling relieves the suppression of SA signaling. Nevertheless, it should be pointed out that because both JA – and ethylene – dependent pathways are involved in regulating *PDF1.2*, changes in this gene expression may not always reflect an alteration in the ethylene – dependent pathway. In fact, although *mpk4* dwarfism was similar to that of the ethylene constitutive triple – response mutant *ctr1*, MPK4 does not act in the ethylene response pathway between CTR1 and EIN2 [78]. Recent studies of an ethylene pathway gene *ERF1* have shown that activation of ethylene responses by *ERF1* overexpression in *Arabidopsis* plants is sufficient to confer resistance to *B. cinerea* but reduces SA – mediated tolerance against *P. s. tomato* DC3000 [74] suggesting a negative regulation between ethylene and SA responses. Despite the above – mentioned antagonistic interactions, there are examples in which both ethylene – and SA – dependent pathways cooperate on defense – related responses. In *Arabidopsis*, both ethylene and SA signal transduction pathways are necessary to mount an effective defense response against *Plectosphaerella cucumerina* [74]. In tomato, the transgenic ethylene – underproducing ACC deaminase line (*ACD*) and the ethylene –insensitive mutant *Nr* show reduced accumulation of SA upon *X. campestris* pv *vesicatoria* infection, resulting in less severe disease symptoms [93]. Taken together, these results demonstrate that both positive and negative interactions between ethylene and SA pathways can be established, depending on the type of pathogen or specific defense responses. This is consistent with the results that *ein2* mutation increases SA accumulation in the *cpr5* mutant but decreases SA levels in the *cpr6* mutant, which represents a distinct resistance pathway regulated by CPR5 [55].

IX. CONCLUSION

As sessile organisms, plants are under frequent attack from a broad spectrum of microbial pathogens, including biotrophic and necrotrophic pathogens, namely biotrophs and necrotrophs respectively. SA is a crucial defense signal molecule against biotrophs. Also ethylene and jasmonate, play a major role in defense responses against necrotrophs. Although SA – dependent and JA/ethylene – dependent pathways induce expression of different sets of PR genes and result in plant resistance to different pathogens, there appear to be considerable interactions between these two pathways in systemic acquired resistance.

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