Life Science Informatics Publications
Research Journal of Life Sciences, Bioinformatics, Pharmaceutical and Chemical Sciences
Journal Home page http://www.rjlbpcs.com/
RJLBPCS
ISSN 2454-6348

Original Review Article **DOI - 10.26479/2018.0401.18**

MOLECULAR MECHANISMS OF HORMONAL REGULATION OF PLANT IMMUNITY TO BACTERIAL PATHOGENS

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ABSTRACT: Recent years have seen a tremendous progress in our understanding of plant immunity in terms of the ever-widening horizon of hormonal influence on it. This review discusses some of the key finding in the hormonal signaling and response pathways in relation to their fine-tuning of plant health status and the molecular underpinnings of plant immunity.

KEYWORDS: PAMP, PRR, PTI, ETI, Hormone

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1.INTRODUCTION

Plant-pathogen interaction involves a co-evolutionary arms race to outsmart one another (Rausher et al. 2001; Boller& He 2009). There has been a tremendous progress in our understanding of plant immunity in recent years, especially with the discovery of various facets of plant innate immunity (Chisholm et al. 2006; Jones & Dangl, 2006; Boller & He, 2009; Dodds & Rathjen, 2010; Spoel & Dong , 2012).To counter pathogenic insults plants have evolved receptors called pattern recognition receptors(PRRs) to identify conserved determinants in the pathogens known as pathogen associated molecular patterns(PAMPs), so as to mount PAMP-triggered immunity(PTI),the first line of defense. In an effort to overcome PTI, pathogens bring into play a set of proteins, the effectors, to bring about effector triggered susceptibility (ETS) through the direct modulation of PRRs or by meddling with downstream pathways. Successful plants, in turn, employ a second set of weapons, the resistance proteins(R gene products) to set in motion effector triggered immunity(ETI), a robust defense response, to get the upper hand (Jones & Dangl 2006;Macho et al. 2014).ETI and PTI have various features in common including a localized burst of reactive oxygen species at the site of attempted

pathogen attack, that culminates in programmed cell death which prevents further spread of the pathogen to the healthy parts of the host (Pieterse et al. 2009). Plant pathogens trigger a wide variety of symptoms indicative of a role of hormones. Cankers and galls of trees, foolish seedling disease of rice, premature senescence, green island in many plants- all points to the fact that hormones have a say in plant-microbe interactions. Recent works have lead to the elucidation of the intricate fashions in which hormonal perturbations can result in disease or disease resistance in plant, depending on the plant-pathogen system, the types of hormones involved, and the environment, among other factors. The focus of this review is on how hormonal signaling may influence the fate of plant-pathogen interactions. Hormones such as jasmonate(JA) / ethylene(ET) and salicylic acid (SA) have long been known to play a role in response to insect herbivory and necrotrophy, and in systemic acquired resistance (SAR), respectively. More recent works have unearthed the roles of other hormones in this regard.

SALICYLIC ACID (SA):

Salicylic acid (SA) plays a major role in disease resistance signaling (Vlot et al 2009).SA biosynthesis occurs during PTI (PAMP-triggered immunity) and ETI (Effector-triggered immunity) (Mishina and Zeiger 2007). Rapid microbe-stimulatedriseinCa^{2+} levels are important early signaling events upstream of SA biosynthesis (Du et al., 2009). When ETI is initiated by TIR-NBS-LRR-type R proteins, SA biosynthesis is mediated by EDS1 and PAD4, lipase-like proteins (Wiermer et al 2005), but when CC-NBS-LRR type proteins trigger ETI, NDR1 functions in the onset of SA production (Barnoux et al., 2011).Ca^{2+}/ calmodulin-binding transcription factor, AtSR1 (also called CAMTA3), represses *EDS1* transcription which reduces SA accumulation (Du et al., 2009).Salicylic acid is a key component of local immunity and systemic acquired resistance (SAR) in plant. Local immunity is often characterized by programmed cell death (PCD) (Brodersen et al. 2005). However, the autophagy pathway proteins like BECLIN1 restrict the spread of PCD beyond the site of attempted attack to the healthy parts of the plant (Liu et al. 2005). Several long-distance signals that trigger SAR have been identified (Park et al. 2007; Jung et al. 2009; Chanda et al. 2011; Fu & Dong, 2013). NONEXPRESSOR of PR GENES 1(NPR1) upon activation by SA acts as a transcriptional co-activator of a large number of defense genes which include *PR1* (Moore et al 2011;Dong 2004), playing a central role in both SAR and induced systemic resistance(ISR).When SA is absent, NPR1 is mostly localized in the cytoplasm, where it remains as multimers through intermolecular disulfide bonds that are facilitated by S-nitrosylation of NPR1 via S-nitrosoglutathione (Tada et al 2008;Robert-Seilaniantz et al. 2011). Upon SA treatment, SA interactions with hemecontaining ROS scavenging enzymes, such as catalase, could result in redox stress leading to the release of NPR1 monomers by the activity of the thioredoxins TRX-H3 and TRX-H5 and their entry into the nucleus (Durner & Klessing 1995; Mou et al 2003; Tada et al 2008). Once inside the nucleus, NPR1 binds to TGA (TGACG motif binding) transcription factors, facilitating their binding to SA-responsive

promoters (Robert-Seilaniantz et al. 2011), ultimately turning on SA response genes such as *PR1*. During this process, NPR1 becomes sumoylated as well as phosphorylated and subsequently ubiquitilated by Cullin 3 E3 ubiquitin ligase with high affinity for phosphorylated NPR1,eventually leading to its degradation by proteasome, probably allowing new NPR1monomers to reinitiate the transcription cycle. Moreover, sumoylation is necessary for enhanced immunity. (Spoel et al 2009; Corne et al 2012; Saleh et al. 2015).Again, NPR1 also imparts immunity as corepressor for WRKY70, a repressor of SA response genes like PR1.SA level rise coincides with dephosphorylation of NPR1 at Ser 55/59 residues through an as yet unknown mechanism, allowing it to be dissociated from WRKKY70 and subsequently sumoylated(Saleh et al. 2015). The SA receptors, NPR3 and NPR4, serve as adaptor proteins between Cullin 3 ubiquitin liagase and NPR1 by facilitating NPR1 proteasomal degradation and have different affinities for SA (Fu et al. 2012; Yan &Dong 2014).SA has recently been shown to trigger potentially harmful DNA damage as part of defense resposnse.SNI1 (suppressor of npr1-1, inducible 1), a subunit of Structural Maintenance of Chromosome (SMC) 5/6 complex, negatively regulate RAD17 and ATR, the DNA damage sensors. *sni1* mutants exhibit DNA damage which results in SA-mediated defense gene expression.(Yan et al. 2013).Furthermore, mutants deficient in various components SA biosynthesis and signaling exhibit a root associated microbiota different from that in the wild type plant, implicating SA in overall health of the plant. Compared to the wild type plants the mutants impaired in SA response have lesser survival rate in wild soil. Although the microbes have been found to utilize SA both as a signaling molecule and nutrient source, the underlying mechanism of this differential microbial assemblage remains elusive at present (Lebeis et al. 2015).Interestingly, a connection between circadian rhythm and plant immunity is emerging (Spoel & van Ooijen, 2014; Hsu & Harmer, 2014). Several genes involved in programmed cell death(PCD) in response to biotrophic pathogens, as part of both PTI and ETI, have been found to be directly controlled by the MYB-like transcription factor CIRCADIAN CLOCK-ASSOCIATED 1(CCA1), a component of the morning clock (Wang et al. 2011; Hsu & Harmer, 2014).Daily fluctuations in the cellular redox status(NADPH and NADP+ levels) follow a rhythmic pattern leading to the strengthening of the defense response at dawn and a reduction in fitness costs in the evening via NPR1-driven temporal regulation of transcription of *LHY* and *TOC1*, morning and evening clock genes, respectively.(Hsu & Harmer, 2014; Zhou et al. 2015).

JASMONATES (JA):

Many jasmonate responses are mediated through the activity of the SCF ^{COI1}E3 ligase complex containing the coronatine-insensitive 1(COI1) F-box protein (Devoto et al 2002).SCF^{COI1}degrades a group of jasmonate signaling repressor(JAZ) proteins, which together with the F-box protein form the coreceptor complex for JA-Ile, in the presence of jasmonoyl-L-isoleucine (JA-Ile) de-repressing positive regulators of JA response genes such as MYC2 (Chini et al 2007; Thines et al 2007; Sheard et al. 2010,).The COI1 protein, which, in itself is degraded via the ubiquitin proteasome pathway, is

stable when in the SCF^{COI1} complex(Yan et al. 2013).JA induction of defense to the necrotrophic pathogen *Botrytis cinerea* is subject to fluctuations in environmental factors. Plants growing under a leaf canopy receive more far-red light compared to the red light, the photosynthetically active radiation. As a means of compensation to access more light, shade-grown plants grow vigorously by a process called shade avoidance response. Shade avoidance response is, on the other hand, hamper JA defense through the stabilization of JAZs and turnover of MYCs.MYC destabilization involves E3 ubiquitin ligases CONSTITUTIVE PHOTOMORPHOGENIC1(COP1) and PLANT U-BOX PROTEIN10(PUB10) (Chico et al 2014; Jung et al. 2015). In the absence of JA, JAZ engagement with MYC2 involves the recruitment of the corepressor TOPLESS(TPL) via the EAR-motif containing protein Novel Interactor of JAZ(NINJA) (Pauwels et al. 2010).JAZ9, for instance, causes a disruption of interaction between MED25, a subunit of the meditor complex, and MYC3, by occupying the N-terminal fold in MYC3 by an intact helix in its JAS motif, in the absence of the hormone.(Zhang et al. 2015).The DELLA repressors add another tier of regulation by sequestering JAZ through competitive binding in the absence of GA, thereby making MYC2 available for binding to the G-box of JA responsive genes to transcriptionally activate them (Navarro et al., 2008; Hou et al.,2010; Wild et al., 2012; Yang et al., 2012).SA and JA confer plant defense against biotrophic and necrotrophic pathogens, respectively (Glazebrook 2005). JA, by itself, triggers responses to herbivory, while, in presence of ethylene (ET), it acts against necrotrophic pathogens (Grant et al 2009). JA and SA signaling are generally mutually exclusive (Kazan and Manners 2008; Koorneel and Pieterse 2008; Kazan et al. 2012; Van der Does et al. 2013). As a result, plant tissues usually can activate either SA or JA signaling, but not both (Grant et al 2009). Balance between SA and JA can determine the outcome of the disease. Selection favours pathogens that can tilt this balance in their favour (Grant et al 2009).JA effect on necrotrophic pathogens is governed by the transcription factors ORA59 and ERF1 through their positive regulatory effects on *PDF1.2* whereas JA response to herbivory and wounding depends on MYC2 regulation of *VSP1* gene. There is an antagonistic relation between these two JA response pathways.(Kazan & Manners, 2013; Wasternack & Hause 2013; Lyons et al. 2013).MYC2 regulation of gene expression is further shaped by chromatin modifiers and remodelers and heterodimerzation with other MYC transcription factors. MED25 activates MYC2 responsive genes following interactions with MYCs (Kazan & Manners, 2013).To take advantage of SA-JA antagonism,many strains of the phytopathogenic hemibiotrophic bacterium *Pseudomonas syringae(Pst)* produce coronatine(COR),a phytotoxin that mimics JA-Ile, resulting in the activation of JA signaling with consequent suppression of SA signaling , rendering the host susceptible(Brooks et al 2005). COR binding to a JAZ/SCF^{COI1}complex promotes ubiquitination followed by degradation of JAZ proteins in 26S proteasome,derepressing JA-response genes(Katsir et al 2008; Robert-Seilaniantz et al 2011). On the contrary, the necrotrophic fungus *Botrytis cinerea* elicits SA pathway as a virulence mechanism by secreting exopolysachharide(EPS) to mitigate JA-induced defense.(El

Oirdi et al. 2011). MYC2 dependent transcription of NAC transcription factors facilitates stomatal opening when the plant is challenged by COR-producing *Pst*.COR also stimulates NAC transcription factor mediated depletion of SA via the downregulation of a SA biosynthesis gene and the activation of a gene coding for SA methyl transferase that converts SA into the inactive volatile methyl salicylate.(Zheng et al. 2012).*Pst* injects the effector protein, AvrB to activate the guard cell H+ ATPase AHA1 to trigger stomatal closure.AHA1, in turn, through an unknown mechanism, facilitates COI1-JAZ interaction to mediate JA signaling for stomatal opening.(Zhou et al. 2015).Similar studies indicate *Pst* effector induced turnover of JAZ via protease or acetyl transferase activities for JA response activation.(Jiang et al. 2013 ; Gimenez-Ibenez et al. 2014). A study using mutant and overexprssor lines of various elements of JA and GA pathways suggests that JAZ repressor binding to DELLA repressors ensures PIF mediated growth promotion, implicating JA in tilting the balance in favour of defense compromising growth (Yang et al. 2012).Aside from JAZ, JAV also functions as a negative regulator of JA mediated defense against insect pest and pathogen and JAV degradation via the ubiquitin-proteasome pathway confers resistance. However, JAV appears to to be unique in being specific to defense, and not developmental, response, adding a new dimension to the growth-defense pivot (Hu et al. 2013).Systemic accumulation of JA serves as a trigger for immune response in tissues distal to the site of infection. However, despite all the progress in our understanding of the JA assisted defense responses, the basis of systemic JA accumulation had remained largely enigmatic until a recent work revealed a correlation between changes in leaf surface potential and JA-Ile level rise. Mutations in *GLUTAMATE RECEPTOR-LIKE* genes which encode an ion channel abolished this effect with a resultant dampening of immune response to wounding (Mousavi et al 2013).

ETHYLENE (ET):

Ethylene receptors (ETR1, ERS1, ETR2, ERS2 and EIN4) in *Arabidopsis* are localized in the endoplasmic reticulum(ER), acting as negative regulators of ET signaling (Robert-Seilaniantz et al 2011). The ethylene receptors interact with another negative regulator, CTR1, encoding a RAF protein kinase (Clark et al 1998). CTR1 negatively controls the downstream elements of the ET pathway and ethylene inactivates CTR1, causing derepression of EIN2 and EIN3, downstream components of the ET signaling pathway(Stepanova et al 2009).EIN3, a short-lived transcription factor, accumulates in the nuclei following an upsurge in ET (Gagne et al 2004; Guo et al 2003). EIN3 positively regulates the expression of *ERF1*that codes for ET-response element binding(EREBP) transcription factors involved in plant defense against necrotrophs (Robert-Seilaniantz et al 2011). ERF1 overexpressors showed enhanced resistance against *Botrytis cinerea*, but are compromised in defense against *Pst* (Berrocal-Lobo et al 2004; Berrocal-Lobo et al 2002). ERF1 regulates the expression of *PDF1.2*, which encodes a defensin (Lorenzo et al 2004).ET stabilizes EIN3 and EIL1 (EIN3 like1), an EIN3 homolog in *Arabidopsis*, by promoting EBF1/EBF2 (F-box proteins) proteasomal degradation (An et al 2010; Chao et al 1997). Contrastingly, EIN3 is

tagged with ubiquitin and degraded in 26S proteasome by $SCF^{EBF1/EBF2}$, in the absence of ET, shutting off ET-response genes(Robert-Seilaniantz et al 2011).Mutations in EIN2, an integral membrane protein which is required for the activity of EIN3 and EIL2 transcription factors upon ET perception, result in reduced expression of *FLS2*transcript and protein. Besides, ethylene insensitive *etr1-1*mutant was impaired in flg22 induced PTI. Further it was shown that EIN3, which binds to the promoters of *ERF1* and *EBF2*in response to ET, can regulate the transcription of *FLS2* and *ein3-1 eil1-1*double mutants like *ein2-5*are compromised in flg22-induced oxidative burst (Boutrot et al PNAS, 2010).Moreover, *etr1* and *ein2* mutants are in ROS generation, stomatal immunity and *FLS2* transcript accumulation (Mersmann et al. 2010).On the other hand, elf18-triggered immunity is regulated in both ET-dependent and ET-independent ways, although unlike in FLS2 pathway, PRR for elf18, *EF-TU RECEPTOR (EFR)* is not downregulated in *ein2* mutant.elf18induces the elevation of *ERF1* and *PR1* transcription through EIN3/EIL1 (Tintor et al. PNAS, 2013).Mitogen activated protein kinase(MAPK) cascade activation is an important early response in PTI and *Arabidopsis thaliana* MPK3 and MPK6 by phosphorylation stabilize ERF6, a transcription factor that regulates *PDF1.2* to confer resistance to *Botrytis cinerea*(Menget al. 2013). Similarly to ERF6, ORA59 also upregulates *PDF1.2*.ET positively regulates *ORA59* transcription through promoter activation by class II TGA transcription factors that are under negative regulation by SA, possibly through *ORA59*promoter-specific negative effect of ROXY19 on the TGA factors.(Zarei et al. 2011; Zander et al. 2012; Chang et al. 2013; Zander et al. 2014; Müller et al. 2015).Danger associated molecular patterns(DAMPs), altered self-molecules, like the Arabidopsis Pep1, which is a 23-amino acid peptide derived from the propeptidePROPEP1, mounts defense upon its perception by thetransmembraneLRR receptor kinases, PEPR1 and PEPR2. *pepr1/pepr2* double mutants exhibit reduced ET-induced resistance against *Botrytis cinerea* and have marked decrease in ET response gene expression, establishing a vital role for ET signaling in DAMP induced immunity (Liu et al. PNAS, 2013). Whereas efl18-initiated activation of the propeptide, *PROPEP2*, relies on EIN2/EIN3/EIL1, *PROPEP3* apparently does not require these components, which underscores the engagement of an ET-dispensable wing of DAMP triggered immunity (Tintor et al., 2013).Again, PEPR pathway confers local immunity requires ET facilitation of JA defense markers, which is subdued by SA pathway, although in PEPR requires coactions of both SA and JA branches of defense response (Ross et al. 2014). Ethylene also plays a significant role in the biosynthesis of callose, an inducible physical barrier to pathogen invasion, through the transcriptional regulation of the MYB transcription factor MYB51 which, in turn, transcriptionally regulate glucosinolate biosynthesis genes (Clay et al. 2009).Surprisingly, in barley unlike in Arabidopsis, SAR induced by bacterial pathogens is SA dispensable, while relying on ERFs as well as ABA (Dey et al. 2014).

AUXIN:

Auxin normally boosts biotrophic pathogens in establishing disease (Chen et al 2007; Navarro et al 2006; Wang et al).On the other hand, auxin has been implicated in enhancing resistance to necrotrophs, such as *Phytophthora cinnammomi,* through the Arabidopsis auxin receptor mutant *tir 1-1* lacking the ability to SCF^{TIR1} E3 ubiquitin ligase mediated proteasomal degradation of AUXIN/ INDOLE-3-ACETIC ACID(AUX/IAA), a repressor of auxin response(Gray et al. 2001;Dharmasiri et al. 2005; Kepinski et al. 2005; Rodrigues et al 2013;Yin et al. 2014,;Eshraghi et al. 2014).Infection by necrotrophic pathogens often lead to changes in auxin physiology a multiple levels starting from biosynthesis, transport, conjugation, signaling to response.(Kidd et al. 2011;Qi et al. 2012; Gonzalez-Lamothe et al. 2012; Mittag et al. 2015). During defense (PAMP-Triggered Immunity(PTI)) induced by flg22(the stretch of conserved 22 amino acids at the N terminus of the bacterial PAMP,flagellin), the mRNA expression of many auxin signaling related genes is suppressed by microRNAs such as miR393 that targets the auxin receptor F-box proteinTIR1 and its paralogs AFB2 and AFB3. Plants overexpressing miR393 are more resistant to *Pseudomonas syringae* which follows a (hemi)biotrophic lifestyle, whereas plants that overexpress AFB1(which is less sensitive to miR393 due to a nucleotide substitution) are more sensitive(Grant et al 2009; Boller& He 2009;Navarro et al 2006).*Pseudomonas syringae* pv *tomato* DC3000 delivers AvrPto effector that interferes with miR393 maturation process (Navarro et al. 2008).In fact, to beat the plant strategy of auxin receptor downregulation, downstream to the receptor recognition event, the hemibiotrophic pathogens like *Pseudomonas syringae* employs the type III effector,AvrRpt2, to promote turnover of AUX/IAA, a repressor of Auxin Response Factor(ARF) mediated auxin responsive gene transcription, to activate auxin response (Cui et al. 2013).Nematode effectors exploit auxin transport and signaling to enhance host susceptibility by interacting with influx carriers or IAA proteins to reduce the abundance of several ARFs to dampen auxin respose.(Lee et al. 2011, Plant Physiol.; Hewezi et al. 2015)PAMPs induce SA accumulation, which antagonizes auxin-induced virulence by repressing auxin-related genes(including TIR1), which stabilizes transcriptional repressors, such as AXR2, of auxin response genes, thus inhibiting auxin responses. (Tsuda et al. 2008; Wang et al. 2007; Robert-Seilaniantz et al. 2011).Auxin signaling represses SA response genes independent of JA.Auxin Response Factor 9(ARF9) regulates the biosynthesis of the phytoalexin, camalexin and glucosinolate, positively and negatively, respectively. Consequently, miR393 overexpressing plants reduced level of camalexin while they have elevated glucosinolate levels,with concomitant susceptibility to necrotroph and resitance to biotroph via SA (Robert-Seilaniantz et al. 2011) Conjugation of auxin to certain amino acids by *GH3* family of amidosynthetases reduces the level of free auxin (Park et al. 2007; Chandler, 2009; Domingo et al. 2009).*GH3.5* Arabidopsis mutant showing elevated expression of the enzyme exhibits SA-dispensable *PR1* upregulation and increased resistance to *Pst*, although exogenous application of SA and Auxin together results in *PR1*expression going down (Park et al. 2007). A recent

report, however, shows that phytopathogenic necrotrphs and biotrophs alike exploit aspartate-conjugated auxin to induce their virulence genes, through the transcriptional activation of *GH3.2*. (Gonzalez-Lamothe et al. 2012).Auxin induced susceptibility to biotroph may not always require interference with SA signaling (Mutka et al. 2013).In fact, it has been shown that a loss-of-function mutant in *WAT1* (*Walls Are Thin1*), a gene required for secondary cell wall formation, is resistant to several vascular pathogens including *Ralstonia solanacearum* and that resistance is due to general repression of tryptophan pathway mediated production auxin and indole glucosinolates, with concomitant overproduction of SA via chorismate, a node of divergence for the biosynthesis of IAA and Indole glucosinolates (Denance et al. 2013). *Fusarium oxysporum*, a root-infecting hemibiotrophic oomycete induces distinct tissue-specific and developmental stage-specific expression pattern of auxin pathway related genes as shown by RNA-seq analysis. (Lyons et al. 2015).However, in spite of the dual roles played by auxin in biotrophic and necrotrophic infection, through auxin biosynthesis and transport mutants, it is established that auxin in absolutely necessary for the smooth transition JA elevation to ultimately SA mediated SAR response.(Truman et al. 2010).In summary, the effect of auxin depends on one or more of the following factors: the life-style, race and nature(whether or not it is virulent) of the pathogen, the tissues involved, the stage of infection, location of the tissue from the site of pathogen attack or attempted infection, (Local defense versus SAR) and crosstalk with other hormones.

GIBBERELLIN (GA):

GA activates $SCF^{SLY/GID2}$ complex to initiate ubiquitilation and 26S proteasome-mediated degradation of DELLA family of GRAS transcriptional repressors turning on GA response genes. A loss-of-function mutant in four of the five Arabidopsis DELLA-encoding genes shows increased resistance to *Pst,* though it is hyper-susceptible to *Alternaria brassicicola,* a necrotrophic fungal pathogen (Santner et al. 2009; Grant et al 2009). Infection by *Pst* upregulates *PR1,* an SA-signaling marker and downregulates *PDF1.2, a* JA/ET-induced gene that encodes a defensin (Navarro et al 2008). DELLAs may potentiate JA signaling, which may explain why the rice “foolish seedling” disease pathogen *Gibberella fujikuroi* produces GA that induces DELLA degradation and thus prevents the host from mounting JA-mediated defense (Grant et al 2009).Stress induces DELLA accumulation, which in turn leads to the upregulation of genes coding for reactive oxygen species (ROS)-detoxification enzymes, lowering ROS levels (Navarro et al 2008). Indeed, DELLAs physically interact with and hinder JAZ repressors from repressing MYC2, a basic helix loop helix transcription factor that binds to the JA responsive promoters to impart JA mediated defense. (Hou et al 2010).Additionally, JA delays DELLA degradation and MYC2 binding to the promoter *RGL3*, a DELLA protein encoding gene, transcriptionally activate it, in positive feedback loop.JAZ interaction with DELLA, on the hand, allows for PIF(PHYTOCHROME INTERACTING FACTOR)mediated transcription of GA responsive genes for growth promotion.(Yang et al. 2012;

Wild et al. 2012).Adding a new twist to the growth-defense dynamics is the revelation that DELLA physically interacts with MYC2 and that both JA and GA act synergistically to relieve the repression of MYC2 activation of biosynthetic genes of sesquiterpines, volatile insect-repelling metabolites (Hong et al 2012). During Arabidopsis seedling development a complex scenario arises centering around DELLAs, as a result of an interplay between light, GA and BR.PIF4(PHYTOCHROME INTERACTING FACTOR4) and PIF3(PHYTOCHROME INTERAC TING FACTOR4),positive regulators of skotomorphogenesis(seedling growth in the dark), promote cell elongation by transcriptional activation of genes associated with the process and is degraded via the phosphorylation-coupled ubiquitin proteasome pathway upon light perception by the red light sensing protein phyB. In the absence of GA, the DELLAs bind PIF4 hampering its transcriptional activity. On the other hand, ROS facilitates SA signaling. On the other hand, light-dependent GA level rise leads to its binding to the cognate GID1 receptor facilitating the destabilization of the DELLAs. Moreover, PIF4 interacts with BZR1, a BR pathway regulator, to fine-tune the expression of the PRE family of transcription factors, positive regulators of cell elongation, in high temperature and light. Taken together, it points to a mechanism of integration of the environmental and hormonal pathways with implications in balancing the trade-off between growth and immunity in Arabidopsis seedling.(de Lucas et al, 2008; Feng et al 2008; Oh et al, 2012).SA and GA work synergistically to eliminate DELLAs, indirectly allowing ROS to accumulate as a result of lower levels of ROS-detoxification enzymes (Grant et al.2009).In rice, contrary to the scenario in Arabidopsis, GA appears to play the opposite role as it causes an impairment of resistance to (hemi)biotrophic pathogens like *Xanthomonas oryzae* pv. *oryzae,* the baceterial leaf blight pathogen, and *Magnaporthe oryzae*, the rice blast fungus, though it positively impacts necrotroph defence (Tanaka et al., 2006;Yanget al., 2008 ; Vleesschauwer et al., 2010; Qin et al., 2013).The positive role of GA in immunity in rice-*Pythium graminicola (Pgr)* interaction appears to be influenced by BR at multiple levels. *Pgr* not only induces the production of GA-degrading enzyme, but also downregulate a GA biosynthetic gene. *Pgr* also takes control of GA signaling by transcriptional activation of GA pathway repressors, *SPINDLY* and *EARLY FLOWERING1(EL1)*,presumably through the antagonistic effect of BR (Vleesschauwer et al. 2012).On the contrary, that GA induces susceptibility was confirmed by mutating and overexpressing *Elongated uppermost internode (Eui)* gene that codes for a GA deactivating enzyme. Whereas *eui* mutants in rice are more susceptible to both bacterial blight and rice blast diseases, *Eui* overexpressors are relatively immune to these diseases (Yang et al.2008).

ABA:

The effects of ABA are dependent on pathogen life-style as well as the stage of infection. At the pre-invasive stage, ABA mostly promote defense by the closure stomata, natural ports of entry for pathogens, in particular, the bacterial pathogens. However, at the early invasive stage it has an ambiguous role whereas at the late invasive stage it mostly potentiates infection (Ton et al. 2009).The

seemingly antagonistic role of ABA may be attributable to the components and mechanisms yet to be revealed. ABA-deficient mutants are hypersensitive to the oomycete *Pythium irregulare* and *Leptosphaeria maculans*, a fungus, although they are more resistant to *Botrytis cinerea* (Adie et al 2007; Kaliff et al 2007; Audenaert et al 2002). WRKY transcription factor,WRKY33, and NAC transcription factor,ATAF1, regulate ABA biosynthesis, negatively and positively, respectively, by binding to ABA biosynthesis genes(for instance,*NCED3*).Reduced ABA level is associated with increased virulence of the fungal necrotroph, *Botrytis cinerea*, while the hemibiotrophic bacterial pathogen, *P. syringae* DC3000 makes increased ABA accumulation a part of its virulence repertoire.(de Torres-Zabala et al., 2007; Wang et al., 2009;Windram et al., 2012; Liu et al. 2015).ABA-induced stomatal closure resists *Pst* entry into the host, however, at later stages, bacterial effectors activate ABA biosynthesis to overcome plant basal defenses. *Botrytis cinerea* and *Cercospora* species can themselves make ABA (Inomata et al 2004; Siewers et al 2004). As these pathogens are generally considered necrotrophs, this suggests a role for ABA during an early biotrophic phase before the pathogens switch to necrotrophy. ABA (and also methyl JA) can suppress callose deposition in response to flg22(De Torres-Zabala et al 2007; Clay et al 2009).In the absence of ABA, type 2 phosphatases(PP2Cs) dephosphorylate SNF1-RELATED PROTEIN KINASE 2s (SnRK2s) which otherwise phosphorylate several proteins including plasma membrane-bound ion channels and transcription factors.Plasma membrane localized NADPH oxidase AtRBOH responsible for the generation of superoxide from molecular oxygen during PAMP-triggered oxidative burst, is also a target of SnRK2s.ABA binding to cytosolic receptors PYRABACTIN RESISTANCE (PYR)/PYR1-LIKE (PYL) inhibition of PP2Cs through physical interaction and complex formation, which in turn allows SnRK2s to phosphorylate their targets.(Hubbard et al. 2010; Cutler et al. 2010).OST1/SNRK2.6 phosphorylates SLAC1, a guard cell-specific chloride channel ,the activation of which causes reduction in guard cell osmotic pressure followed by turgor pressure reduction and stomatal closure.(Vahisalu et al. 2008; Negi et al. 2008; Geiger et al. 2009; Lee et al. 2009; Chen et al. 2010). Similarly phosphorylation-induced inhibition of the inward-rectifying potassium channel KAT1 promotes stomatal closure (Miyakawa et al 2013). Stomatal immunity, a pre-invasive immunity, involves perception of PRRs such as lipopolysaccharide (LPS) and flg22.flg22 binding to FLS2 triggers SA elevation, which through exerts a positive effect on the ABA mediated shutting of stomata in a process requiring OST1 and nitric oxide(NO) (Melotto et al. 2006; Cao et al. 2009).However, a recent report suggests that flg22 induced stomatal closure takes place independently of ABA; rather, it occurs through the activation of set of MAPKs(MAPK3 and MAPK6) and the generation of Fatty Acid Hydroperoxides (FAHs) by Lipoxigenase 1(LOX1), causing a SA mediated opening of outward-rectifying anion channel resulting in stomatal closure.(Montillet et al. 2013).

CYTOKININ (CK):

Cytokinin plays a pivotal role in plant pathogen interaction.CK signaling involves its perception by receptor kinases, AHK2, AHK3 and AHK4, localized in the plasma membrane or endoplasmic reticulum. Receptor activation initiates a phosphorelay mediated by phosphotrasfer proteins (AHPs) with the eventual phosphorylation of transcription factors called ARRs. The ARRs have been categorized into two functionally distinct groups: Type B ARRs and Type A ARRs, which regulate CK signaling positively and negatively, respectively.(Hwang et al. 2012).CK favours the development of clubroot disease caused by *Plasmodiophora brassicae* in Arabidopsis(Siemens et al 2006). Transgenic plants overexpressing cytokinin oxidase/dehydrogenase genes exhibited resistance against *Plasmodiophora brassicae* suggesting that cytokinin favours the development of the disease (Siemens et al 2006). *Agrobacterium tumefaciens*, the causal agent of crown gall disease causing tumor-like growth, integrates T-DNA, which harbours a gene for cytokinin biosynthesis (such as the *ipt* gene), into the host plant genome, which may favour the pathogen by at turning the infected part into a sink (Choi et al. 2011).Long back the hormone was implicated in the activation of a Cdc25-like phosphatase that removes an inhibitory phosphate group from the Cdc kinase(Zhang et al 1996). Aside from this, cytokinin upregulates *CYCD3* gene that codes for a *D-type cyclin* (Soni et al 1995;Riou-Khamlichi et al 1999).Thus cytokinins (along with auxins), play a crucial role in cell cycle regulation by causing abnormal cell proliferation observed in crown gall. *Rhodococcus fascians*, another gall-inducing pathogen, itself produces certain kinds of cytokinins which are not degraded by plant cytokinin oxidase/ dehydrogenases. AHK4 (ARABIDOPSIS HISTIDINE KINASE 4), a cytokinin receptor that senses the *R. fasciens*-derived cytokinins, is also transcriptionally activated in a positive feedback loop during the infection (Choi et al. 2011; Radhika et al. 2015).Many biotrophic fungal pathogens like *Claviceps purpurea*, the causal agent of ergot of rye, produces copious amounts of cytokinins to potentiate disease (Gironet al. 2013; Hinsch et al. 2015).Global transcript profiling indicates that CKs may be involved in ETI in rice-*Xathomonas oryzae*pv. *oryzae* pathosystem, as the rice expressing the resistance gene*Xa21* show an elevation in CK biosynthesis genes transcripts.(Peng et al. 2015).A recent study shows that *Pseudomonas syringae* pv. *tomato* DC3000 (Pto) effector HopQ1 suppresses PTI by reducing the transcript level of *FLS2*, a PRR, by activating cytokinin signaling (Hann et al. 2014).Despite the well-documented negative role CK in plant immunity, a recent work highlights its positive influence on defense. The study shows that along with TGA3, the cooperative binding of ARR2, a Type B ARR, to *PR1* promoter, following flg22 treatment in Arabidopsis leads to enhanced resistance to *Pseudomonas syringae* pv. *tomato* DC3000 (Pto).The requirement of SA-activated nuclear accumulation active coactivator NPR1 in this process points to the augmentative role CK plays in SA-induced defense pathway (Choi et al. 2010).A modeling of immune network in Arabidopsis-*Pst*, further buttresses TGA transcription factor dependency of SA-CK synergism in *PR1* expression in defense, while it uncovers an antagonistic regulatory nature of

CK and Auxin (Naseem et al. 2012; Naseem & Dandekar, 2012;Naseem et al. 2015). However, CK-SA relationship can also be antagonistic in case of the obligate biotrophic oomycete *Hyaloperonospora arabidopsidis* and the disease outcome may be influenced by the relative concentration of exogenously applied CK concentration, which may be attributed to CK-induced metabolic adjustment (Argueso et al. 2012).

BRASSINOSTEROIDS (BR):

Brassinosteroids play an important part in growth regulation in plant. A critical component of brassinosteroid signaling, BRI1 associated kinase 1 (BAK1), a leucine-rich repeat receptor kinase(LRR-RK), is involved in the regulation of basal defense and programmed cell death(PCD) in plants(Chinchilla et al. 2007;Kemmerling et al. 2007;Heese et al. 2007). Acting as co-receptor BAK1 is known to interact with the BR receptor, BRI1, to mediate BR signal transduction in plants (Li et al.2002b; Nam and Li 2002).The pattern recognition receptor (PRR), FLS2, a ligand-binding LRR-RK that recognizes the bacterial PAMP flg22, a conserved region in flagellin. Importantly, FLS2 initiates PAMP-triggered immunity(PTI) through BAK1which is upregulated in response to PAMPs (such as flg22 and elf18) and mutant *bak1* plants in Arabidopsis are incapable of PAMP responses as shown by loss of ROS burst and growth inhibition following flg22 perception(Chinchilla et al. 2007;Heese et al. 2007).Though BAK serves both BRI1, the brassinosteroid receptor, and FLS as a co-receptor, simultaneous treatment of plant with brassinosteroids (BR) and flg22 caused BR-induced suppression of PTI whereas flg22 failed to have a similar impact on BR-driven growth signaling, tilting the balance in favour of growth compromising immunity, which points to a BAK1-independent process (Albrecht et al. 2012).An optimum level of endogenous BR is required for flg22-triggered immunity. Furthermore, as shown by another study, BR effect on flg22 signaling may also be BAK1-dependent,since plants overexpressing BRI1 negatively regulated BAK1-dependent PTI brought about by flg22,possibly through BRI1 sequestration of BAK1.Conversely, BRI1-actvated pool of BAK1 may feed signaling downstream to FLS leading to promotion of immunity (Belkhadir et al. 2012).This balance is likely influenced by the relative abundance, requirement and affinities of BRI1, BAK1 and FLS2 in both the pathways. Consistent with the model that constitutive activation of resistance has its associated fitness costs, it has recently been demonstrated that BAK1 overexpression in Arabidopsis results in dwarfism (Jones & Dangl, 2006;Van der Hoorn et al. 2008; Domínguez-Ferreras et al. 2015). flg22 perception by the FLS2-BAK1 co-receptor complex initiates sequential phosphorylation events, following the BAK1-dependent phosphorylation of BIK1, a cytoplasmic leucine rich repeat receptor-like kinase, ultimately causing the phosphorylation of FLS2 and BAK1 by BIK1 (Lu et al. 2010; Lin et al. 2014). BIK1 is also phosphorylated by BRI1 upon activation by BR. Phosphorylation of BIK1 causes its dissociation from BRI1 turning on growth signaling (Lin et al. 2013).BIK1 uses the NADPH oxidase RBOHD as a substrate and the phosphorylation of RBOHD following perception of the bacterial PAMPs EF-Tu and flagellin by

EFR and FLS2, respectively, causes ROS mediated immunity (Kadota et al. 2014; Li et al. 2014; Kadota et al. 2015).Constitutive activation of defense has associated fitness costs, necessitating immune homeostasis which seems to be partly achieved by the timely turnover of BIK1 following phosphorylation by the calcium dependent protein kinase CPK28 (Monaghan et al. 2014).The inopportune phosphorylation and activation of BAK1 is also under tight negative control by protein Ser/Thr phosphatase type 2A (PP2A) and BIR2, an RLK and a substrate for BAK1, maintains poise in the absence of any pathogen elicitation, by steric exclusion of FLS2 and prevention of the receptor complex formation through physical interaction with BAK1(Halter et al. 2014;Segonzac et al. 2014).Notably,*bir2* mutants show perfect BR-regulated growth response which points to a point of divergence in the growth-defense equation (Halter et al. 2014).At the transcriptional level, BZR2(BRASSINAZOLERESISTANT 2), the BR-responsive bHLH transcription factor which is negatively regulated by both DELLA and BIN2(BR INSENSITIVE 2),upregulates WRKY transcription factors that target immune response genes abrogating immunity.(Lozano-Durán et al. 2013). Expectedly, armour of pathogen effectors are aimed at various BR signaling associated proteins so as to exert a crippling effect on plant immunity, especially PTI. The black rot pathogen *Xanthomonas campestris* pv. *campestris* type III effector AvrAC, an uridylyltransferase, overcomes both PTI and ETI, via uridylation of BIK1 and RIPK, respectively. Uridylation leads to a reduction in the kinase activity of BIK1 with the resultant attenuation of PTI (Feng et al. 2012).Likewise; BAK1 appears to be the virulence target ofa suit of pathogen effectors, with the corresponding resistance protein for the modified BAK1 still to be identified. Similarly, *P. syringae* pv. *Tomato* DC3000 effector, HopF2, inhibits flg22 stimulated BIK1 phosphorylation (Zhou et al. 2014).The observation that mutant *bak1* plants exhibit enhanced susceptibility to necrotrophic pathogens *Alternaria brasicicola* and *Botrytis cinerea*, whereas they show increased resistance to *H. parasitica* appears to support the dual role of BAK1 in immunity (Kemmerling et al. 2007).The positive regulation of flg22 signaling mediated by BR was shown to cause increased resistance in rice and tobacco (Nakashita et al. 2003).Contrastingly, in rice- *Pythium graminicola* pathosystem, the pathogen hijacks BR signaling to escape GA and SA mediated immune response (Vleesschauwer et al. 2012).

PHYTOSULFOKINE (PSK):

Phytosulfokine is a sulfated pentapeptide hormone originally isolated from the mesophyll culture of Asparagus.PSK is a cleavage product of a 80-amino acid precursor protein.Posttranslational addition of two sulfate moieties at the tyrosine residues in the Golgi by tyrosylsulfotransferase(TPST)is required for its physical interaction with its cognate receptor the phytosulfokine receptor(PSKR) resulting in receptor activation followed by heterodimerization with the co-receptor somatic embryogenesis receptor-like kinases (SERKs)through the stabilization of the island domain of PSKR for interaction with the SERKs (Matsubayashi et al. 2001;Yang et al. 2001; Matsubayashi, 2003; Fukuda et al. 2007; Matsubayashi et al. 2006; Wang et al. 2015). PSKR is a LRR-RK with an

extracellular leucine-rich repeat domain and a cytosolic kinase domain with guanylate cyclase activity (Matsubayashi, 2003; Kwezi et al. 2011).The bifurcation of the pathway and the downstream targets of the kinase and the second messenger cGMP in the signaling pathways are poorly defined. Ca2+ has recently been implicated in striking a balance between the kinase and guanylatecyclase activities of PSKR (Muleya et al. 2014).This hormone is primarily involved in vascular differentiation pollen tube elongation and cell proliferation (Sauter 2015).However, its roles in plant defense are only beginning to be understood.PSK orchestrates the growth-defense dichotomy by favouring growth over defense, as both *pskr1*and *tpst* mutants in Arabidopsis exhibit enhanced resistance to the hemibiotropic bacterial pathogen *Pst*(Igarashi et al. 2012).The biotrophic pathogens often induce changes at the level to efficiently colonize the host and they appear to co-opt phytosulfokine pathway by taking advantage of the capacity of the pathway for tissue redifferentiation and dedifferentiation(Rodiuc et al. 2015).However, the downstream components of PSK signaling which are responsible for the observed effects in relation to defense response are largely unknown. Of the PSK receptors PSKR1, but not PSKR2, was implicated in plant immunity. PSKR1 dampens denfense against the biotrophic bacteria, *Pseudomonas syringae* pv. *tomato* DC3000.,while it intensifies immunity to the necrotrophic fungus *Alternaria brassicicola*(Mosher et al. 2013).

CONCLUSION:

There are still other hormones like nitric oxide, strigolactones, polyamines, karrikins and systemin, whose roles have not been discussed here. More importantly, hormones often do not work in isolation, as there exist crosstalks between hormones that further shape up plant-microbe interactions. Often the roles of hormones differ depending on whether they are endogenously produced by the plant or manufactured by the pathogen, as in case of cytokinin (Choi et al 2011).Challenges for future research lie in understanding how complex signal interactions are functional in ecological context where a single plant may be simultaneously subjected to various types of pathogen like the necrotrophs, the biotrophs, or even the beneficial microbes in a range of environmental conditions(humidity, temperature, photoperiod etc) with the host being left to choose between the various options (Corne et al 2012). The recent finding of SA receptors, NPR3 and NPR4, should be read as a significant step forward (Fu et al. 2012; Wu et al 2012). If NPR1 degradation is a prerequisite for programmed cell death (PCD) as is the situation at the site of attempted infection by an avirulent pathogen, unlike in the systemic tissue, then how does the healthy uninfected plants where a likely scenario is the presence of a basal level of or the absence of SA that allows for NPR4-NPR1 interaction followed by NPR1 degradation, prevent the needless PCD? (Attaran & He, 2012; Fu et al., 2012). Moreover, how does one reconcile the facts that in contrast to *npr1* mutant, *npr3npr4* double mutant shows enhanced resistance and, NPR3 and NPR4 are behind NPR1 turnover which is required for SAR establishment and the optimum induction of defense genes (Spoel et al 2009; Attaran & He, 2012; Fu et al. 2012)? Further study should address this ambivalence. The molecular connectivity between the so-called

executioners of PCD such as the plant metacaspases and the SA mediated PCD is lacking, at present. Now efforts should be also directed towards obtaining a high-resolution structure of the SA receptors (Attaran & He, 2012). How the non-coding RNAs influence resistance in the context of hormonal regulation is far from being known. It would be interesting to see if there is any significant variation in the epigenomic landscape of the somatic versus the germline cells and at the single cell versus tissue level in terms of DNA methylation status, histone modification patterns and noncoding RNA expression profile, up to a number of plant progenies after their successive parental progenies(the whole plant or only the root part) were grown in totally aseptic conditions for a few generations and their relative response upon inoculation with pathogens. Little is known about how the same hormone might influence the outcome of a disease differently by sometimes promoting disease while, at others, conferring resistance. Future work should make clear whether and how transgenerational epigenetic memory of resistant trait may be governed by hormones. Even though the environment plays a significant role in disease development, our understanding of environmental epigenetics of plant disease is rudimentary. Furthermore, the ultimate goal of the plants is to ward off the pathogens and staying immune. However, the detailed molecular mechanisms in relation to the pathogen targets and the relative contributions to immunity of some of the widely cited markers such as the antimicrobial proteins PR1 and PDF1.2 and the antimicrobial metabolites like the phytoalexins that are induced by the hormones and pathogens alike, are, at best, superficial, and bring up the question as to whether they are necessary and sufficient to prevent the disease, and call for further investigation. With the missing links in the hormone signaling pathways in the plant being known and the pathogen strategies discovered, our notion of plant-pathogen relationships will likely undergo a sea change and we would be in a position to manage biotic stress in plant.

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