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Chapter 4

ABIOTIC STRESS RESPONSE IN PLANTS: INTEGRATIVE GENETIC PATHWAYS AND OVERLAPPING REACTIONS BETWEEN ABIOTIC AND BIOTIC STRESS RESPONSES

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ABSTRACT

In all ecosystems, plants continuously face environmental stress and consequently are forced to respond with defensive and adaptive strategies. These responses require the activation of several signaling pathways that induce expression of specific genes. An effective response requires that the biological system have the genetic background to support the necessary molecular players that permit the assembly of essential integrative genetic pathways. In the most complicated scenario, plants must contend against more than one abiotic stress, pest, or pathogen at the same time, forcing an integral and complete defense response to adjust plant physiology. Certain molecular players act as hubs or master regulators to integrate signals from

different regulatory pathways activated by two or more types of abiotic stress or forms of biotic stress. Some defense and adaptive genes are transcriptionally regulated not only by a specific abiotic stress but also by biotic stress, exposing the existence of overlapping pathways. The genes shared between these networks appear to allow plants to prioritize their responses and ensure their survival by using their resources efficiently. The well-documented shared or convergence points in the response to abiotic and biotic stress in plants exhibit a superimposed complexity, as exemplified by induction of certain defense genes by biotic stress in presence only of a specific environmental condition (temperature and humidity). Interestingly, accumulating data strongly support the hypothesis that the convergence points between abiotic and biotic stress pathways also modulate the post-embryonic developmental program which is one of the most conspicuous adaptive strategies to cope with environmental stress. Pathogen attack or abiotic stresses such as nutrient scarcity alter cell division and cell differentiation processes, and consequently the plant architecture is modified. Plant growth regulators, such as auxins, cytokinins, ethylene, and jamonic acid, as well as reactive oxygen species, play crucial roles in the early steps of the convergence between these multiple stress signals. The advancement in powerful molecular tools, including transcriptome and proteome analysis, whole-genome sequencing, and bioinformatic studies are enabling disection of networks in abiotic and biotic signaling cascades and identification of the overlapping reactions and key factors that fulfill very important roles as integrative signals in plants.

I. INTRODUCTION

Different forms of abiotic and biotic stress invariably limit the production of crops in the approximately 1.5 billion hectares of arable land in the world (UNESCO, 2009), a problem that is far from solved and that will be exacerbated constraining plant growth and productivity. Problems of lower yields due to drought are serious; irrigation will not be a long-term viable solution since water grows scarcer and large-scale desalinization is not yet viable. High and low temperatures, acid soils, and soils with high content of metal ions reduce productivity in many cropping areas. Solutions to these problems could be diverse, but a better understanding of the physiology and genetic of tolerance in plants to stress will be essential as we attempt to mitigate these limitations and to increase crops yields. Maximizing yields by breeding for resistance, or at least tolerance, to abiotic and biotic stress is of great importance. Increases in crop yields in developing countries are of high priority since these areas exhibit the highest levels of increasing population and consequently increasing demands on

the food supply. Furthermore, the effects of climate change will be of major impact on lower latitudes that are occupied primarily by developing countries.

In the past two decades, great advances in plant molecular biology have permitted new discoveries about phenotype plasticity, defined as the capacity of a given plant genotype to produce different phenotypes in different environmental conditions (Sonia, 2010). Several genes that determine phenotypes of abiotic and biotic stress tolerance or resistance and involved signaling pathways have been identified. The study of gene function has been advanced firstly by whole genome sequences and bioinformatics approaches. At the same time, molecular biology techniques have been used to identify points of convergence where the signaling due to various stresses collides to activate specific response reactions. Formidable progress in the area of defense mechanisms to environmental stress in plants has been achieved, and the task now is to dissect the collections of players in the signaling pathways that transduce the messages from two or more cascades and identify the interconnections that exist among signaling routes. It is well known that when two or more types of stressing factors (biotic or abiotic; for instance, pathogen attack and heat stress or high relative humidity) coincide, the defense reaction to one stress impairs the reaction to the other. The knowledge of how biotic and abiotic environmental cues affect the final size and architecture of plants are progressing. We are beginning to understand in an integral way the biochemical, molecular, cellular, and physiological mechanisms involved in individual phenotypic plasticity. More effort is needed to draw the complete networks assembled in the intricate abiotic or biotic stress signaling responsible for control of the molecular, cellular, and physiological mechanisms of acclimation or adaptation.

II. How Plants Integrate Regulatory Genetic Pathways

During 450 millions of years of their existence on the earth, the environment has imposed changing and stressing conditions on land plants. These conditions have forced the evolution of elaborate molecular systems that respond to dynamic situations with physiological changes that have allowed plants to adapt efficiently to life in the varied climates of the earth. These elaborate molecular systems have enabled the survival and adaptation in each plant species that reign over planet surface. In biological systems, it is well known that evolution has led to integrated and well engaged signaling pathways, that enable the organisms to respond to

stressing conditions with the lesser waste of energy. An early event in plants after perception of environmental stresses is the activation of signaling cascades that in turn leads to the reprogramming of the profile of expressed genes including stress-responsive genes. In biotic stress, plant innate immunity is activated after perception of pathogen-associated molecular patterns (PAMPs) in charge of pattern recognition receptors (PRRs) or upon resistance (R) protein-mediated recognition of pathogen race-specific effector molecules (Figure 1). R proteins have been identified in many pathosystems, but only several PRRs have been described. In Arabidopsis several members of leucine-rich repeat with protein kinase activity have been found as PAMP receptors, which suggest members of this family function as pattern recognition receptors, and that heterologous expression of PAMP recognition systems is functional (Lacombe *et al*., 2010).

A number of molecular players fulfill the relevant function of integrating signals from signaling pathways activated in responses to different types of biotic and abiotic stresses. A good example is Med25, a subunit of the *Arabidopsis thaliana* Mediator complex. Mediator is a phylogenetically conserved multiprotein complex of 20 to 30 subunits (depending of the organism) organized into three linked modules (the head, middle, and tail) and a detachable kinase module. The tail module is thought to interact primarily with DNA-bound transcription factors, while the head and middle modules bind to the C-terminal domain of RNA polymerase II (Kidd *et al*., 2009). In this way Mediator promotes the assembly and activation of transcription complexes on core promoters, interacts with RNA Pol II in the initiation of transcription, and serves as a primary conduit of regulatory information from enhancers to promoters (Kuras *et al*., 2003). Mediator is thought to be an integrator of regulatory signals that converge on promoters of stress-responsive genes, in this way, several subunits have been found functionally necessary in the transcription activated in response to diverse stress-specific signaling pathways *(*Kim *et al*., 2006). Consistent with this, the Med25 subunit is a common target of three transcription factors involved in different pathways activated in response to salt, drought, and heat stress and also has a function in signaling that regulates flowering time in response to light conditions (Elfving *et al.*, 2011). That means that in these signaling cascades, Med25 (as part of Mediator) is the last signal receptor-transducer, just after these three transcription factors that have been found involved in stress response pathways, and just before the action of RNA Pol II (Figure 1). Med25 has a conserved activator-interacting domain (ACID) located in the 551-680 amino acid segment through which the molecule interacts with DNA-bound transcription regulatory factors. Transcription factors DREB2A, ZFHD1, and a MYB-like protein have been identified as Med25 interactors. DREB2A interacts with cis-

acting dehydration-responsive element/C-repeat (DRE/CRT) involved in cold and drought stress–responsive gene expression and, in a constitutively active form, enhances drought tolerance (Sakuma *et al*., 2006). The expression of ZFHD1 is induced by drought, high salinity and abscisic acid, and *Arabidopsis* plants overexpressing ZFHD1 have higher drought tolerance than wild-type plants (Tran *et al*., 2007). MYB-like protein activates expression of transcripts specifically expressed in plants subjected to a combination of drought and heat stress (Rizhsky *et al*., 2004). Furthermore, in *Arabidopsis*, the *PHYTOCHROME AND FLOWERING TIME1* (*PFT1*) gene, which encodes the MEDIATOR25 (Med25) subunit of Mediator, is required for jasmonate-dependent defense gene expression and resistance to leaf-infecting necrotrophic fungal pathogens (Kidd *et al*., 2009) (Figure 1, biotic stress).

Another example of a molecular player that integrates signals is the Target of Rapamycin (TOR), a Ser/Thr kinase conserved in fungi, insects, mammals, and photosynthetic eukaryotes. Rapamycin is an anti-proliferative drug produced by *Streptomyces hygroscopicus* (Schmelzle and Hall, 2000) originally described as an antifungal agent (Vezina *et al*., 1975). Rapamycin first binds to FK506-binding protein (FKBP12) and this complex inhibits the target TOR Ser/Thr kinase. In contrast to its activity in other eukaryotes, Rapamycin does not seem to affect TOR function in plants (Mahfouz *et al.*, 2006), although in the unicellular green alga *Chlamydomonas reinhardtii* TOR and FKBP12 homologs have been identified and characterized, and *Chlamydomonas* cells are sensitive to Rapamycin (Crespo *et al.*, 2005). TOR has a phosphatidylinositol 3-kinase domain in its C-terminal region, adjacent to the FKBP12-rapamycin-binding (FRB) domain through which it establishes interaction with the FKBP12-rapamycin complex. At the N-terminus, TOR has two blocks of HEAT motifs; these motifs are involved in protein-protein interactions (Andrade, 1995) Through the HEAT motifs, *Arabidopsis* TOR interacts with RAPTOR1 (a TOR regulatory protein), and RAPTOR1 regulates the activity of S6 kinase (S6K) in response to osmotic stress. S6K in turn phosphorylates ribosomal protein S6 (RPS6), through which regulates translation (Mahfouz *et al.*, 2006). TOR inactivation leads to a nutrient-starvation response, suggesting that TOR is involved in the reaction to nutrient deficiency (Barbet *et al*., 1996). In *Arabidopsis*, AtFKBP12 interacts with AtFIP37, a protein involved in embryogenesis and endosperm development, placing AtFKBP12 in regulation of the cell cycle and in developmental processes (Vespa *et al*., 2004). In yeasts and mammals it has been found that TOR is a central controller of cell growth (Schmelzle and Hall, 2000). In plants, TOR links embryonic development, cell growth, yield, stress resistance, and mRNA translation, integrating energy levels, nutrient availability, and stress information

(Abraham, 2005; Mahfouz *et al*., 2006). In summary, TOR acts as a hub that optimizes cellular resources for growth, playing a central regulatory role in stress and growth signaling pathways. Med25 and TOR are two molecules that exemplify how plants have evolved to respond efficiently by integrating several pathways through key master regulators (Figure 1).

Signal integration from different stress signaling pathways is also mediated more simply than by Med25 and TOR through the protein-binding elements in the promoters of some defense genes. It is known for example that abiotic and biotic stresses such as salinity, drought, abscisic acid (ABA), and fungal inoculation induce similar patterns of expression of the members of the *14.3.3* gene family (*GF14b* and *GF14c*). The 14-3-3 proteins participate in cellular regulatory pathways as adapters, chaperones, activators, or repressors and execute important steps in signal transduction and metabolism (Chen *et al*., 2006). These *GF14* genes share the characteristic of *cis*-elements in their promoter regions that are responsive to abiotic stress and pathogen attack (Figure 1). The *14-3-3s* family genes are also subject to regulation by certain transcript factors (Chen *et al*., 2006). In rice, the promoter regions of *OsGF14b*, *c*, *e* and *f* genes contain low-temperature response elements (LTRE), whereas *OsGF14b*, *c*, *d* and *g* genes contain cooper response elements (CuRE), in addition to other elements related to abiotic stress such drought-responsive elements (DRE) and binding sites for MYB transcription factors widely distributed in promoter regions of *OsGF14s* (Yao *et al*., 2007). Likewise, in *Arabidopsis* at least five of the 29 *cytochrome P450* genes are induced by abiotic and biotic stress including the pathogens *Alternaria brassicicola* or *Alternaria alternata*, paraquat, rose bengal, UV stress (UV-C), heavy metal stress ($CuSO_4$), mechanical wounding, drought, high salinity, low temperature, or hormones (salicylic acid, jasmonic acid, ethylene, and ABA). Five of these *cytochrome P450* genes (*CYP81D11*, *CYP710A1*, *CYP81D8*, *Cyp71B6* and *CYP76C2*) are co-induced by metal stress ($CuSO_4$), paraquat, salinity, ABA, and pathogen inoculation. In these *cytochrome P450* genes as in the *14.3.3* gene family, *cis*-acting elements include W-box (DNA binding sites for WRKY transcription factors), P-box (a positive *cis*-acting regulator of pathogen defense), and MYB recognition sites (Narusaka *et al*., 2004). These data show clearly that plants have evolved diverse response elements to assemble special architectures in the regulatory regions of defense genes, which allow an integrated and efficient response against diverse environmental stressing constraints (Figure 1).

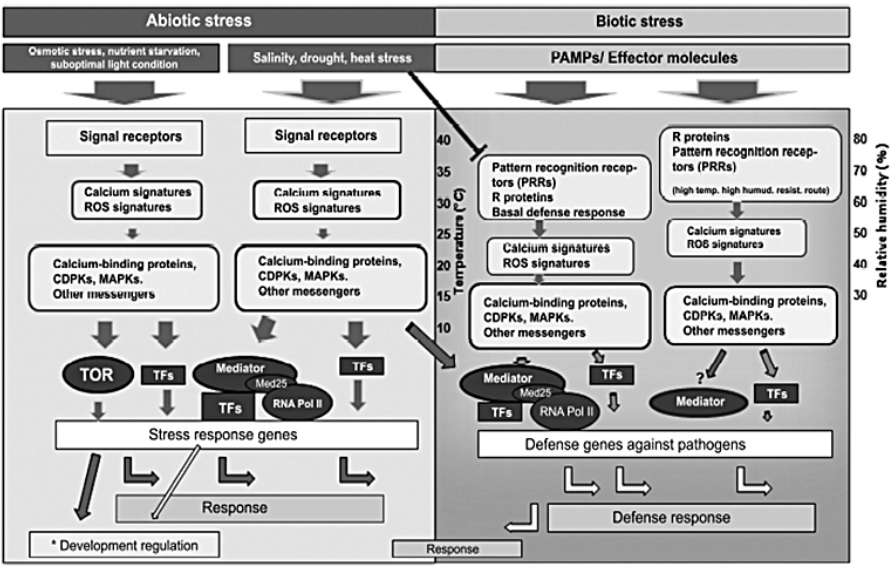

Figure 1. Signal cascade pathways in response to abiotic and biotic stress. In abiotic stress, besides classical transcription factors (TFs), TOR and Mediator (with Med25) as key regulator in the signal transduction to activate specific stress response genes. In biotic stress, cascade signaling through classical pattern recognition receptors (PRRs), R protein and Basal defense response, impaired by high temperature and high humidity, whereas several described R proteins act even in high temperature. Med25 also included in signal transduction to the expression of defense genes in response to pathogen. Furthermore, plant defense genes against pathogens, are induced in response to forms of abiotic stress. TOR and Med25 also involved in regulation of the development program.

III. Superimposed Complexity in the Defense Mechanisms to Biotic Stress in Specific Abiotic Stress Conditions

The defense reaction to pathogen in some pathosystems exhibits another level of complexity. Variable environmental conditions are common during the plant life cycle and, as could be expected, stressing conditions, in the climate for example, exert another stress pressure in the host in a pathosystem. The pressure of this extra stress is manifested in the plant cells and at the end in the molecular mechanisms that plant implements to respond to these spliced stressing factors. In this direction, several studies with mutants of *Arabidopsis thaliana* show a resistance phenotype that depends on environmental conditions, specifically

temperature and relative humidity; under low humidity and cold temperatures the resistance is enhanced, and in high relative humidity and high temperature the resistance phenotypes are suppressed (Yoshioka *et al*., 2001; Mosher *et al.*, 2010). Similarly, in *A. thaliana* and *Nicotiana benthamiana*/*Pseudomonas syringae* pathosystems, the basal resistance and (*R*) gene-mediated resistance are reduced at moderately elevated temperatures (Wang *et al*., 2009). Temperature sensitivity is thus present in defense mediated by different classes of R proteins, and also, often correlates with lesser hypersensitive response (Figure 1, biotic stress side), an observable fact not attributable to pathogen-secreted effectors. The negative effect of high temperature on the defense reaction is a common phenomenon reported in plant disease resistance against biotrophic and hemibiotrophic pathogens (Wang *et al*., 2009). Indeed, studies on plant-bacterial pathosystems indicate that the expression of virulence genes tends to increase below temperatures of optimal microorganism growth, and several bacterial effectors are secreted preferentially in a range of temperature between 18 and 22°C (Van Dijk *et al*., 1999).

That means that the advent of increasing environmental temperatures may impair (*R)* gene-mediated disease resistance and result in more extensive disease development, although there are examples of *R* genes that exhibit biological performance in the opposite direction with higher efficacy at high temperatures (Uauy *et al*., 2005; Fu *et al*., 2009; Webb *et al*., 2010) (Figure 1, biotic stress side). The detrimental temperature effect on *R*-gene-mediated and basal disease resistance indicates that genetic pathways activated in response to biotic stress are modulated by environmental factors to variable levels. The biological basis of high temperature could be explained by considering the impact of temperature on protein denaturation and aggregation and on nucleic acid denaturation; this in turn affects protein-protein or protein-nucleic acid interactions along all steps of the cascade pathway, for example in the avirulence product-R protein recognition and in the transcription factor-promoter sequence interaction. Thus, higher temperatures result in lower affinity between interacting factors, less specific recognition of pathogen molecules, and less optimal interactions between interacting players downstream in the signaling cascade, including the transcription factor and the response element in the respective gene, and finally non-optimal *R* gene expression.

In an environment with periods of high temperature, plants besides to cope with pathogen challenge, must respond with a physiology change to adapt and survive to the stressing condition. Plants could respond with a process of acclimation termed thermotolerance; in this process the changes at molecular level imply new profiles of expressed genes with emphasis on stress-related proteins. A well-characterized response to heat stress is the expression of heat shock proteins

(HSPs) that work as molecular chaperones assisting in folding, ATP-dependent refolding, intracellular distribution, assembly and degradation of proteins, and stabilizing partially unfolded states for thermoprotection (Lee and Vierling, 2000; Wegele *et al*., 2004). A characteristic effect of high temperature is damage to cellular structures and membrane dysfunction. Heat stress affects cell physiology by increasing the fluidity of membrane lipids, a structure whose interior lipid bilayer is normally highly fluid. In fact, normal cell function requires membrane lipid bilayers that are largely fluid, a common state at physiological temperatures. In accordance with this, cytoplasmic membrane function depends critically on the physical state of lipid bilayer, making it susceptible to changes in environmental temperature (Mansilla *et al*., 2004). The cell membrane is a structure for anchorage of membrane proteins such as signal receptors, ion-channels, and the machinery of translocation. Furthermore, high temperature results in high transpiration and this in turn may increase the number of particles dissolved in the cell water content (i.e., an increase in solute potential, ψ_s) and decrease the water potential (ψ_w). Peripheral proteins located on the membrane surface commonly are water-soluble with mostly hydrophilic surfaces. Conditions that disrupt ionic and hydrogen bond interactions would affect the biological function of R proteins located in the cytoplasmic membrane or in the cytosol, restraining the activation of the defense response at the first step in the signal cascade.

IV. Modulating Program of Post-Embryonic Development by Biotic and Abiotic Cues

All land-dwelling plants are attached to the soil and they cannot avoid the unfavorable conditions prevailing in its surroundings, as a consequence, they always are exposed to multivariate environmental cues. Plants evolved to perceive and integrate these biotic and/or abiotic cues and adjust their growth and development according to multivariate inputs. This capacity of plant genotype for producing different phenotypes under different environmental conditions, called phenotypic plasticity, allows plants to change dramatically their final body appearance to cope with environmental heterogeneity (Valladares *et al*., 2007; Capron *et al*., 2009; Sonia, 2010). This capacity is maintained even in mature plants in part due to their typical apical growth.

As part of their sessile lifestyle, plants show a particular kind of growth and development. Their mature cells are enclosed in a polysaccharide rigid matrix which maintains them attached to their neighbors. Unlike metazoans, plant

development is given by the progressive addition of new cells through the activity of local populations of totipotent cells termed meristems (Meyerowitz, 2002; Nakajima and Benfey, 2002; Taiz and Zeiger, 2010). The plant development is guided by a genetically controlled program and it can be divided in two main stages: a) embryonic development (ED) and b) post-embryonic development (PED). During the ED, through a suite of highly regulated and reproducible stages, the fertilized egg cell rises into an embryo. In the embryo, the primary meristems, body axes and major tissue layers are established (Jurgens, 2001; Nakajima and Benfey, 2002; Willemsen and Scheres, 2004; Capron *et al*., 2009). Almost all the body of the mature plant is generated during the PED, which begins during germination, as soon as the mitotic activity of meristems commences. Both the primary shoot meristem (SM) and primary root meristem (RM) occupy opposite ends of the main body axis. The first, located in the upper part, gives rise to new organs such as leaves, stems, secondary meristems, flowers and fruits. The second, at the bottom part, originates the root system (Willemsen and Scheres, 2004; Vernoux *et al*., 2010; Teotia and Lamb, 2011). The genetic program of PED regulates: i) primary meristems activity, ii) *de novo* formation of secondary meristems and organs, and iii) cell elongation, determining the final plant size and shape (Sánchez-Calderón *et al*., 2005; Taiz and Zeiger, 2010). Due to the high plasticity of the program of PED, the size and shape of plant can change according to environmental cues.

In soil, biotic and abiotic components are distributed heterogeneously, and there, supplies of nutrients and water are very limited, localized, and variable. Also, a broad range of chemical and physical processes occurs due to intrinsic soil characteristics and the action of biotic factors (Lynch, 1995; McCully, 1995). As soon as the primary root emerges from the seed, it has to grow through these hostile media. As growth goes on, *de novo* lateral roots are formed from the primary root. Primary and lateral roots form the root system (RS), which has an spatial configuration called root architecture (Lynch, 1995). According to RS morphology and architecture, the root-soil interaction area, called rhizosphere, can increase exponentially. Is in this area where the intricate and multivariate interactions among plants, chemical and physical soil components and macro and microorganisms take part. This complexity of biotic and abiotic cues must to be sensed and integrated by plants in order to adjust their program of PED. For example, as nutrients are distributed in a patching heterogeneous pattern, plants change their PED during their life in order to find the nutrient-rich regions, and in fact they develop more total root biomass in richer regions. Moreover, plants selectively destine more resources to those roots developing in those regions with increasing nutrient level availability, despite having other roots growing in

already richer regions (Hodge, 2004; Shemesh *et al*., 2010). Nutrients such as nitrogen (N), phosphorus (P), potassium (K) and sulfur (S) have been reported to modulate the program of PED (Zhang and Forde, 1998; Forde and Lorenzo, 2001; Williamson *et al*., 2001; Kutz *et al*., 2002; Lopez-Bucio *et al*., 2002; Ashley *et al*., 2006).

When growing under limiting P conditions, plants of *Arabidopsis thaliana* show dramatic changes in root architecture such as reduction in primary root length, increased formation of lateral roots and greater formation of root hairs. P deficiency induces a change of program of PED from determinate to indeterminate. Typically, on indeterminate PED, the newly formed root cells are added by the mitotic activity of primary meristem. These cells then get away from the meristem and increase their length, and the elongation process ends when the cells start to differentiate. When plants are P starved, cell division in the primary root meristems gradually reduces and the cells start to prematurely differentiate until total inhibition of cell elongation and loss of meristematic activity occur (meristem exhaustion). At the end, root tips change their physiological characteristics and the exhausted meristem becomes a structure which takes part in P uptake (Williamson *et al*., 2001; Lopez-Bucio *et al*., 2002; Sánchez-Calderón *et al*., 2005). In this process, root tips locally detect P deficiency, this response being mediated by at least LPR multicopper oxidase genes (Sánchez-Calderón *et al*., 2005; Svistoonoff *et al*., 2007). Recently, iron (Fe) has been reported to play a role as well in the control of these PED reprogramming (Ward *et al*., 2008). This change of root architecture is due to the fact that, in both meristematic and elongation areas, the content of reactive oxygen species (ROS) is reduced as long as the determined PED goes on. The *low phosphorus insensitive 4* (*lpi4*) mutant does not show the typical P deficiency growth phenotype, neither the ROS reduction in root tips. Interestingly, the addition of jasmonate (JA) to low P availability medium rescues the wild type (WT) growth phenotype, suggesting that this phytohormone, commonly related to biotic stress, is taking part over developmental root system changes in response to nutritional stress (Chacón-López *et al*., 2011), and could be a link integrating both abiotic and biotic stress response signals. However, addition of JA in media also inhibits the primary root growth in WT plans independently of the media P content (Berger *et al*., 1996). Crosstalk can be inferred by analyzing if a specific mutant shares components or check-points regarding responses to abiotic and biotic stresses. For example, the *coi1* (coronatine insensitive1), *jar1* (jasmonate resistant1) and *jin1* (jasmonate insensitive1) mutants are affected in JA signaling (Feys *et al*., 1994; Berger *et al*., 1996; Staswick *et al*., 2002). *COI1*encodes a F-box protein, an integral component of multi-protein complexes implicated in ubiquitination. JAR1, a JA amino acid

synthetase, is required to activate JA for signaling. *JIN1*encodes a transcription factor of the helix-loop-helix type which transcriptionally regulates the expression of JA-responsive genes (Xie *et al*., 1998; Lorenzo *et al*., 2004; Staswick and Tiryaki, 2004). Genetic damage in these *loci* modifies the sensitivity of these mutants to bacterial and fungal pathogens and insect attack (Feys *et al*., 1994; Anderson *et al*., 2004; Lorenzo *et al*., 2004; Dombrecht *et al*., 2007); besides, it reduces the root development sensitivity to JA, clearly indicating that *COI1, JAR1* and *JIN1* are necessary for root development and defense responses. Future work using *coi1, jar1* and *jin1* is necessary to confirm the crosstalk among changes in root development specifically induced by P starvation and plant defense responses. Finally, in the shoot, the shade avoidance syndrome and some specific defense responses are regulated by integrated jasmonate and light signals (Kazan and Manners, 2011).

CONCLUSION

Until recently, it was thought that plant responses to biotic or abiotic stresses involved a unidirectional signaling. However, in the past decade our knowledge about the sophisticated signaling pathways that have evolved to deal with environmental changes has increased dramatically. We now understand how plants sense and integrate the multivariate biotic and abiotic stressors allowing intricate crosstalk among different signaling pathways in the response to the many environmental stresses. In this context, the proteins Med25 and TOR are key molecular players in this integration of multiple stressor signals. The identification of these control points shared in signaling networks, and work toward an understanding of how they are regulated and how they in turn regulate downstream responses is of paramount importance, as we seek to determine how plants modulate their development, physiology and metabolism in a wide spectrum of phenotypic responses. We expect that in the near future, this research will enable the development of new generations of tolerant crop varieties. These new strains should have better performance under stressful environmental constraints, leading to higher yields and productivity, necessary as we seek to respond to the food demands of the increasing world population.

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