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REVIEW ARTICLE

Biotechnological implications from abscisic acid (ABA) roles in cold stress and leaf senescence as an important signal for improving plant sustainable survival under abiotic-stressed conditions

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Abstract

In the past few years, the signal transduction of the plant hormone abscisic acid (ABA) has been studied extensively and has revealed an unanticipated complex. ABA, characterized as an intracellular messenger, has been proven to act a critical function at the heart of a signaling network operation. It has been found that ABA plays an important role in improving plant tolerance to cold, as well as triggering leaf senescence for years. In addition, there have been many reports suggesting that the signaling pathways for leaf senescence and plant defense responses may overlap. Therefore, the objective was to review what is known about the involvement of ABA signaling in plant responses to cold stress and regulation of leaf senescence. An overview about how ABA is integrated into sugars and reactive oxygen species signaling pathways, to regulate plant cold tolerance and leaf senescence, is provided. These roles can provide important implications for biotechnologically improving plant cold tolerance.

Keywords: *ABA; abiotic stress; signal transduction; leaf senescence; gene regulatory network; signaling pathways; biotechnology*

Introduction

Successful adaptation of a plant species is dependent upon the programming of critical growth stages so that the plant can capitalize on favorable weather periods during the growing season (Dominique and Andrew, 2010). Plants have evolved a variety of adaptive mechanisms that allow them to optimize growth and development while coping with environmental stresses. These mechanisms include seed and bud dormancy, photoperiod sensitivity, and low-temperature response and others (Fowler *et al.*, 1999; Caius, 2010; Clint and Malcolm, 2007). Seed dormancy delays germination until after the embryo has gone through an after-ripening period. The overwinter survival of buds of many temperate zone trees and shrubs is dependent on a dormancy stage that starts in the late summer or early fall and ends after exposure to an extended period of cold or increasing day length in the spring. In addition to trees, many other dicots and grasses have a photoperiod response that can advance or delay flowering. Vernalization is a requirement for growth at low temperatures before a plant will flower. Most winter annual and biennial plants have a vernalization requirement. Low-temperature acclimation is an ability of plants to cold acclimate when exposed to gradually decreasing temperatures below a specific threshold. This is the most common mechanism that plants have evolved for adapting to low-temperature stress and examples of plants with the capacity to cold harden can be found in most species (Fowler *et al.*, 1999; Nilson and Assmann, 2007; von Caemmerer and Baker, 2007; David and Jacqueline, 2010).

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Cold acclimation, a process in plants that requires days to weeks for full development, is induced primarily by temperatures below 10°C, and by a short photoperiod in certain species of trees and grasses (Gusta *et al.*, 2005). During the above process, abscisic acid (ABA) plays an important role. The important aspect of ABA on plants' responses promoting tolerance to cold stress as well as the induction of leaf senescence has been studied for years. In addition, there have been reports indicating the regulation of leaf senescence by cold stimulus (Chen *et al.*, 2002; Cutler, 2005). All above facts trigger our interest to clarify the actual role of ABA signaling pathway in the regulatory network of cold and leaf senescence signaling pathways. Therefore in this review, we try to elucidate the possible mechanisms by which the ABA signaling pathway is involved in the cold and senescence signaling pathways through analyzing the genes whose products are components of the biochemical and regulatory pathways of cold and leaf senescence. We also try to provide an overview about the participation of ABA in integrating other signaling such as sugars, reactive oxygen species (ROS) as well as ethylene (ET) to regulate cold tolerance and leaf senescence. Due to the fact that whether ABA takes participation in the leaf senescence response of plants to cold stress still remains unknown, we hope this review can give a new insight into the possible function of ABA in mediating the overlap between the cold and senescence signaling pathways.

Abscisc acid acts as desired signals

The plant hormone ABA is an important regulator of growth and development, affecting diverse processes such as leaf senescence, seed dormancy and germination of seeds, cell division and elongation. It is also found to be involved in the responses enhancing tolerance to abiotic stresses including freezing and drought (Finkelstein *et al.*, 2002; Davies and Jones, 1991; Giraudat *et al.*, 1994; Chaves *et al.,* 2009; Ashraf, 2010).

Like many other plant hormones such as auxin (Indole Acetic Acid), cytokinin (a purine derivative), ABA is small soluble organic molecule. It is synthesized in almost all cells, but its transport from roots to shoots, and the recirculation of ABA in both xylem and phloem are important aspects of its physiological role (Sauter *et al.*, 2001). In addition, this small molecule can move freely in the plants, which makes it a desired candidate as a secondary signal.

ABA functions through ABA triggered signaling to proteins and genes. Nowadays, research in ABA signaling has been focused largely on gene expression. A wide variety of genes are ABA regulated at the transcriptional level, including genes involved in stress defense, and senescence responses, secondary metabolism and cell wall biosynthesis, Amino acid metabolism, carbohydrate metabolism, fatty acid and lipid metabolism and transport, transcript regulation, and signal transduction (Li *et al.*, 2002). For incidence, transcriptome analyses have shown that ABA dramatically alters genomic expression in *Arabidopsis thaliana* (Hoth *et al.*, 2002; Seki *et al.*, 2002). More than 1, 300 ABA-regulated genes were identified by random massive sequencing of *Arabidopsis* transcripts, of which half showed decreased expression in response to ABA (Hoth *et al.*, 2002).

Mediators of ABA-triggered gene expression include the transcriptional regulator VIVIPAROUS1 (VP1)/ABI3, as well as bZIP and APELATA2 (AP2)-type transcription factors (TFs) ((Finkelstein *et al.*, 2002). Through characterizing and comparing the promoter regions of these ABA-induced genes, it has been found there existed a major cis-acting element (PyACGTGGC) named the ABA responsive element (ABRE) in ABA-responsive gene expression (Yamaguchi-Shinozaki and Shinozaki, 2005; Guiltinan *et al.*, 1990). The bZIP TFs interact as dimers with ABREs,, which are ACGT-containing 'G-boxes' in promoter elements (Hattori *et al.*, 2002). Moreover, optimal ABA responsiveness usually requires a second cis element or 'coupling element' (CE), which is similar to an ABRE or a dehydration-responsive element (DRE). ABRE-targeting bZIP TFs and DRE-targeting AP2 TFs interfere with ABA-controlled gene expression (Kizis and Pages, 2002; Narusaka *et al.*, 2003).

Plant cold tolerance and ABA

Plant cold tolerance

Low temperature is one of the major environmental stresses that many plants have to deal with during their life cycle. Cold stress causes several dysfunctions at the cellular level that include damage to membranes, generation of ROS, protein denaturation and accumulation of toxic products, etc (Bowers, 1994). In nature, plants may exhibit cold tolerance due to gradual exposure to low non-freezing temperatures, the process known as cold acclimation (Thomashow, 1999).

Cold acclimation is associated with multiple mechanisms that include alterations in (i) gene expression (ii) membrane composition and cryobehaviour (Orvar *et al.*, 2000) (iii) accumulation of cryoptotectants such as proline, sugars and antioxidants (Kang and Saltveit, 2001), and (iv) elevation of ABA (Lang *et al.*, 1994).

Thanks to the advent of molecular biology, today some of the events of cold signal perception, transduction and cold acclimation are defined at the molecular level (Shinozaki and Yamaguchi-Shinozaki, 2000; Browse and Xin, 2001; Zhu, 2001).

Nowadays, many genes involved in cold response and tolerance have been discovered through transcriptome analysis, a powerful tool which can be achieved using microarray technology. The functions of these gene products are classified into two major categories, one involved in stress tolerance, such as chaperones, late embryogenesis abundant proteins, enzymes for osmolyte biosynthesis and detoxification enzymes and the other in gene expression and signal transduction in abiotic stress response, such as protein kinases, TFs, and enzymes in phospholipids metabolism (Shinozaki and Yamaguchi-Shinozaki, 2000). In *Arabidopsis*, these genes are called rd (responsive to dehydration), erd (early responsive to dehydration), lti (low-temperature induced), kin (cold induced) and cor (cold-regulated). These genes are

also induced by dehydration (due to water deficit or high salt) and can be collectively called cold-responsive genes.

Although how plants improve cold tolerance has been investigated to a large extent, how plants sense cold is still poorly understood. A fall in temperature can reduce the fluidity of membranes, and this appears to be an effective direct sensor of cold in cyanobacteria. Another possibility is that, in light, photosynthetic cells may sense cold through an effect on photosystem II excitation pressure (Gray *et al.*, 1997; Huner *et al.*, 1998). Molecular studies have shown that cold acclimation in higher plants is complex involving multiple regulatory pathways (Fowler and Thomashow, 2002), furthermore while some of the signal transduction pathways interact, others are independent (Xin and Browse, 2000). Thus more than one sensor of cold is possible. Figure 1 has demonstrated the best known events which occurred in plants under low temperature.

ABA roles in increasing plant cold tolerance and its mechanisms

As an important signal molecule, ABA can mediate abiotic stress signal transduction and tolerance. It has been found that ABA accumulates in response to many abiotic stresses, such as drought and salt (Xiong and Zhu, 2003). Cold stress also increases endogenous ABA levels in plants but to a much lesser extent. In herbaceous plants, exogenous application of ABA to plants growing at normal growth temperatures has been found to induce cold tolerance (Nayyar *et al.*, 2004; Xing and Rajashekar, 2001). The transient increase in ABA during cold stress, along with the enhancement of freezing tolerance by applied ABA, indicate that ABA must be playing a critical role in cold acclimatization (Plieth *et al.*, 1999; Toldi *et al.*, 2009).

Aside from the herbaceous plants, it also has been confirmed that ABA-controlled processes are central to cold acclimation and development of freezing tolerance in woody plants. Compared to herbaceous species, cold acclimation capacity is much higher in temperate zone woody plants. Herbaceous species can get through cold acclimation just by exposure to low temperature (Thomashow, 1999), whereas, cold acclimation in woody plants is more complicated. First, short day-length initiates the cold acclimation; the complete winter hardiness, however, is achieved by subsequent low temperature. It has been observed that two main types of cold acclimation exist in woody plants: (a) the seasonal acclimation for over-wintering and (b) the acclimation for daily temperature fluctuations during the growth season. In the field, if the tree does not reach an adequate level of cold hardiness before the first of autumn, it will be damaged. Previous reports have revealed that in woody plants, low temperature can increase the endogenous ABA levels (Welling *et al.*, 1997; Baldwin *et al.*, 1997; Li *et al.*, 2002), and the cold stimulus can be substituted by exogenous ABA, resulting in an increase in freezing tolerance at normal growth temperatures (Li *et al.*, 2003a). On the other hand, ABA could be also related to bud dormancy in trees, which is critical for over-wintering to woody plants. Measurements of endogenous ABA in some woody plants have shown that a short photoperiod induced an increase in ABA levels, and then they remained at a constant higher level (Myking, 1997; Li *et al.*, 2003b). Furthermore, applied ABA can induce growth cessation and dormancy development under a short photoperiod (Li *et al.*, 2003a). As for woody plants, the ability to sense a short photoperiod to reach the growth cessation, as well as the onset of dormancy, is a key step for freezing tolerance in the winter. Therefore, the effect that ABA has on the enhancement of cold tolerance in woody plants seems not unlikely.

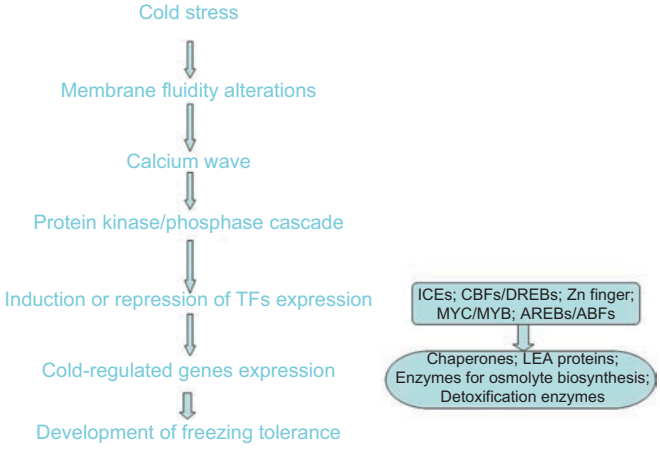

Figure 1. Proposed events occurring in plants under low temperature treatment.

In addition to the above evidences, various ABA mutants showing in *Arabidopsis* also give us insights into the critical role ABA takes in cold tolerance. For example, *Arabidopsis* mutants such as abi1, los5 (low expression osmotically responsive gene los5/aba3 (ABA deficient3) and los6/aba1 appear to be impaired in their ability of cold acclimation (Gilmour and Thomashow, 1991; Mantyla *et al.*, 1995; Xiong *et al.*, 2001). In the los5/aba3 and los6/aba1 mutants, Cor/Lea expression levels are reduced under LT stress (Xiong *et al.*, 2002).Although cold signal transduction pathways lack full understanding due to the complex regulatory network, along with the crosstalk with other signal pathways, the general mechanism of ABA-mediated development in cold tolerance has been established, particularly in herbaceous plants.

By determining cis-acting elements in the cold-responsive gene promoters, two major cis-acting elements–ABRE and CRT/DRE (C-repeat/DREs)–which function in ABA-dependent and ABA-independent gene expression, respectively, have been identified. In respect of diverse cis-acting elements, these can be activated by binding with distinguished transcriptional factors (TFs). By using yeast one-hybrid screening methods, cDNAs encoding the basic-domain leucine zipper (bZIP) TFs, referred to as ABRE-binding proteins (AREB) or ABRE-binding factors (ABFs), were isolated. The AREB/ABF proteins bind to ABRE and activate ABA-dependent gene expression (Uno et al., 2000; Choi, 2000; Chu *et al.*, 2005; Shao *et al.* 2005, 2007–2009). In terms of ABA-independent genes, C-repeat binding factors/dehydration-responsive element binding proteins (CBFs/DREBs) are considered as key components

in the transcriptional regulation of these genes (Thomashow, 2001; Gilmour *et al.*, 1998; Verslues *et al.*, 2006).

Since it has been evident CBF (DREB1) genes act as nodes of regulatory network in Arabidopsis response to cold stress, which are conserved in many plants, nowadays, more attention have been put on researching the CBF family. Up to now the CBF family, known either as CBF1, CBF2, and CBF3 or DREB1b, DREB1c, and DREB1a, has been well understood in *Arabidopsis* (Gilmour *et al.*, 1998; Kasuga *et al.*, 1999). The homologous components of the Arabidopsis CBF cold response pathway in many plants such as rice and strawberry (Dubouzet *et al.*, 2003; Owens *et al.*, 2002; Shao *et al.*, 2007) have also been found.

Although it had been thought that the CBFs regulated pathway was ABA-independent, so far, it has been proven that there exists CBF4, which has been demonstrated to be inducible by ABA and drought, but not by cold. It could be involved in the activation of the CRT/DRE by ABA (Haake *et al.*, 2002). In addition, further studies show that the CBF1-3 transcript levels also increase in response to elevated ABA. All the above findings indicate that activation of the CRT may also occur via a novel ABA-inducible signaling pathway, using the normally cold-inducible CBFs. Thus, it would mean that cross-talk is occurring between what has been considered two separate pathways involving two distinct sets of transcriptional factor and that under certain circumstances the drought pathway is recruiting CBF (Knight *et al.*, 2004; Shao *et al.*, 2007–2009).

In addition to the AREB/ABF and CBF/DREB family, other transcriptional factors involved in cold transduction pathways including ICE (Inducer of CBF expression) and MYB/MYC (Gilmour *et al.*, 1998; Abe, 2003) (Figure 2) have been reported.

Overlap between ABA and sugar pathways in the regulation of cold tolerance

Similar to ABA, soluble sugars have been verified to play a critical role in cold acclimation on the basis of the fact they are most commonly detected in various species of land plants that have undergone seasonal cold acclimation, including angiosperms, gymnosperms, and lower land plants such as bryophytes (Sakai and Larcher, 1987; Rutten and Santarius, 1992; Chu *et al.*, 2005; Shao *et al.*, 2007, 2008).

Although the functions that the sugars provide in improving cold tolerance has been well deciphered, nowadays, more and more attention has been put on the important hormone-like functions the soluble sugars take as primary messengers in signal transduction. Meanwhile, it has been concluded that the sugar signal pathway is integrated with the ABA signal pathway.

Using a Relevance Vector Machine, a novel promoter classification method, Li *et al.* (2006) identified discriminatory features in the promoter sequences of genes and showed that genes can be upregulated by both glucose and ABA. These genes are involved in stress, defense, and senescence responses, secondary metabolism and cell wall biosynthesis, amino acid metabolism, carbohydrate metabolism, fatty acid and lipid metabolism and transport, transcript regulation, and signal transduction. More than 12% of the genes induced by both glucose and ABA are involved in stress responses, indicating overlapping regulation by glucose and ABA (Li *et al.*, 2006). In the case of cold stress, expression of key regulators of cold stress responses such as CBF3, COR15A, and RD29A were induced by both glucose and ABA. Constitutive expression of CBF3 in transgenic *Arabidopsis* plants induces expression of target COR (cold-regulated) genes to enhance freezing tolerance in nonacclimated plants (Gilmour *et al.*, 2000). Expression of COR15A and RD29A is regulated by CBF3, suggesting that both glucose and ABA may contribute to the regulation of cold stress tolerance. Moreover, there might be a correlation between sugars and ABA signaling pathways.

ABA, genes and leaf senescence

Leaf senescence is the final stage in leaf development, and a better understanding of senescence is important not only for

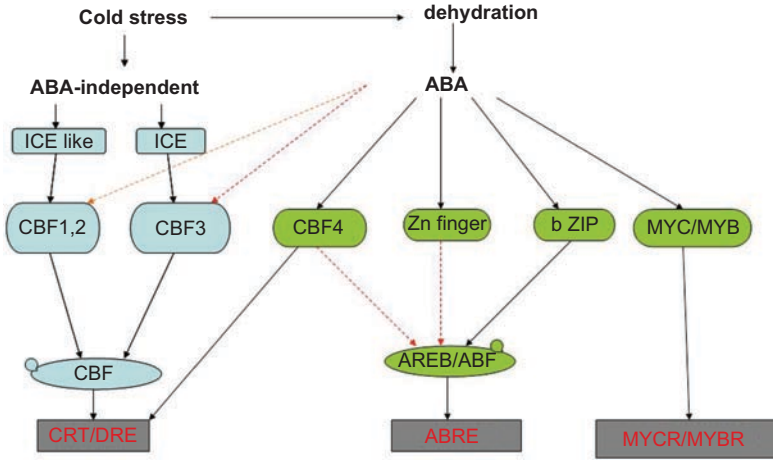

Figure 2. Known transcriptional regulatory network of the genes involved in cold stress response. ABA-independent transcriptional factors are colored blue, while ABA independent factors are colored green. Small circles indicate post-transcriptional modification, such as phosphorylation. Transcription factor binding sites are represented as rectangles at the bottom of the figure. Dotted lines indicate possible regulation.

purely scientific reasons, but also for practical purposes. Leaf senescence is not simply the aging-dependent passive death of a leaf, but is a tightly organized and controlled process during which cell components are degraded in a coordinated fashion. When nutrients have been relocated to other parts of the plant body, the cell finally dies (Gan and Amasino, 1997; Nooden *et al.*, 1997).

Conspicuous visual symptoms of leaf senescence are the loss of chlorophyll pigments (yellowing), desiccation, and eventual abscission. Cellular and molecular events contributing to these visual symptoms involve chloroplast disintegration, a decline in photosynthesis, and the loss of proteins and nucleic acids (Smart, 1994; Chandlee, 2001; Buchanan-Wollaston, 1997). Additional internal symptoms of senescence are a decreased ability to accumulate proteins and nucleic acids due to enhanced degradation and/or reduced synthesis (Smart, 1994; Buchanan-Wollaston, 1997). The catabolism of chlorophylls, lipids, proteins as well as nucleic acids allows large amounts of nutrients to be released and mobilized subsequently to growing leaves, developing seed, or storage tissues (Buchanan-Wollaston, 1997; Quirino *et al.*, 2000; Fang *et al.*, 2008).

Although senescence occurs in an age-dependent manner in many species (Nooden, 1988), its initiation and progression can be modulated by a variety of environmental factors such as temperature, mineral deficiency, and drought conditions, as well as by internal factors such as plant growth regulators (Grbic and Bleecker, 1995; Nam, 1997; Weaver *et al.*, 1997; Dai *et al.*, 1999). It has been proven that drought, darkness, leaf detachment, and the hormones ABA and ET induce leaf yellowing, whereas to different extents (Lim and Nam, 2005).

Genes involved in the leaf senescence

Like other developmental processes, leaf senescence is actively regulated by differential gene expression. In order to clarify the molecular mechanisms by which this dynamic process happens, numbers of senescence-associated genes (SAGs) have been identified (Lim and Nam, 2005; Chandlee, 2001). These SAGs encode diverse proteins including RNases, proteases, lipases, proteins involved in the mobilization of nutrients and minerals, transporters, TFs, proteins related to translation, and antioxidant enzymes. In contrast, senescence-downregulated genes mainly encode proteins involved in photosynthesis (Buchanan-Wollaston *et al.*, 2003; Gan, 2003). For instance, among the identified senescence-induced genes are genes encoding proteases, RNases, Gln synthetase, metallothioneins, protease regulators, 1-aminocyclopropane-1-carboxylate oxidase, lipases, glyoxylate cycle enzymes, catalase, adenosine triphosphate sulfurylase CytP450, aspartyl proteases, glyceraldehyde-3-phosphate dehydrogenase, pathogenesis-related proteins, hydrolases, phosphoenolpyruvate carboxykinase, endoxyloglucan transferase, components of the ubiquitin–proteasome pathway. Nonetheless, transcript levels for photosynthetic genes such as rbcS (small subunit of Rubisco) and cab (chlorophyll a/b-binding protein) decline (Bate *et al.*, 1991).

Since aside from natural senescence, there also are various types of leaf senescence which can be induced by abiotic as well as biotic stresses, more and more research focuses on comparing the genes regulated during the stress-induced leaf senescence with the genes involved in the natural leaf senescence. Based on the comparison between the gene-expression profile of artificially induced senescence in detached leaves and that of natural senescence in intact leaves, it has been evident that many, but not all of these genes, exhibit similar patterns of expression (Becker and Apel, 1993; Weaver *et al.*, 1997; 1998;). In addition, induction of certain defense-related genes has been reported to occur during leaf senescence (Quirino *et al.*, 1999). The involvement of genes associated with natural leaf senescence in ozone-induced leaf senescence (Miller *et al.*, 1999) has also been found.

ABA triggers leaf senescence or vice versa?

Due to all of the above physiological and biochemical investigations, it can be concluded that leaf senescence is actually regulated by a complex array of endogenous and environmental factors (Graaff *et al.*, 2006). Among these factors, ABA has been suggested to affect leaf senescence significantly. ABA has been considered a senescence promoter for a long time. Even though evidence for an *in vivo* role is rather poor compared with ET (Madhu *et al.*, 1999; Tadas *et al.*, 1999), there still appear to be large numbers of findings supporting the role ABA takes as a leaf senescence inducer. Foliar spraying with ABA has been shown to promote senescence in rice (Ray *et al.*, 1983) and maize (He and Jin, 1999).

Also, it has been verified that treatment with ABA can induce leaf yellowing, which is a good indicator of leaf senescence (Yang *et al.*, 2003; Fang *et al.*, 2008). On the other hand, an increase in endogenous ABA appears to coincide with senescence of leaves (Gepstein and Thimann, 1980; Yang *et al.*, 2002; Shao *et al.*, 2007–2009), which gives another demonstration that ABA functions in leaf senescence. In addition to this evidence, the existence of many ABA mutants supports the involvement of ABA in leaf senescence induction as well. It is known that Arabidopsis mutants, with deficiencies in ABA biosynthesis or signaling, exhibit altered or delayed senescence (Gan, 2003; Lim and Nam, 2005; Passioura, 2007; Galiba *et al.*, 2009).

Although many studies have shown that leaf senescence is a highly plastic trait, affected by a range of internal and external factors such as ABA, ET, dehydration, and darkness (Smart, 1994), the mechanisms by which these factors induce leaf senescence are not well elucidated. With the identification of SAGs, far more is known about the molecular mechanisms than before. One model is that each of these factors may induce a subset of SAGs. As for ABA, in Arabidopsis, it has been found that ABA can promote senescence and induce expression of several SAGs. Plus the fact that SAGs can be upregulated by ABA, the expression profiles of ABA-related genes alter in the process of leaf senescence as well. It has been demonstrated that

ABA-responsive genes express to a large extent and this is consistent with increasing ABA levels during leaf senescence and also indicates senescence-dependent alterations in ABA signaling. Nevertheless, what is complicated is that investigations also exist concluding that leaf senescence downregulates the ABA-response genes.

Since some ABA-response genes are up- and others down-regulated, it seems that the impact of ABA as a regulator during leaf senescence is not straightforward.

Interactions of ABA and ET in the regulation of leaf senescence

It has been well known for a long time that the plant hormone ET can influence leaf senescence as an accelerator. The importance of ET signaling during senescence is illuminated by a delayed senescence phenotype of ET-insensitive mutants such as ein2 in Arabidopsis (Oh *et al.*, 1997). Etr1-1, another Arabidopsis thaliana ET-insensitive mutant also exhibits a delayed leaf senescence phenotype, and this delay is accompanied by a corresponding delay in the expression of SAGs (Grbic and Bleecker, 1995).

Moreover, among those SAGs, some are found to encode enzymes involved in the EL biosynthesis such as ACC synthase and ACC oxidase. Interestingly, the expression of ACC synthase, the rate-limiting enzyme in ET biosynthesis results in a spike of ET release, which then triggers an increase in ABA synthesis and H_2O_2 production (Hansen and Grossmann, 2000; Grossmann *et al.*, 2001). Inhibitors of ACC synthase or of ACC oxidase, which catalyzes the conversion of ACC to ET, inhibit ABA production, indicating that ET induces ABA synthesis (Hansen and Grossmann, 2000). Thus, it is likely that the role of ABA in regulating leaf senescence is at least partly related to ET.

Interactions of ABA and sugars in the regulation of leaf senescence

During leaf senescence, the sugar content elevates considerably (Wingler *et al.*, 1998; Masclaux *et al.*, 2000; Quirino *et al.*, 2001; Stessman *et al.*, 2002) and senescence-like symptoms, such as leaf yellowing; can be triggered by sugars (Wingler *et al.*, 1998), which implicates the role of sugars in the induction of leaf senescence. Additional proof is given by work with mutants and transgenic plants. Tomato plants, overexpressing the sugar sensor hexokinase-1 from Arabidopsis, show accelerated senescence (Dai *et al.*, 1999), nevertheless, senescence is delayed in the Arabidopsis glucose-insensitive2 mutant lacking hexokinase-1 (Moore *et al.*, 2003).

What raises a question is that the majority of the sugar insensitive Arabidopsis mutants are either ABA insensitive (abi mutants) or ABA deficient (aba mutants), demonstrating that ABA plays an important role in sugar responses. However, until now, little was known about the interactions of sugar and ABA signaling in senescing plants, which might pose challenges as well as chances to clarify the crosstalk between the sugars and the ABA signaling pathways in the regulation of leaf senescence.

Interactions of ABA and ROS in the regulation of cold tolerance and leaf senescence

It is well established knowledge that utilization of molecular oxygen may be preceded by a series of single electron transfers, which generate ROS such as superoxide anion (O_2^-), hydrogen peroxide (H_2O_2), and other free radicals that react with, and thereby damage DNA, proteins, and lipids (Bowler *et al.*, 1992). Earlier studies have indicated that the production of ROS displays an elevation under drought and cold stimuli. (Bowler *et al.*, 1992; Fryer, 1992; Shao *et al.*, 2008). These ROS originated from plasma membranes and have been manifested to serve as a signal in plant cells to elicit various defense responses to cold stress (Prasad *et al.*, 1994), however, the mechanisms remain poorly understood. These days, increasing evidence has indicated that ABA can cause an increased generation of O_2 (Jiang and Zhang, 2001) and H_2O_2 (Guan *et al.*, 2000; Grennan, 2008), and thus induce the expression of various antioxidant genes. In this way, the ROS released under cold stress might be cleaned away and lead to the enhancement of cold tolerance. However, what is complicated is that H_2O_2 has been demonstrated to be a signal molecule as well. Along with the observed participation of H_2O_2 in ABA-induced activities of antioxidant enzyme (Gong *et al.*, 1998; Jiang and Zhang, 2002; Chaves *et al.*, 2009), it is difficult to say which signal is the main induced factor.

Regarding leaf senescence, ROS can initiate lipid peroxidation and lipid peroxidation has been considered to be an important mechanism of leaf senescence; thus the existence of ROS-induced leaf senescence can be concluded. On the other hand, the correlation between ABA signal and ROS signaling involved in the leaf senescence seems similar to the that participating in cold signaling pathway. Mariya *et al.* (2010) provided new evidence that ABA plays key roles in signal transducing from the changing environment to plants.

Conclusions and perspectives

There is no doubt that ABA plays a crucial role in promoting plant tolerance to cold, as well as inducing leaf senescence(Galiba *et al.* 2009). Through exploring the genes included in the regulatory pathways of cold tolerance, as well as leaf senescence, we obtain an overview about the molecular basis on which the ABA signaling pathway participates in the regulatory network of cold and leaf senescence pathways. Other signaling including sugars, ET and ROS are also implicated in the regulation of plant cold tolerance and leaf senescence, and the crosstalk between ABA and these signaling pathways has been established, although the relationship among them remains poorly characterized.

Recent studies have shown that the signaling pathways for leaf senescence and plant defense responses to cold may overlap, because several genes are activated both during senescence and by cold stimuli. It will be interesting to determine to what extent the pathway activated by senescence shows similarity to the pathway involved in plant defense response to cold and which TFs are involved in

transcriptional regulation during leaf senescence and plant defense responses. Through these investigations, we may obtain new insights into important inter-mediatory roles of ABA in the cold signal transduction pathway leading to leaf senescence. Also, the detection of the crosstalk between ABA and other signaling such as sugars, ET, and ROS pathways will offer more opportunities to illuminate the molecular mechanisms by which various signaling transduction pathways are integrated in plant life (Davies *et al.*, 2005; Ashraf, 2009).

Although great advances have been made in recent years in understanding the molecular basis of plant response and tolerance to abiotic stresses and hundreds of abiotic-responsive genes have been identified and the cellular function of many has been resolved, a huge gap still remains between the findings at the molecular level and the application of this knowledge at the whole plant level in the field. In addition, there is also a need in both public and private research sectors for crosstalk between disciplines involved in the molecular sciences and those seeking practical solutions to improve crop performance under abiotic conditions.

Declaration of interest

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