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The regulatory roles of ethylene and reactive oxygen species (ROS) in plant salt stress responses

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Abstract Soil salinity is one of the most commonly encountered environmental stresses affecting plant growth and crop productivity. Accordingly, plants have evolved a variety of morphological, physiological and biochemical strategies that enable them to adapt to saline growth conditions. For example, it has long been known that salinity-stress increases both the production of the gaseous stress hormone ethylene and the *in planta* accumulation of reactive oxygen species (ROS). Recently, there has been significant progress in understanding how the fine-tuning of ethylene biosynthesis and signaling transduction can promote salinity tolerance, and how salinity-induced ROS accumulation also acts as a signal in the mediation of salinity tolerance. Furthermore, recent advances have indicated that ethylene signaling modulates salinity responses largely via regulation of ROS-generating and ROS-scavenging mechanisms. This review focuses on these recent advances in understanding the linked roles of ethylene and ROS in salt tolerance.

Keywords Ethylene · ROS · Salt stress · Salt tolerance

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Introduction

Soil salinity is one of the most widespread plant abiotic stresses, affecting more than 6 % (~900 million hectares) of total world land area (Tuteja 2007; Munns and Tester 2008). This salt-affected land currently includes ~23 % of cultivated land, and is a problem which is worsening, particularly on agricultural land where crop growth is dependent upon irrigation. Almost all of the most important crop plants are glycophytes, and are therefore sensitive to salinity (Munns and Tester 2008; Cheeseman 2015). Moderate levels of environmental salt (e.g. 100 mM NaCl) are sufficient to cause dramatic decreases in the yield of most crops (Frommer et al. 1999; Munns and Tester 2008). High soil salinity is therefore a severe and growing problem, and is preventing the achievement of sustainable agriculture (Greenway and Munns 1980; Zhu 2002; Roy et al. 2014). There is therefore an urgent need to advance understanding of how different plants respond to salt stress, with the aim of developing crop plants better able to maintain growth and productivity on salinized lands via genetic modification or marker-assisted breeding.

Salinity-stress biology and the mechanisms of plant response to high salinity have been areas of study at physiological, biochemical, and molecular levels for many decades (Flowers et al. 1977; Greenway and Munns 1980). Plants have evolved many different strategies for tolerating high salt concentrations. In general, following the initial onset of salt stress, plants suffer two phases of stress, a rapid osmotic stress and a slower ionic stress (reviewed in Munns and Tester 2008). Accordingly, there are three major physiological adaptive mechanisms of salt tolerance: osmotic stress tolerance, maintenance of ion (especially Na^+ and K^+) homeostasis, and compartmentalization of Na^+ to reduce cytosolic Na^+ concentrations (reviewed in Zhu 2003; Deinlein et al. 2014). Many of the components involved in

the regulation of Na^+ and K^+ homeostasis have been characterized (e.g. SOSs, HKTs and NHXs; Zhu 2003; Sunarpi et al. 2005, Ji et al. 2013). However, the mechanisms of rapid osmotic phase tolerance remain poorly understood.

It has long been known that salinity-stressed plants exhibit dramatically altered levels of many endogenous signaling molecules, including abscisic acid (ABA), ethylene, gibberellin (GA), reactive oxygen species (ROS), nitric oxide (NO), and others. These signaling molecules, and their downstream signaling components, have been shown to play essential roles in salinity tolerance responses (Matsui et al. 2008; Harberd et al. 2009; León et al. 2014). For example, high environmental Na^+ concentrations lead to increased ABA levels and ABA signaling (Zhu 2002; Shinozaki and Dennis 2003), which in turn play vital roles in the regulation of transcriptome-level salinity responses (Xiong et al. 2001; Matsui et al. 2008). We previously found that the DELLA proteins, gibberellin-opposable growth inhibitors of the GA signaling pathway, play critical roles in plant tolerance to salt stress (reviewed in Harberd et al. 2009; Xu et al. 2014), and that salt-activated ABA and ethylene signaling pathways regulate plant growth and/or development via integration at the level of DELLA function (Achard et al. 2006). In addition, the salinity-induced reactive nitrogen species NO is an important regulator of salinity responses, and likely plays a role downstream of ABA (León et al. 2014).

Ethylene and ROS are important signaling molecules mediating numerous important biological processes, including root and root hair growth, cell fate determination, and responses to biotic and abiotic stress (Foreman et al. 2003; Apel and Hirt 2004; Chen et al. 2005; Steffens 2014). It has long been known that salinity stress increases the *in planta* production of ethylene and ROS. Recently, significant progress has been made in understanding the mechanism of how by which ethylene and ROS act as signaling molecules in mediating salinity tolerance, with important studies revealing that ethylene signaling modulates salinity stress response largely via regulation of ROS-generating and ROS-scavenging mechanisms (Jiang et al. 2013; Peng et al. 2014; Li et al. 2015a; Xia et al. 2015). This present review focuses on recent advances in understanding of the roles of ethylene and ROS in plant salinity response and of the crosstalk between these two classes of signaling molecules (Fig. 1).

Fine-tuning of ethylene biosynthesis and ethylene signaling promotes salt tolerance

Ethylene is often considered to be a plant stress hormone, because its synthesis is induced by various biotic and abiotic environmental stresses (Cao et al. 2007; Kazan 2015). Like many other environmental stresses, salinity promotes the production of ethylene in various species by modulating the activity of enzymes regulating ethylene biosynthesis (e.g. ACS2 and ACS7 in *Arabidopsis*; Achard et al. 2006; Dong et al. 2011). In addition, either endogenous overproduction of ethylene (as in the *eto1* mutant) or treatment with the ethylene precursor ACC can overcome the salt-induced restraint of *Arabidopsis* seed germination (Divi et al. 2010). Using a soil-based mutant screen system, we previously found that, at the vegetative growth stage of the life cycle, an increase in in vivo ethylene production (as in the *eto1*, *eto2* or *eto3* mutants) promotes the salinity tolerance of *Arabidopsis* plants grown on saline soil when transpiration is active, but not that of plants grown in in vitro conditions where transpiration is inhibited (Jiang et al. 2013). These observations indicate that salinity-induced ethylene is a potent promoter of salt tolerance at various developmental stages and in various conditions. These ideas are further supported by recent studies of rice SALT TOLERANCE1 (SIT1, a lectin receptor-like kinase), which positively regulates salinity tolerance by promoting the activity of the MAPK3/6 protein kinase and promoting ethylene production (Ai et al. 2014). However, in some other cases, elevated ethylene levels can adversely affect salinity tolerance. For instance, *Arabidopsis* plants overexpressing wheat *ACO1* display elevated ethylene levels, but decreased salinity tolerance (Chen et al. 2014). In addition, the *Arabidopsis acs7* mutant, which displays reduced ethylene production, exhibits increased salt tolerance at the seed germination stage (Dong et al. 2011). Although it has been suggested that the salinity tolerance of the *acs7* mutant might be the consequence of changed ABA sensitivity and altered transcript levels of some salinity-induced genes, the exact mechanism is yet to be understood (Dong et al. 2011). Moreover, a recent report has indicated that ethylene treatment of rice plants confers salt hypersensitivity (Yang et al. 2015). Taken together these various studies indicate that *in planta* ethylene levels can either negatively or positively affect the salinity sensitivity of plants, suggesting that fine-tuning of ethylene biosynthesis might be essential to salinity tolerance in plants.

Ethylene is perceived by endoplasmic reticulum-localized receptor kinases. Five ethylene receptor-encoding genes have been identified in the *Arabidopsis* genome: *ETHYLENE RESPONSE1* (*ETR1*), *ETR2*, *ETHYLENE INSENSITIVE4* (*EIN4*), *ETHYLENE RESPONSE SENSOR1* (*ERS1*), and *ERS2* (Hua and Meyerowitz 1998; Chen et al. 2005; Gallie 2015). Ethylene receptors negatively regulate ethylene responses, and ethylene activates downstream signaling pathways by deactivating these receptors. In essence, ethylene binds to the N-terminal region of the ethylene receptors with the help of a copper cofactor,

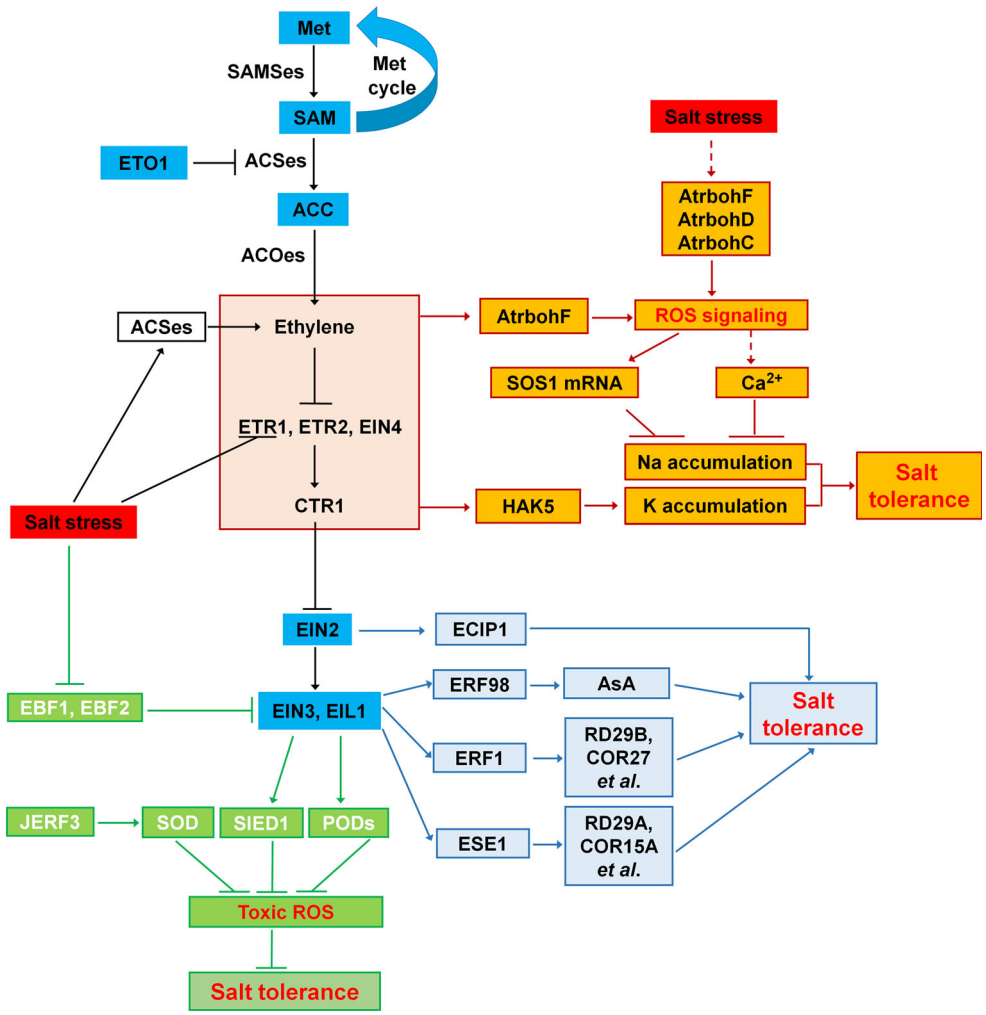

Fig. 1 Regulation of plant salt tolerance by the ethylene and ROS pathways. The figure is based mainly on findings from *Arabidopsis thaliana*. Arrows or lines with bars indicate positive or negative regulation, respectively. *ACC* aminocyclopropane-1-carboxylic acid synthase; *ACOes* ACC oxidases, *CTR1* constitutive triple response 1, *ACSes* 1-aminocyclopropane-1-carboxylic acid synthases, *AsA* ascorbic acid, *AtrbohF Arabidopsis thaliana* respiratory burst oxidase homolog F, *COR27* cold-regulated gene 27, *COR15A* cold-regulated protein 15a, *EBF1* EIN3-binding F-box protein 1, *EBF2* EIN3-binding F-box protein 2, *ECIP1* EIN2 C-terminus interacting protein 1, *EIL1* ethylene insensitive 3-like 1, *EIN2* ethylene insensitive 2, *EIN3* ethylene insensitive 3, *EIN4* ethylene insensitive 4, *ERF1* ethylene response factor 1, *ERF98* ethylene response factor 98, *ESE1* ethylene and salt inducible 1, *ETO1* ethylene overproducer 1, *ETR1* ethylene receptor 1, *ETR2* ethylene receptor 2, *HAK5* high affinity K^+ transporter 5, *JEFR3* jasmonate and ethylene response factor 3, *Met* methionine, *MPK3/6* MAP kinase 3/6, *PODs* peroxidase, *ROS* reactive oxygen species, *RD29A, RD29B, SAM S*-adenosylmethionine, *SAMSes* SAM synthetases, *SIED1* salt-induced and EIN3/EIL1-dependent 1, *SIT1* salt intolerance 1, *SOD* superoxide dismutase

resulting in deactivation of a Raf-like kinase CONSTITUTIVE TRIPLE RESPONSE1 (CTR1). Deactivation of CTR1 in turn allows a C-terminal section of ETHYLENE INSENSITIVE2 (EIN2) to be cleaved and translocated into the nucleus, thus stabilizing the transcription factors ETHYLENE INSENSITIVE3 (EIN3), EIN3-like1 (EIL1) and EIL2, and consequently activating transcription of downstream targets (e.g. ethylene response factors (ERFs): reviewed in Wang et al. 2002; Guo and Ecker 2004; Zhao and Guo 2011; Ju et al. 2012; Qiao et al. 2012; Wen et al. 2012; Merchante et al. 2013; Ju and Chang 2015; Zheng and Zhu 2016). In addition, cytoplasmic EIN2 mediates ethylene signaling also via imposing the translational repression of *EIN3 BINDING F-box* 1 (*EBF1*) and *EBF2* mRNA (Li et al. 2015a; Merchante et al. 2015). Accumulating evidence indicates that ethylene signaling positively regulates plant salt tolerance, including the following key observations. (1) Expression of the *ETR1* gene is downregulated by salinity treatment in *Arabidopsis*, whilst loss of ETR1 or EIN4 function confers accelerated germination under salinity conditions (Cao et al. 2007; Wilson et al. 2014); in contrast, *etr1-1*, *ein4-1* and *etr2-1* gain-of-function mutants are insensitive to ethylene and display salt hypersensitivity compared to wild type (Cao

et al. 2008). (2) Loss of CTR1 function leads to constitutive ethylene response and salt tolerance at both germination and vegetative growth stages (Achard et al. 2006; Cao et al. 2007; Jiang et al. 2013; Peng et al. 2014). (3) Loss of EIN2 function leads to delayed germination and decreased survival rate in saline conditions (Wang et al. 2007; Peng et al. 2014), suggesting that EIN2 positively regulates salt tolerance; further studies have indicated that EIN2 positively regulates salt tolerance not only by affecting the level of the EIN3 protein (Peng et al. 2014), but also by negatively regulating ABA synthesis or through physical interaction with an MA3 domain-containing protein (ECIP1, a positive regulator of salt tolerance) (Ghassemian et al. 2000; Wang et al. 2007; Lei et al. 2011). (4) A recent report indicates that salinity leads to accumulation of the EIN3 protein via both EIN2-dependent and EIN2-independent pathways and by promoting the degradation of the F-box protein EBF1 and EBF2, suggesting that salinity-induced accumulation of EIN3 confers salt tolerance (Peng et al. 2014); this is supported by genetic evidence showing that overexpression of EIN3 promotes salinity tolerance, whilst loss of EIN3 function confers salinity sensitivity in *Arabidopsis* (Peng et al. 2014). (5) It has been repeatedly confirmed that overexpression of EIN3 target genes (e.g. *ERFs* and *SALT-INDUCED EIN3/EIL1-DEPENDENT1* (*SIED1*)) in various species leads to salt tolerance (Zhang et al. 2004; Zhang et al. 2011; Cheng et al. 2013; Imen et al. 2014). (6) Salinity induces transcriptional changes of many genes in an EIN3/EIL1-dependent manner (Peng et al. 2014). All of the above evidence indicates that salinity can lead to enhanced ethylene signaling (e.g. by stabilizing EIN3 and EIL1), which in turn can act as a potent promoter of salt tolerance. However, Yang et al. (2015) in contrast found that overexpression *OsEIL1* and *OsEIL2*, rice homologs of EIN3, causes salt hypersensitivity at the seedling stage, whilst loss of *OsEIL1* and *OsEIL2* function promotes salt tolerance, suggesting that EIN3-dependent ethylene signaling negatively, rather than positively, affects salinity tolerance in rice, at least at the seedling stage.

ROS homeostasis is essential for the ethylene regulation of salt tolerance

Reactive oxygen species (ROS), such as $^1O_2$, H_2O_2, O_2^- and HO, are highly reactive molecules, capable of causing oxidative damage to protein, DNA and lipids (Apel and Hirt 2004; Miller et al. 2010). However, as well as being toxic molecules, ROS also act as important signaling molecules regulating many important biological processes, such as growth, development, and responses to abiotic and biotic stresses (Foreman et al. 2003; Miller et al. 2008). Because ROS play a dual role in plants, ROS synthesis and ROS scavenging machineries are tightly regulated to achieve appropriate levels of ROS at different developmental stages and in different growing environments (Foreman et al. 2003; Jayakumar et al. 2014). It has long been known that, in abiotic and biotic stress conditions, the levels of ROS in plant tissues can be dramatically elevated. In particular, salinity stress causes elevated ROS levels, and these elevated ROS levels play a dual role in the salinity responses of plants: (1) ROS act as toxic byproducts causing oxidative damage; (2) ROS act as signaling molecules mediating salt tolerance (Chung et al. 2008; Miller et al. 2008; Ma et al. 2012; Jiang et al. 2013). Because the oxidative damage aspects of salinity-stress induced ROS have already been extensively reviewed (Miller et al. 2010; Mittler et al. 2011; Steinhorst and Kudla 2014), we here focus on the signaling roles of ROS, with special focus on its roles in ethylene-dependent salinity tolerance mechanisms.

Previous studies have shown that a variety of ethylene-regulated biological processes are associated with the regulation of ROS homeostasis (Wilkinson and Davies 2009; He et al. 2011; Jiang et al. 2013; Peng et al. 2014; Xia et al. 2015). For example, ethylene promotes tolerance to potassium deprivation by promoting ROS production (Jung et al. 2009; Schachtman 2015). Also, ethylene mediates UV-B- and ozone-induced stomatal closure through peroxidase-dependent H_2O_2 synthesis in *Vicia faba* (Wilkinson and Davies 2009; He et al. 2011). We previously found that the *soil-salinity sensitive1-1* (*sss1-1*) mutant of *Arabidopsis* displays a salt hypersensitivity phenotype due to loss-of-function of AtrbohF (one member of a family of ten NADPH oxidases in *Arabidopsis* that catalyze production of ROS) and the consequent absence of salinity-induced ROS accumulation in root vasculature, suggesting that AtrbohF confers salinity-induced vasculature-specific ROS accumulation and salinity tolerance (Jiang et al. 2012). Moreover, we found that, whilst an increase in ethylene production (e.g. in *eto1*) or constitutive activation of ethylene signaling (e.g. in *ctr1*) leads to increased accumulation of ROS in root vascular tissue and increased salt tolerance, *eto1 atrbohf* and *ctr1 atrbohf* double mutants both lack salinity-induced ROS accumulation and are hypersensitive to salinity stress (with a sensitivity similar to that of the *atrbohf* single mutant). In addition, we found that ethylene signaling positively regulates the levels of transcripts encoding AtrbohF in root vasculature via the ETR1-CTR1-dependent signaling pathway, suggesting that ethylene signaling promotes salt tolerance by enhancing tissue-specific, AtrbohF-dependent ROS biosynthesis in root vasculature tissue (Jiang et al. 2013). Intriguingly, another study indicated that ethylene affects stomatal aperture also through regulation of AtrbohF-dependent ROS production in guard cells (Desikan

et al. 2006). These studies thus indicate that AtrbohF-dependent ROS production may be an important downstream mediator of ethylene signaling cascades. The idea that ethylene promotes salinity tolerance by increasing ROS production is further supported by studies of the rice receptor-like kinase SIT1. The SIT1 protein resides on the plasma membrane, and its kinase activity is rapidly activated by salinity stress, causing activation of MPK3/6, in turn promoting ethylene synthesis and ROS production, and hence increasing the salt tolerance of rice (Ai et al. 2014).

In addition to ethylene, ABA and JA treatments also induce stomatal closure via AtrbohF- and AtrbohD-dependent mechanisms (Kwak et al. 2003; Suhita et al. 2004), and there are reports indicating the interconnection of ethylene, JA and ABA in the regulation of stomatal closure. For example, ethylene treatment inhibits ABA-induced stomatal closure (Tanaka et al. 2015), and *ost1* (an ABA-insensitive mutant) displays compromised JA-induced stomatal closure (Suhita et al. 2004), whilst *jar1* (a JA-insensitive mutant) exhibits diminished ABA-induced stomatal closure (Suhita et al. 2004). Nevertheless, the overall relationships between ethylene-, JA- and ABA-regulated stomatal closure remain largely unknown (Murata et al. 2015).

As shown above, ethylene in many cases positively regulates the biosynthesis of ROS. However, there are also alternative cases in which ethylene appears to negatively regulate ROS production in specific circumstances. For example, overexpression of JERF3, an ERF protein, enhances tolerance to salt, drought, and freezing in tobacco seedlings by reducing ROS accumulation (Wu et al. 2008). In addition, a recent study indicated that salinity-induced accumulation of EIN3/EIL1 promotes salinity tolerance, likely by enhancing ROS scavenging at the seedling stage in Petri-dish conditions (Peng et al. 2014). This study showed that EIN3/EIL1 regulates transcriptional change of over 200 genes under salinity conditions: 92 *salt-induced EIN3/EIL1-dependent* (*SIED*) genes and 121 *salt-repressed EIN3/EIL1-dependent* (*SRED*) genes were found. Genes encoding oxidoreductases were found to be enriched in *SIED*, whilst many genes encoding peroxidases (PODs) were found to be induced by salt treatment via an EIN3/EIL1-dependent mechanism. Accordingly, an *ein3 eil1* double mutant displayed lower levels of POD-encoding transcripts, higher levels of ROS, and consequent hypersensitivity to salt stress (Peng et al. 2014). These results thus suggest an alternative mechanism to explain how EIN3 promotes salt tolerance, namely by enhancing the scavenging of toxic ROS.

The ROS steady-state is regulated by a complex network comprising more than a hundred genes (Mittler et al. 2004). It is therefore not surprising to find that ethylene promotes both ROS synthesis and scavenging at different developmental stages, under different growth conditions, and in different tissues following the onset of salinity treatments (Jiang et al. 2013; Peng et al. 2014). In addition, ethylene-dependent salinity tolerance mechanisms have been shown to have wide-ranging crosstalk with other salt tolerance pathways (Xia et al. 2015). It is therefore possible that under different experimental conditions, and at different developmental stages, ethylene signaling interacts preferentially with different pathways and thus differentially regulates ROS synthesis and scavenging mechanisms. However, the exact mechanisms for these differential preferences is yet to be understood.

Ethylene signaling regulates Na^+/K^+ homeostasis via ROS-dependent and ROS-independent mechanisms

High soil sodium ion (Na^+) concentrations cause inhibition of K^+ uptake, increased loss of K^+, and an increase in Na^+ uptake, thus leading to a decrease in plant K^+/Na^+ ratio (Zhu 2003; Shabala and Cuin 2008). It has long been known that maintenance of Na^+/K^+ homeostasis is essential for soil-salinity tolerance. A variety of mechanisms contributing to maintenance of Na^+/K^+ homeostasis in high salinity conditions have been characterized, including: reduced net Na^+ uptake into the root (i.e. decreased Na^+ influx and/or increased Na^+ efflux) and reduced xylem loading (Zhu 2003); retrieval of Na^+ from the transpiration stream xylem sap during transport to the shoot (Sunarpi et al. 2005; Munns and Tester 2008); sequestration of Na^+ into vacuoles to avert ion toxicity in the cytosol (Amtmann and Sanders 1999); and excretion of excess Na^+ by the salt glands characteristic of some halophytic plants (Smith et al. 2010). Among the best understood of the mechanisms regulating Na^+ homeostasis are the SALT OVERLY SENSITIVE1 (SOS1) (Shi et al. 2000; Qiu et al. 2002; Lin et al. 2009; Zhou et al. 2015; reviewed in Zhu 2003 and Ji et al. 2013) and HIGH-AFFINITY K^+ TRANSPORTER1 (HKT1) HKT1 pathways (Mäser et al. 2002; Sunarpi et al. 2005). Either tissue-specific (e.g. vasculature-specific) overexpression of *AtHKT1* or ectopic overexpression of *AtSOS1* causes reduced shoot Na^+ accumulation, thus lowering the Na^+/K^+ ratio and in turn promoting the salinity tolerance of *A. thaliana* (Schachtman and Schroeder 1994; Shi et al. 2000; Møller et al. 2009; Yang et al. 2009; reviewed in Zhu 2002 and Horie et al. 2009).

Previous studies have indicated that ROS likely play important roles in the regulation of Na^+ and K^+ homeostasis (Apel and Hirt 2004; Miller et al. 2010; Mittler et al. 2011). For example, studies of poplar callus cells indicated

that salinity-induced production of H_2O_2 promotes salt tolerance by maintaining reduced Na^+ and increased K^+ levels, thus lowering the Na^+/K^+ ratio (Zhang et al. 2007; Sun et al. 2010). Ma et al. (2012) found that, under in vitro conditions, the *atrbohd atrbohf* double mutant, but not the *atrbohd* or *atrbohf* single mutants, exhibit overaccumulation of Na^+ in saline conditions. This overaccumulation of Na^+ adversely affects K^+ influx, suggesting that AtrbohD and AtrbohF redundantly regulate Na^+/K^+ homeostasis (at least under the particular conditions used in this study). Ma et al. (2012) also showed that *atrbohd atrbohf* displays reduced K^+ influx not only in saline conditions, but also in control (non-saline) conditions, suggesting that ROS produced by AtrbohD and AtrbohF is essential for the regulation of K^+ homeostasis. In contrast, we recently showed that AtrbohF alone confers essential regulation of Na^+ homeostasis and salt tolerance in plants grown in soil (Jiang et al. 2012). We found that the salinity-induced, AtrbohF-mediated increase in root vascular ROS levels restricts xylem-sap Na^+ content, thus reducing transport of Na^+ from root to the shoot, resulting in a reduced Na^+/K^+ ratio and contributing to soil-salinity tolerance (Jiang et al. 2012). Despite the wealth of evidence indicating that salinity-induced ROS production confers regulation of Na^+ and K^+ homeostasis, the exact molecular mechanism of this regulation remains largely unknown. One possible mechanism is that ROS regulate Na^+ and K^+ homeostasis by regulating the activity of PM Ca^{2+}-permeable channels, which in turn regulate the activity of downstream components such as MPKs, SOS2 and other CBLs (Mori and Schroeder 2004; Pottosin et al. 2014). This possibility is attractive because ROS regulation of cytosolic free Ca^{2+} concentrations are known to play important roles in regulating root hair growth and in long-distance signal propagation (Foreman et al. 2003; Steinhorst and Kudla 2014). Alternatively, ROS may regulate Na^+ and K^+ homeostasis via effects on the stability of SOS1 (*Salt Overly Sensitive 1*, encoding the PM Na^+/H^+ anti-porter) mRNA and on the activity of the PM H^+-ATPase (Zhang et al. 2007; Chung et al. 2008; Ma et al. 2012).

Ethylene has also been shown to play important roles in the regulation of Na^+ and K^+ homeostasis. Yang et al. (2013) showed that ethylene can facilitate the retention of K^+ in saline conditions. Our recent study found that, following onset of salinity treatment, and in conditions where transpiration is active, ETR1-CTR1 dependent ethylene signaling not only promotes K^+ retention, but also significantly contributes to inhibition of root-to-shoot Na^+ delivery by regulating AtrbohF-dependent vasculature-specific ROS production (Jiang et al. 2013). This finding provides genetic evidence that AtrbohF-ROS regulation of Na^+ homeostasis acts downstream of ethylene signaling. Intriguingly, we also found in contrast that ethylene promotes the retention of K^+ via an AtrbohF-independent mechanism, possibly via increase in the level of transcripts encoding AtHAK5 (a high-affinity K^+ transporter) in saline conditions. Because ethylene can increase *AtHAK5* transcript levels in either low K^+ or high Fe stress conditions (Jung et al. 2009; Li et al. 2015b), and because the effect of low K^+ on levels of transcripts encoding AtHAK5 is AtrbohC-dependent (Shin and Schachtman 2004), it is possible that ethylene promotes the retention of K^+ in saline conditions via a mechanism dependent upon the function of another NADPH oxidase (i.e. AtrbohC rather than AtrbohF). However, this possibility requires further experimental verification.

Conclusions and perspectives

There has recently been significant progress in our understanding of the roles of ethylene and ROS in plant salinity responses, and of how ethylene and ROS interact with one another to regulate salt tolerance. Nevertheless, many questions remain unanswered, amongst which are the following. (1) Although the majority of the studies reviewed above suggest that increased levels of ethylene and associated activation of the ethylene signaling pathway are positively associated with salinity tolerance, there are instances where such increases, at particular developmental stages, or under particular experimental or environmental conditions, or in different species, are suggested to negatively affect salinity tolerance. The molecular basis of these cases of negative association remain largely unknown. (2) Current knowledge suggests that ethylene promotes salinity tolerance either by enhancing ROS scavenging (thus attenuating oxidative damage) or by promoting root-vasculature-specific accumulation of ROS (thus enhancing Na^+/K^+ homeostasis). Nevertheless, the precise molecular mechanisms by which ethylene tunes the ROS scavenging and ROS generating machinery to maintain proper ROS levels in different tissue and cellular compartments remains unclear. (3) The activity of AtrbohF is regulated at both transcriptional and posttranscriptional levels. Although previous reports have indicated that ethylene positively regulates AtrbohF transcript levels, it remains unclear if ethylene affects the activity of AtrbohF at the posttranscriptional level, for example, by regulating the protein kinases (e.g. OST1 and CIPK26) that directly phosphorylate and regulate the activity of AtrbohF (Sirichandra et al. 2009; Kimura et al. 2013). (4) It has been suggested that elevated ROS can cause increased cytosolic free Ca^{2+} concentrations, which in turn can lead to a reduced Na^+/K^+ ratio. Meanwhile, the ROS produced by AtrbohF has been shown to play an important role in the regulation of Na^+/K^+ homeostasis and salt tolerance. Further work is

needed to determine if AtrbohF-produced ROS enhances the Na^+/K^+ ratio by Ca^{2+}-dependent mechanisms. (5) Like other stress signals, salinity results in the enhanced production of ROS that act as signaling molecules to trigger acclimation responses. In the future, the application and development of new cellular imaging and real-time detection tools will advance our understanding of the tissue- and cell-specificity of salinity-induced ROS responses. Such studies will further advance our understanding of the roles of ethylene and ROS in salt tolerance, and will potentially provide new routes towards the development of salt-tolerant crops.

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Author contribution MZ, CJ, JACS and NPH conceived and wrote the paper.

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