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Crosstalk Between Ethylene and JA/ABA/Sugar Signalling in Plants Under Physiological and Stress Conditions

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ABSTRACT

Growth, development and defence responses of plants are governed through signalling networks that connect inputs from nutrient status, hormone cues and environmental signals. Plant hormones as endogenous signals are essential for modulating plant defence responses and developmental processes. Ethylene (ET), a gaseous hormone, is widely established as a regulator of these processes. Over the last two decades, substantial research reports have revealed the interaction between ET and other endogenous cues, including abscisic acid (ABA), sugars and jasmonates (JAs). However, these reports showed numerous conflicting or contrasting conclusions. For example, some reports revealed that ET and ABA/sugar/JA signalling synergistically regulate plant growth, development and defence responses, whereas other reports demonstrated that these cues antagonistically modulate these processes. This suggests that the crosstalk between ET and JA/ABA/sugar signalling is very complex, that is, these hormones can function either antagonistically or coordinately, dependent on the given biological process (e.g., under physiological or stress conditions). Further analysis found that whether synergistic or antagonistic actions exist between ET and JA/ABA/sugar signalling is determined by the induction/inhibition of their respective master transcription factors in these pathways. We here summarise the most recent advances and outstanding questions and/or challenges in the area of crosstalk between ET and ABA/sugar/JA signalling under physiological or stress conditions.

1 | Introduction

In most growth/developmental and response to environments, ethylene (ET) achieves its function by interacting with other phytohormones (Ma et al. 2014). Among them, ET widely interacts with other hormone signals at multiple biochemical levels to achieve its diverse functions. The interactions between ET and other hormones have been reviewed elsewhere (Yoo, Cho, and Sheen 2009; Zhao and Guo 2011). We here focus on crosstalk between ET and jasmonic acid (JA)/abscisic acid (ABA)/sugar signalling in plants under physiological and stress conditions. We further analyse why numerous conflicting or contrasting conclusions under physiological or stress conditions are generated.

2 | ET Synergy With JA Signalling Occurs Under Physiological Conditions, Whereas ET Antagonism of JA Signalling Arises Under Stress Conditions

Studies have claimed that both ET and JA function synergistically and concomitantly to modulate root hair development (Zhu et al. 2006) and plant defence against necrotrophic

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fungi (Penninckx et al. 1998; Thomma et al. 1998; Thomma et al. 1999) (Figure 1A). Further analysis found that JAs may elevate root hair production, by an interaction with the canonical ET signal transduction pathway (several ET signalling components [ETR2, EIN2, EBF1/EBF2, and EIN3/EIL1] are involved) (Zhu et al. 2006). However, the protein interactions through transcription factor level and/or regulation at other levels remain to be determined. Similarly, although they investigated ET and JA function synergistically plant defence against necrotrophic fungi (Penninckx et al. 1998; Thomma et al. 1998, 1999), the interactions of key factors remain to be determined. On the other hand, other reports show that JA and ET exhibit opposite influences on plant growth, development and defence responses. JA delays flowering (Robson et al. 2010); suppresses the ozone-induced spreading of cell death (Rao et al. 2000; Tuominen et al. 2004), seed germination (Miersch et al. 2008), and hypocotyl elongation upon light irradiation (Chen, Sonobe, et al. 2013); raises freezing tolerance (Hu et al. 2013); and promotes anthocyanin accumulation (Qi et al. 2011). Conversely, ET has been shown to promote flowering (Ogawara et al. 2003), increase ozone-induced spreading of cell death (Overmyer et al. 2000), seed germination (Linkies et al. 2009; Linkies and Leubner-Metzger 2012), promote hypocotyl elongation in the light (Zhong et al. 2012), inhibit freezing tolerance (Shi et al. 2012) and reduce anthocyanin accumulation (Jeong et al. 2010) (Figure 1B,D). Interestingly, ET by EIN3 concomitantly activates two contrasting pathways, that is, the PIF3 (the transcription factor phytochrome-interacting factor 3)-dependent hypocotyl growth-promoting pathway and an ERF1 (ET response factor 1)-mediated hypocotyl growth suppressing pathway (Zhong et al. 2012). However, the protein interactions of ET-mediated hypocotyl growth remain to be determined (Chen, Sonobe, et al. 2013). Moreover, ET may inhibit anthocyanin biosynthesis by several ET signalling components (ETR2, EIN2 and EIN3/EIL1) suppressing the expression of *SUC1* (encoding a sucrose transporter) in *Arabidopsis* (Jeong et al. 2010) (Figure 1B,D). In fact, later studies have proved that EIN3 directly inhibits the expression of *SUC2* (encoding a major sucrose transporter) in *Arabidopsis* (Tong et al. 2022; Mu et al. 2022). By contrast, the JA-ZIM-domain proteins (such as JAZ [jasmonate ZIM-domain]) interact with the WD-Repeat/bHLH/MYB complexes (TT8 and GL3 [bHLH transcription factor, Transparent Testa8 and Glabra3], and MYB75 and GL1 [R2R3 MYB transcription factors, MYB75 and Glabra1]) to promote JA-mediated anthocyanin biosynthesis (Qi et al. 2011).

Therefore, ET synergy with JA signalling occurred under low exogenous JA levels, whereas ET antagonism of JA signalling was manifested under high exogenous JA levels. That is, under low exogenous JA (0.01 μM), ET synergises with JA signalling to regulate root hair development (Zhu et al. 2006) and plant defence against necrotrophic fungi (Penninckx et al. 1998; Thomma et al. 1998; Thomma et al. 1999). However, under higher exogenous JA (25 μM JA vs. 0.01 μM), an increase of 2500-fold, anthocyanin accumulation is promoted (Qi et al. 2011), whereas excess ET inhibits anthocyanin accumulation (Jeong et al. 2010). Also, ET antagonism of JA signalling related to other phenotypes, for example, flowering, ozone-induced spreading of cell death, seed germination, hypocotyl elongation and freezing tolerance, used JA under stress conditions (Overmyer et al. 2000; Rao et al. 2000; Ogawara et al. 2003; Tuominen et al. 2004; Miersch et al. 2008; Linkies et al. 2009; Robson et al. 2010; Jeong et al. 2010; Zhong et al. 2012; Shi et al. 2012; Linkies and Leubner-Metzger 2012; Hu et al. 2013; Chen, Dodd, et al. 2013). Notably, although ET antagonises JA signalling in the regulation of anthocyanin biosynthesis (Jeong et al. 2010; Qi et al. 2011), it has been reported that JA does not play a role in the natural developmental regulation of anthocyanin accumulation (Tamari et al. 1995).

Although many reports have revealed that ET synergy with JA signalling occurs under physiological conditions, whereas ET antagonism of JA signalling rises under stress conditions, the relative molecular mechanism remains to be determined. Only a few reports further investigated relative molecular mechanisms. For example, cold stress negatively modulates the

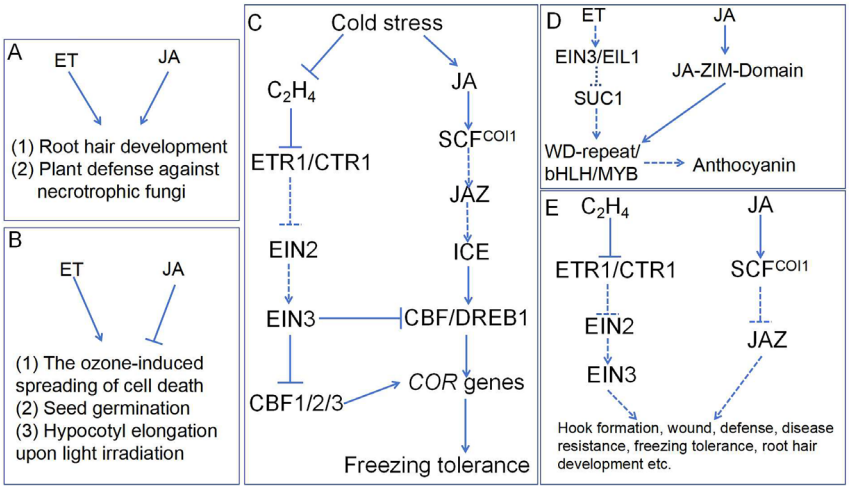

FIGURE 1 | Ethylene (ET) synergy or antagonism with jasmonic acid (JA) signalling in the regulation of plant growth, development and defence responses. (A) ET synergy with JA signalling. (B, D) ET antagonism with JA signalling. (C) ET antagonism with JA signalling regulates freezing tolerance. (E) ET or JA canonical signal transduction pathways synergistically and antagonistically regulate plant growth, development and defence. Solid lines indicate direct regulation, whereas dotted lines indicate either indirect regulation or regulation in an unknown manner. Blunt arrows indicate negative regulation and pointed arrows indicate positive regulation.

freezing tolerance in *Arabidopsis* by slowly accumulating EIN3 protein by the cold stress-EIN3-CBF1/2/3-COR gene pathway (Shi et al. 2012; Figure 1C). Exogenously applied 1-aminocyc lopropane-1-carboxylic acid (ACC, a precursor to ET) was excess levels (10 μM) for observing genetic phenotypes and analysing gene expression and protein expression. By contrast, cold stress-mediated JA signalling positively regulates the freezing tolerance in *Arabidopsis* by the JA-ICE-EIN3-CBF-COR/RD pathway (Hu et al. 2013; Figure 1C). In this study, exogenously applied JA was also excess levels (100 μM) for analysing gene expression and protein expression and observing genetic phenotypes (Figure 1C). It needs to be further investigated if these functions under excess ET and JA levels are consistent under physiological environments. It has been proposed that both synergistic and antagonistic patterns of interaction between JA and ET would assist plant adaptation to fluctuating environments by the JA-SCF^{COI1}-JAZ-MYC-CYP79B3-camalexin and ET-ETR1/CTR1-EIN2-ERF1-PDF1.2 molecular modules (Song et al. 2014).

By carefully analysing relative reports, we found that hook formation, wound, defence, disease resistance, freezing tolerance and root hair development are synergistically and antagonistically regulated by ET or JA canonical signal transduction pathways (Figure 1E). Although these authors provided some interpretations for their contradictory conclusions, including data resulting from different organs or developmental stages, further analysis is necessary.

During plant growth and development, the use of high concentrations of exogenous JA/ET raises concerns about biological significance, specificity and physiological relevance. Therefore, results from these types of studies should be carefully analysed and interpreted.

2.1 | ACC Can Function as a Signal Independent of ET

Under exogenously applied low (0.0001–0.01 μM) or high (0.05–2.5 μM) ACC, root growth is always inhibited in *Arabidopsis* (Cheng et al. 2009). We further analysed this paper and found that root length of *ein3* mutant was decreased under exogenously applied low ACC and increased under high/excess ACC (Cheng et al. 2009), indicating ET signalling promotes root growth under physiological conditions. However, root length of *ein2* mutant was increased under both exogenously applied low and high/excess ACC (Cheng et al. 2009), indicating ET signalling inhibits root growth under physiological conditions. Obviously, these conclusions are contradictory. On the other hand, root length of *aba2-2* mutant was increased under both exogenously applied low ACC (Cheng et al. 2009), indicating ABA signalling promotes root growth under physiological conditions. This may be caused by using exogenously applied ACC but not ET. Recent reports have revealed that ACC can function as a signal independent of ET (Mou et al. 2020; Li et al. 2020), supporting our deduction. Moreover, in rice, root growth is promoted under exogenously applied low ET (0.01 ppm) and inhibited high/excess ET (0.1–100 ppm) (Ma et al. 2014), which is not consistent with using ACC of the findings in Cheng et al. (2009). This further supports our deduction.

2.2 | ET and ABA Signals Synergistically and Negatively Regulate Root Growth Under High/Excess ABA (But Not High ACC/ET) Conditions, Whereas Two Signals Synergistically and Positively Modulate Root Growth Under Physiological Conditions

It has been widely reported that in *Arabidopsis*, ET and ABA synergistically regulate root growth, that is, in *Arabidopsis*, the ABA pathway inhibits root growth through ET pathway (Beaudoin et al. 2000; Ghassemian et al. 2000; Cheng et al. 2009; Ma et al. 2014; Huang et al. 2022). However, based on our analysis, we conclude that unlike JA/ET interactions, ET and ABA signals synergistically and negatively regulate root growth under high/excess ABA (but not high ACC) conditions, whereas two signals synergistically and positively modulate root growth under physiological conditions (Figure 2A,B).

Root growth is promoted by exogenously applied low ABA (0.01–0.1 μM) or physiological conditions and is inhibited by exogenously applied high/excess ABA (0.1–100 μM) (Ghassemian et al. 2000). This indicates that low ABA promotes root growth, whereas high/excess ABA inhibits root growth. Furthermore, root length is increased in *abi1-1* mutant under exogenously applied high ABA (10 μM) (Ghassemian et al. 2000), supporting ABA signalling inhibits root growth under high ABA levels. Similarly, the root length of *Arabidopsis ein3-1* mutant was elevated under high ABA conditions, indicating ET signalling inhibits root growth under high ABA conditions (Ghassemian et al. 2000). However, in rice, root growth is promoted under exogenously applied low ET (0.01 ppm) and inhibited high/excess ET (0.1–100 ppm) (Ma

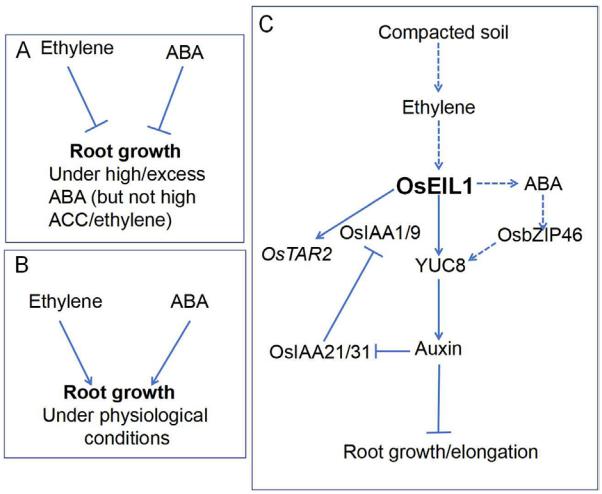

FIGURE 2 | Ethylene (ET) synergy with abscisic acid (ABA) signalling in the regulation of root growth. (A) ET and ABA signals synergistically and negatively regulate root growth under high/excess ABA (but not high 1-aminocyclopropane-1-carboxylic acid [ACC]/ET) conditions. (B) ET and ABA signals synergistically and positively modulate root growth under physiological conditions. (C) ET regulates root growth by modulating ABA and auxin levels. Figure A,B is the compendious interpretation of Figure C. Solid lines indicate direct regulation, whereas dotted lines indicate either indirect regulation or regulation in an unknown manner. Blunt arrows indicate negative regulation and pointed arrows indicate positive regulation.

et al. 2014), indicating high/excess ACC/ET inhibits root growth, whereas low ACC/ET promotes root growth. This is similar to ABA regulatory mode. Therefore, two reports indicate that ET and ABA signals synergistically and negatively regulate root growth under high ABA conditions, whereas two signals synergistically and positively modulate root growth under physiological conditions (Figure 2A,B).

Another report (Beaudoin et al. 2000) shows that root length significantly declined with increasing exogenously applied high/excess ABA levels (0.1–100 μM). Furthermore, the root growth of *abi1-1* mutant is increased relative to wild-type seedlings under exogenously applied high/excess ABA levels (0.1–100 μM), but not high ACC (0.2 μM). These findings indicate that ABA signalling inhibits root growth under high/excess ABA, but not high ACC. However, the root growth of *ein2-45* mutant is promoted under either high ACC (0.2 μM) or high/excess ABA levels (0.1–100 μM), indicating ET inhibits root growth under either high ACC or high/excess ABA. This report indicates that ET and ABA synergistically and negatively regulate root growth under high/excess ABA conditions, but not under high ACC conditions (Figure 2A,B).

Although it has been widely reported that ET and ABA signals synergistically and negatively regulate root growth, the relative molecular mechanism remains to be not widely revealed. In rice, some reports revealed the molecular mechanism of root growth. Compacted soil stimulates ET production by smaller air-filled soil pores. The accumulation of ET elevates OsEIL1 activation, which in turn promotes the expression of auxin biosynthesis by OsYUC8 (Huang et al. 2022; Qin et al. 2023). The elevated auxin response in epidermal cells of elongation and meristematic zones blocks epidermal cell elongation, which in turn inhibits root elongation. At the same time, ET signalling indirectly elevates ABA biosynthesis within vascular tissues, triggering radial expansion of root cortical cells, thus inhibiting root elongation in compacted soil (Huang et al. 2022). In parallel, ET signalling indirectly increases ABA biosynthesis, and elevated ABA mediated-OsbZIP46 activates the expression of YUC8 to induce auxin biosynthesis, which in turn suppresses epidermal cell elongation and thereby inhibits root elongation. As a result, short and swollen roots are formed (Qin et al. 2023). Moreover, upon ET treatment, the induced-auxin signalling results in early degradation of OsIAA21/31, thereby allowing the elevation of OsEIL1-activated *OsTAR2* expression by OsIAA1/9 to amplify signal. The induced roles of OsIAA1/9 involve the recruitment of the histone acetyltransferase OsGCN5 for histone modification and chromatin activation, which leads to root elongation (Zhou et al. 2022).

Together, three reports indicate that ET and ABA signals synergistically and negatively regulate root growth under high/excess ABA (but not high ACC) conditions, whereas two signals synergistically and positively modulate root growth under physiological conditions.

3 | ET Antagonism of ABA Signalling Regulates Seed Germination Rate/Early Seedling Growth Under Stress Conditions

Seed germination rate/early seedling growth of the *aba2* mutant was higher/accelerated relative to that of wild-type seeds following far-red/red light treatment (Seo et al. 2006), under high salt (NaCl)/ABA concentration (González-Guzmán et al. 2002). In contrast, the seed germination rate/early seedling growth of the *ein2* mutant was reduced/delayed relative to that of wild-type seeds under high ABA, osmotic stress and salt stress (Wang et al. 2007). However, under physiological conditions, seed germination/early seedling growth was not significantly different between *aba2*, *ein2* and wild-type (González-Guzmán et al. 2002; Seo et al. 2006; Wang et al. 2007). Together, these data suggest that ABA antagonises ET signalling to control seed germination rate/early seedling growth under high ABA concentrations, but not under physiological conditions. Therefore, it is important to consider these specific conditions, as an inaccurate appreciation could lead to incorrect conclusions. To further investigate the molecular mechanism of ABA antagonism of ET signalling, Cheng et al. (2009) analysed microarray and genetic epistasis data and measurements of endogenous ET and ABA levels of the related mutants under basal conditions. They concluded that ET and ABA facilitate their antagonistic effects on seed germination/early seedling growth by controlling hormonal biosynthesis/catabolism. However, this study did not appreciate that ABA antagonism of ET signalling is specific to stress/high ABA levels conditions and does not occur under physiological conditions.

4 | ET Antagonism of ABA Signalling in Crops

In addition to *Arabidopsis*, ET antagonism of ABA signalling widely regulates crop growth and development. In rice, high concentrations of ET in grains suppress the cell division of endosperm and the filling of grains, at the same time ABA antagonises the negative influences of ET. Therefore, a lower ratio of ET to ABA in rice spikelets is necessary for maintaining a faster grain filling rate (Yang et al. 2006). In this study, authors made their conclusions on the basis of endogenous ABA and ACC/ET contents, that is, under physiological conditions. Therefore, their conclusions are physiologically relevant and thus have biological significance and specificity.

In guard cell response, whereas both ET and ABA obviously induce stomatal closure, ABA can antagonise ET-stimulated stomatal closure through suppressing the ABA signalling transduction pathway (Tanaka et al. 2005; Desikan et al. 2006). In wheat, this contrasting influence of ABA and ET interaction is more distinct under either soil drying conditions or older leaves (Chen, Dodd, et al. 2013).

Therefore, ET and ABA have extensive interactions in regulating plant growth, development and adaptive responses. However, their interaction is complicated, depending on tissue/organ, biological process, species and growth conditions.

5 | Sugars Induce ABA Biosynthesis and Thus Complicate ET and ABA Interactions

ET and ABA signalling confer antagonistic effects on hyponastic growth (Benschop et al. 2007), stomatal aperture (Tanaka et al. 2005), post-germination seedling growth (Zhou et al. 1998), seed germination (Beaudoin et al. 2000; Ghassemian et al. 2000), disease resistance and defence gene expression (Anderson

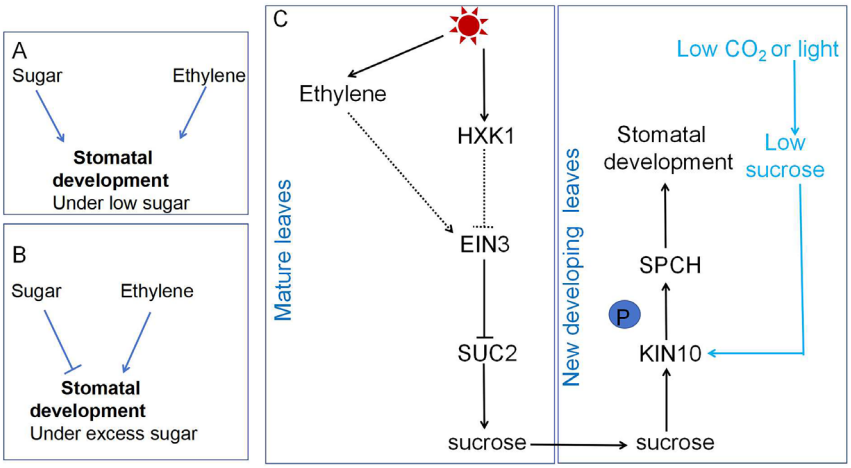

FIGURE 3 | Sugar antagonism of ethylene (ET) signalling occurs under excess sugar, whereas sugar synergy with ET signalling is manifested under low sugar availability. (A) ET and sugar signals synergistically regulate stomatal development under high/excess sugar conditions. (B) ET and sugar signals antagonistically modulate stomatal development under low sugar conditions. (C) ET and sugar signals synergistically regulate stomatal development by relative pathway. Figure A,B is the compendious interpretation of Figure C. Solid lines indicate direct regulation, whereas dotted lines indicate either indirect regulation or regulation in an unknown manner. Blunt arrows indicate negative regulation and pointed arrows indicate positive regulation.

et al. 2004; De Paepe et al. 2004). Data indicates that in the absence of exogenous sugars, seed germination/seedling development was not significantly different between the *aba2* mutant and wild-type seedlings (González-Guzmán et al. 2002). However, in the presence of applied exogenous sugars, seed germination/seedling development of the *aba2* mutant was promoted relative to wild-type seedlings (Beaudoin et al. 2000). This is because exogenous sugars induce ABA synthesis and this subsequently complicates hormone responses. Significantly, supplied exogenous ET or ABA levels were at least 100- to 1000-fold higher than endogenous levels found in plants. We noticed that the expression of the JA/ET-responsive antifungal defensin, *PDF1.2*, and the ABA-responsive, *RD22*, has a contrasting relationship between physiological and high ABA concentration conditions. The expression of these two genes was upregulated under physiological conditions and downregulated under high levels of applied ABA. However, the conclusions of Anderson and coauthors, who investigated the interaction of ABA and JA, based their conclusions only on conditions of high levels of exogenous ABA (Anderson et al. 2004). Thus, the physiological relevance of their conclusions should be carefully considered.

6 | Glucose Antagonism of ET Signalling Occurs Under Excess Glucose, Whereas Glucose Synergy With ET Signalling is Manifested Under Low Glucose Availability

To obtain a more obviously phenotype, excess plant hormones/sugars have been widely applied when studying the crosstalk between ET and other signalling cues. This appears to complicate plant hormone/sugar responses and may even trigger conclusions opposite to those that might have been obtained if more physiological concentrations had been used. Thus, low exogenous sucrose induces endogenous ET production and thus promotes stomatal development. In contrast, high exogenous sucrose inhibits endogenous ET production and thus inhibits stomatal development (Jeong et al. 2010; Han et al. 2020, 2022; Bao et al. 2023).

Under low glucose availability (1%–2%), there is a synergistic interaction between glucose and ET signalling, promoting plant growth and development in *Arabidopsis* (Jeong et al. 2010), rice (Philosoph-Hadas, Meir, and Aharoni 1985) and tobacco (Kobayashi and Saka 2000). However, glucose antagonism of ET signalling occurs under higher or excess glucose levels (such as 5% glucose) (Jang et al. 1997; Zhou et al. 1998; Moore et al. 2003; Cho, Sheen, and Yoo 2010; Karve, Xia, and Moore 2012). Furthermore, under low glucose availability, glucose signalling is uncoupled from ET signal transduction during early seedling development (Cho, Sheen, and Yoo 2010). Therefore, glucose synergy towards ET signalling is manifested under low glucose availability/physiological conditions (Philosoph-Hadas, Meir, and Aharoni 1985; Kobayashi and Saka 2000; Jeong et al. 2010), whereas glucose antagonism of ET signalling occurs under high glucose levels/stress conditions (Jang et al. 1997; Zhou et al. 1998; Moore et al. 2003; Cho, Sheen, and Yoo 2010; Karve, Xia, and Moore 2012). Thus, glucose synergy towards ET signalling positively regulates root hair growth under low exogenous sugar conditions (Karve, Xia, and Moore 2012; Feng et al. 2017).

Gong et al. (2021) indicated that under low glucose levels because sucrose/glucose inhibits stomatal development, ET signalling promotes stomatal development. This conclusion does not align with numerous papers (Jang et al. 1997; Zhou et al. 1998; Moore et al. 2003; Cho, Sheen, and Yoo 2010; Karve, Xia, and Moore 2012), that is, these reports conclude that glucose antagonism of ET signalling occurs under high glucose levels/stress conditions. This conclusion of Gong et al. (2021) is based on glucose antagonism of ET signalling under low glucose availability. Therefore, their conclusion is contradictory to a few reports, that is, low exogenous sucrose induces endogenous ET production and thus promotes stomatal development, whereas high exogenous sucrose inhibits endogenous ET production and thus inhibits stomatal development (Jeong et al. 2010; Han et al. 2020, 2022; Bao et al. 2023) (Figure 3A,B). Therefore, supplied exogenous sugar levels are pivotal in making conclusions.

Under typical CO_2/light conditions, generated glucose signalling is sensed via a known glucose sensor HXK1, thereby decreasing EIN3 stability, which in turn increases the accumulation of the key sucrose transporter, SUC2. A glucose-HXK1-EIN3-SUC2-sucrose module facilitates the transport of sucrose from mature leaves to newly developing leaves (Bao et al. 2023). Within newly developing leaves, transported sucrose promotes the stability of the protein kinase, KIN10. Subsequently, KIN10 positively regulates SPCH, the master regulator of stomatal development, which in turn promotes formation of stomata (Han et al. 2020). Therefore, under typical CO_2/light conditions, a sucrose-KIN10-SPCH pathway promotes stomatal development within newly developing leaves in response to transported sucrose (Figure 3C).

7 | Whether Synergistic or Antagonistic Actions Between ET and JA/ABA/Sugar Signalling May Be Determined by the Content Fluctuation of Their Respective Master Transcription Factors in These Pathways

To investigate relative molecular mechanism of the crosstalk between ET and JA/ABA/sugar signalling, further analysis is necessary. Glucose antagonism of ET signalling occurs under excess glucose, whereas glucose synergy with ET signalling is manifested under low glucose availability. These different results with different sugar levels are caused by EIN3 status. ET accumulation activates a core ET signalling component, EIN3, by its canonical signal transduction pathway (Cho, Yoo, and Sheen 2006; Binder et al. 2007). Excess glucose promotes EIN3 degradation (Yanagisawa, Yoo, and Sheen 2003), whereas low glucose enhances EIN3 stability (Bao et al. 2023). By contrast, EIN3 is degraded, in the absence of ET, by protein ubiquitination (Guo and Ecker 2003; Potuschak et al. 2003), whereas increased ET elevates EIN3 stabilisation (Yanagisawa, Yoo, and Sheen 2003). Excess sugars inhibit ET production, whereas low sugars promote ET production (Jeong et al. 2010; Bao et al. 2023). As a result, EIN3-mediated glucose antagonism of ET signalling is triggered under excess glucose, whereas EIN3-mediated glucose synergy with ET signalling is caused under low glucose availability.

Similar to glucose antagonism or glucose synergy of ET signalling, ET synergy with JA signalling occurs under physiological conditions, whereas ET antagonism of JA signalling transpires under stress conditions (Figure 1). For example, freezing tolerance is antagonistically regulated by ET or JA canonical signal transduction pathways (Shi et al. 2012; Hu et al. 2013; Figure 1E). This is because cold stress can induce excess JA accumulation (Hu et al. 2013), whereas cold stress can decline ET accumulation (Shi et al. 2012). JA contents fluctuated with treatment time under cold condition (4°C) (Hu et al. 2013), that is, the incipient cold treatment promoted JA production, whereas the late cold treatment reduced JA production. Thus, excess ET promotes EIN3 degradation (Cho, Yoo, and Sheen 2006; Binder et al. 2007), whereas it is likely that excess JA enhances EIN3 stabilisation. This needs to be confirmed by experiments. As a result, EIN3 mediated-freezing tolerance is antagonistically modulated through ET or JA canonical signal transduction pathways (Shi et al. 2012; Hu et al. 2013; Figure 1E). Both synergistic and antagonistic patterns of interaction between JA and ET would assist plant adaptation to fluctuating environments by the JA-SCF^{COI1}-JAZ-MYC-CYP79B3-Camalexin and ET-ETR1/CTR1-EIN2-ERF1-PDF1.2 modules (Shi et al. 2012; Hu et al. 2013; Song et al. 2014). Therefore, the content fluctuation of respective master transcription factors (such as the transcription factor EIN3) in these pathways has an important biological significance.

Similar to glucose antagonism or synergy of ET signalling, ET and ABA signals synergistically and negatively regulate root growth under high/excess ABA (but not high ACC/ET) conditions, whereas the two signals synergistically and positively modulate root growth under physiological conditions (Figure 2). Sugars induce ABA biosynthesis and thus complicate ET and ABA interactions (Cheng et al. 2009). Thus, glucose may have a similar regulatory pattern to ABA in the regulation of plant growth and development. Therefore, because excess glucose promotes EIN3 degradation (Yanagisawa, Yoo, and Sheen 2003) and low glucose enhances EIN3 stabilisation (Bao et al. 2023), whether ABA signalling has a similar regulatory pattern remains to be determined.

Together, synergistic or antagonistic actions between ET and JA/ABA/sugar signalling may be determined by their respective master transcription factors in these pathways (specifically, the EIN3 transcription factor).

8 | Crosstalk Between ET and JA/ABA/Sugar Signalling is Complex

Collectively, the main reasons for the complexity of the interactions between ET and JA/ABA/sugar signalling are as follows: (1) excess plant hormones/sugars are applied. Often in some published reports, there is no biological significance for the data obtained, specifically, to observe plant growth and development of normal physiological status (these data may only have biological significance under abiotic and biotic stress). This is because endogenous ET and ABA/sugar/JA levels (physiological levels) are at least 100- to 1000-fold lower. (2) The substrate in which plants are grown, the tissue analysed (whole seedlings or leaves) and the plant hormone/sugars concentrations used for either growing plants or performing treatments. Without a clear rationale and extensive controls, it is often difficult to integrate all the resulting experimental conclusions into a single model. This may be because researchers often do not make clear distinctions between physiological and stress conditions. (3) Different organs or developmental stages have different susceptibility to the same nutrient and hormone concentrations. (4) These findings suggest that the crosstalk between ET-JA/ABA/sugar signalling is very complicated and dependent on tissue/organs, biological processes, and plant species. Why plants have so different interplay remains to be determined. However, it is possible that multiple interplay manners allow plants to be more adapted to the altering environmental conditions with different growth and developmental stages. (5) With the progress of science and technology, some new knowledge breaks previous cognisance. Therefore, the faultiness of old cognisance is exposed,

for example, recent reports have revealed that ACC can function as a signal independent of ET (Mou et al. 2020; Li et al. 2020). Thus, that ACC substitutes for ET may be not suitable, and will lead to incorrect conclusions.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing is not applicable to this article as no new data were created or analysed.

References

Anderson, J. P., E. Badruzsaufari, P. M. Schenk, et al. 2004. "Antagonistic Interaction Between Abscisic Acid and Jasmonate-Ethylene Signaling Pathways Modulates Defense Gene Expression and Disease Resistance in Arabidopsis." *Plant Cell* 16: 3460–3479.

Bao, Q. X., X. R. Mu, C. Tong, et al. 2023. "Sugar Status in Preexisting Leaves Determines Systemic Stomatal Development Within Newly Developing Leaves." *Proceedings of the National Academy of Sciences of the United States of America* 120: e2302854120.

Beaudoin, N., C. Serizet, F. Gosti, and J. Giraudat. 2000. "Interactions Between Abscisic Acid and Ethylene Signaling Cascades." *Plant Cell* 12: 1103–1115.

Benschop, J. J., F. F. Millenaar, M. E. Smeets, M. van Zanten, L. A. Voesenek, and A. J. Peeters. 2007. "Abscisic Acid Antagonizes Ethylene-Induced Hyponastic Growth in *Arabidopsis*." *Plant Physiology* 143: 1013–1023.

Binder, B. M., J. M. Walker, J. M. Gagne, et al. 2007. "The *Arabidopsis* EIN3 Binding F-Box Proteins EBF1 and EBF2 Have Distinct but Overlapping Roles in Ethylene Signaling." *Plant Cell* 19: 509–523.

Chen, J., K. Sonobe, N. Ogawa, et al. 2013. "Inhibition of *Arabidopsis* Hypocotyl Elongation by Jasmonates Is Enhanced Under Red Light in Phytochrome B Dependent Manner." *Journal of Plant Research* 126: 161–168.

Chen, L., I. C. Dodd, W. J. Davies, and S. Wilkinson. 2013. "Ethylene Limits Abscisic Acid- Or Soil Drying-Induced Stomatal Closure in Aged Wheat Leaves." *Plant, Cell & Environment* 36: 1850–1859.

Cheng, W. H., M. H. Chiang, S. G. Hwang, and P. C. Lin. 2009. "Antagonism Between Abscisic Acid and Ethylene in *Arabidopsis* Acts in Parallel With the Reciprocal Regulation of Their Metabolism and Signaling Pathways." *Plant Molecular Biology* 71: 61–80.

Cho, Y. H., J. Sheen, and S. D. Yoo. 2010. "Low Glucose Uncouples hexokinase1-Dependent Sugar Signaling From Stress and Defense Hormone Abscisic Acid and C2H4 Responses in *Arabidopsis*." *Plant Physiology* 152: 1180–1182.

Cho, Y. H., S. D. Yoo, and J. Sheen. 2006. "Regulatory Functions of Nuclear hexokinase1 Complex in Glucose Signaling." *Cell* 127: 579–589.

De Paepe, A., M. Vuylsteke, P. Van Hummelen, M. Zabeau, and D. Van Der Straeten. 2004. "Transcriptional Profiling by cDNA-AFLP and Microarray Analysis Reveals Novel Insights Into the Early Response to Ethylene in *Arabidopsis*." *Plant Journal* 39: 537–559.

Desikan, R., K. Last, R. Harrett-Williams, et al. 2006. "Ethylene-Induced Stomatal Closure in *Arabidopsis* Occurs via AtrbohF-Mediated Hydrogen Peroxide Synthesis." *Plant Journal* 47: 907–916.

Feng, Y., P. Xu, B. Li, et al. 2017. "Ethylene Promotes Root Hair Growth Through Coordinated EIN3/EIL1 and RHD6/RSL1 Activity in *Arabidopsis*." *Proceedings of the National Academy of Sciences of the United States of America* 114: 13834–13839.

Ghassemian, M., E. Nambara, S. Cutler, H. Kawaide, Y. Kamiya, and P. McCourt. 2000. "Regulation of Abscisic Acid Signaling by the Ethylene Response Pathway in *Arabidopsis*." *Plant Cell* 12: 1117–1126.

Gong, Y., J. Alassimone, R. Varnau, N. Sharma, L. S. Cheung, and D. C. Bergmann. 2021. "Tuning Self-Renewal in the *Arabidopsis* Stomatal Lineage by Hormone and Nutrient Regulation of Asymmetric Cell Division." *eLife* 10: e63335.

González-Guzmán, M., N. Apostolova, J. M. Bellés, et al. 2002. "The Short-Chain Alcohol Dehydrogenase ABA2 Catalyzes the Conversion of Xanthoxin to Abscisic Aldehyde." *Plant Cell* 14: 1833–1846.

Guo, H., and J. R. Ecker. 2003. "Plant Responses to Ethylene Gas Are Mediated by SCF(EBF1/EBF2)-dependent Proteolysis of EIN3 Transcription Factor." *Cell* 115: 667–677.

Han, C., Y. Liu, W. Shi, et al. 2020. "KIN10 Promotes Stomatal Development Through Stabilization of the SPEECHLESS Transcription Factor." *Nature Communications* 11: 4214.

Han, C., Y. Qiao, L. Yao, et al. 2022. "TOR and SnRK1 Fine Tune SPEECHLESS Transcription and Protein Stability to Optimize Stomatal Development in Response to Exogenously Supplied Sugar." *New Phytologist* 234: 107–121.

Hu, Y., L. Jiang, F. Wang, and D. Yu. 2013. "Jasmonate Regulates the Inducer of Cbf Expression-C-Repeat Binding Factor/DRE Binding factor1 Cascade and Freezing Tolerance in *Arabidopsis*." *Plant Cell* 25: 2907–2924.

Huang, G., A. Kilic, M. Karady, et al. 2022. "Ethylene Inhibits Rice Root Elongation in Compacted Soil via ABA- and Auxin-Mediated Mechanisms." *Proceedings of the National Academy of Sciences of the United States of America* 119: e2201072119.

Jang, J. C., P. León, L. Zhou, and J. Sheen. 1997. "Hexokinase as a Sugar Sensor in Higher Plants." *Plant Cell* 9: 5–19.

Jeong, S. W., P. K. Das, S. C. Jeoung, et al. 2010. "Ethylene Suppression of Sugar-Induced Anthocyanin Pigmentation in *Arabidopsis*." *Plant Physiology* 154: 1514–1531.

Karve, A., X. Xia, and B. Moore. 2012. "*Arabidopsis* Hexokinase-Like1 and Hexokinase1 Form a Critical Node in Mediating Plant Glucose and Ethylene Responses." *Plant Physiology* 158: 1965–1975.

Kobayashi, H., and H. Saka. 2000. "Relationship Between Ethylene Evolution and Sucrose Content in Excised Leaf Blades of Rice." *Plant Production Science* 3: 398–403.

Li, D., E. Flores-Sandoval, U. Ahtesham, et al. 2020. "Ethylene-Independent Functions of the Ethylene Precursor ACC in *Marchantia polymorpha*." *Nature Plants* 6: 1335–1344.

Linkies, A., and G. Leubner-Metzger. 2012. "Beyond Gibberellins and Abscisic Acid: How Ethylene and Jasmonates Control Seed Germination." *Plant Cell Reports* 31: 253–270.

Linkies, A., K. Müller, K. Morris, et al. 2009. "Ethylene Interacts With Abscisic Acid to Regulate Endosperm Rupture During Germination: A Comparative Approach Using *Lepidium sativum* and *Arabidopsis thaliana*." *Plant Cell* 21: 3803–3822.

Ma, B., C. C. Yin, S. J. He, et al. 2014. "Ethylene-Induced Inhibition of Root Growth Requires Abscisic Acid Function in Rice (*Oryza sativa* L.) Seedlings." *PLoS Genetics* 10: e1004701.

Miersch, O., J. Neumerkel, M. Dippe, I. Stenzel, and C. Wasternack. 2008. "Hydroxylated Jasmonates Are Commonly Occurring Metabolites

of Jasmonic Acid and Contribute to a Partial Switch-Off in Jasmonate Signaling." *New Phytologist* 177: 114–127.

Moore, B., L. Zhou, F. Rolland, et al. 2003. "Role of the *Arabidopsis* Glucose Sensor HXK1 in Nutrient, Light, and Hormonal Signaling." *Science (New York, N.Y.)* 300: 332–336.

Mou, W., Y. T. Kao, E. Michard, et al. 2020. "Ethylene-Independent Signaling by the Ethylene Precursor ACC in *Arabidopsis* Ovular Pollen Tube Attraction." *Nature Communications* 11: 4082.

Mu, X. R., C. Tong, X. T. Fang, et al. 2022. "Feed-Back Loop Promotes Sucrose Accumulation in Cotyledons to Facilitate Sugar-Ethylene Signaling-Mediated, Etiolated-Seedling Greening." *Cell Reports* 38: 110529.

Ogawara, T., K. Higashi, H. Kamada, and H. Ezura. 2003. "Ethylene Advances the Transition From Vegetative Growth to Flowering in *Arabidopsis thaliana*." *Journal of Plant Physiology* 160: 1335–1340.

Overmyer, K., H. Tuominen, R. Kettunen, et al. 2000. "Ozone-Sensitive *Arabidopsis rcd1* Mutant Reveals Opposite Roles for Ethylene and Jasmonate Signaling Pathways in Regulating Superoxide-Dependent Cell Death." *Plant Cell* 12: 1849–1862.

Penninckx, I. A., B. P. Thomma, A. Buchala, J. P. Métraux, and W. F. Broekaert. 1998. "Concomitant Activation of Jasmonate and Ethylene Response Pathways Is Required for Induction of a Plant Defensin Gene in *Arabidopsis*." *Plant Cell* 10: 2103–2113.

Philosoph-Hadas, S., S. Meir, and N. Aharoni. 1985. "Carbohydrates Stimulate Ethylene Production in Tobacco Leaf Discs: II. Sites of Stimulation in the Ethylene Biosynthesis Pathway." *Plant Physiology* 78: 139–143.

Potuschak, T., E. Lechner, Y. Parmentier, et al. 2003. "EIN3-Dependent Regulation of Plant Ethylene Hormone Signaling by Two *Arabidopsis* F Box Proteins: EBF1 and EBF2." *Cell* 115: 679–689.

Qi, T., S. Song, Q. Ren, et al. 2011. "The Jasmonate-ZIM-Domain Proteins Interact With the WD-Repeat/bHLH/MYB Complexes to Regulate Jasmonate-Mediated Anthocyanin Accumulation and Trichome Initiation in *Arabidopsis thaliana*." *Plant Cell* 23: 1795–1814.

Qin, H., J. Wang, J. Zhou, et al. 2023. "Abscisic Acid Promotes Auxin Biosynthesis to Inhibit Primary Root Elongation in Rice." *Plant Physiology* 191: 1953–1967.

Rao, M. V., H. Lee, R. A. Creelman, J. E. Mullet, and K. R. Davis. 2000. "Jasmonic Acid Signaling Modulates Ozone-Induced Hypersensitive Cell Death." *Plant Cell* 12: 1633–1646.

Robson, F., H. Okamoto, E. Patrick, et al. 2010. "Jasmonate and Phytochrome A Signaling in *Arabidopsis* Wound and Shade Responses Are Integrated Through JAZ1 Stability." *Plant Cell* 22: 1143–1160.

Seo, M., A. Hanada, A. Kuwahara, et al. 2006. "Regulation of Hormone Metabolism in Arabidopsis Seeds: Phytochrome Regulation of Abscisic Acid Metabolism and Abscisic Acid Regulation of Gibberellin Metabolism." *Plant Journal* 48: 354–366.

Shi, Y., S. Tian, L. Hou, et al. 2012. "Ethylene Signaling Negatively Regulates Freezing Tolerance by Repressing Expression of CBF and Type-A ARR Genes in *Arabidopsis*." *Plant Cell* 24: 2578–2595.

Song, S., H. Huang, H. Gao, et al. 2014. "Interaction Between MYC2 and ETHYLENE INSENSITIVE3 Modulates Antagonism Between Jasmonate and Ethylene Signaling in *Arabidopsis*." *Plant Cell* 26: 263–279.

Tamari, G., A. Borochov, R. Atzorn, and D. Weiss. 1995. "Methyl Jasmonate Induces Pigmentation and Flavonoid Gene Expression in *Petunia* Corollas: A Possible Role in Wound Response." *Physiologia Plantarum* 94: 45–50.

Tanaka, Y., T. Sano, M. Tamaoki, N. Nakajima, N. Kondo, and S. Hasezawa. 2005. "Ethylene Inhibits Abscisic Acid-Induced Stomatal Closure in *Arabidopsis*." *Plant Physiology* 138: 2337–2343.

Thomma, B. P., K. Eggermont, I. A. Penninckx, et al. 1998. "Separate Jasmonate-Dependent and Salicylate-Dependent Defense-Response Pathways in *Arabidopsis* Are Essential for Resistance to Distinct Microbial Pathogens." *Proceedings of the National Academy of Sciences of the United States of America* 95: 15107–15111.

Thomma, B. P., K. Eggermont, K. F. Tierens, and W. F. Broekaert. 1999. "Requirement of Functional Ethylene-Insensitive 2 Gene for Efficient Resistance of *Arabidopsis* to Infection by *Botrytis cinerea*." *Plant Physiology* 121: 1093–1102.

Tong, C., C. Li, X. Y. Cao, et al. 2022. "Long-Distance Transport of Sucrose in Source Leaves Promotes Sink Root Growth by the EIN3-SUC2 Module." *PLoS Genetics* 18, no. 9: e1010424.

Tuominen, H., K. Overmyer, M. Keinänen, H. Kollist, and J. Kangasjärvi. 2004. "Mutual Antagonism of Ethylene and Jasmonic Acid Regulates Ozone-Induced Spreading Cell Death in *Arabidopsis*." *Plant Journal* 39: 59–69.

Wang, Y., C. Liu, K. Li, et al. 2007. "*Arabidopsis* EIN2 Modulates Stress Response Through Abscisic Acid Response Pathway." *Plant Molecular Biology* 64: 633–644.

Yanagisawa, S., S. D. Yoo, and J. Sheen. 2003. "Differential Regulation of EIN3 Stability by Glucose and Ethylene Signalling in Plants." *Nature* 425: 521–525.

Yang, J., J. Zhang, Z. Wang, K. Liu, and P. Wang. 2006. "Post-Anthesis Development of Inferior and Superior Spikelets in Rice in Relation to Abscisic Acid and Ethylene." *Journal of Experimental Botany* 57: 149–160.

Yoo, S. D., Y. Cho, and J. Sheen. 2009. "Emerging Connections in the Ethylene Signaling Network." *Trends in Plant Science* 14: 270–279.

Zhao, Q., and H. W. Guo. 2011. "Paradigms and Paradox in the Ethylene Signaling Pathway and Interaction Network." *Molecular Plant* 4: 626–634.

Zhong, S., H. Shi, C. Xue, et al. 2012. "A Molecular Framework of Light-Controlled Phytohormone Action in *Arabidopsis*." *Current Biology* 22: 1530–1535.

Zhou, L., J. C. Jang, T. L. Jones, and J. Sheen. 1998. "Glucose and Ethylene Signal Transduction Crosstalk Revealed by an *Arabidopsis* Glucose-Insensitive Mutant." *Proceedings of the National Academy of Sciences of the United States of America* 95: 10294–10299.

Zhou, Y., B. Ma, J. J. Tao, et al. 2022. "Rice EIL1 Interacts With OsIAAs to Regulate Auxin Biosynthesis Mediated by the Tryptophan Aminotransferase MHZ10/OsTAR2 During Root Ethylene Responses." *Plant Cell* 34: 4366–4387.

Zhu, C., L. Gan, Z. Shen, and K. Xia. 2006. "Interactions Between Jasmonates and Ethylene in the Regulation of Root Hair Development in *Arabidopsis*." *Journal of Experimental Botany* 57: 1299–1308.