Mini Review

Friends or Foes: New Insights in Jasmonate and Ethylene Co-Actions

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One strategy for sessile plants to adapt to their surrounding environment involves the modulation of their various internal phytohormone signaling and distributions when the plants sense environmental change. There are currently dozens of identified phytohormones in plant cells and they act in concert to regulate plant growth, development, metabolism and defense. It has been determined that phytohormones often act together to achieve certain physiological functions. Thus, the study of hormone–hormone interactions is becoming a competitive research field for deciphering the underlying regulatory mechanisms. Among phytohormones, jasmonate and ethylene present a fascinating case of synergism and antagonism. They are commonly recognized as defense hormones that act synergistically. Plants impaired in jasmonate and/or ethylene signaling are susceptible to infections by necrotrophic fungi, suggesting that these two hormones are both required for defense. Moreover, jasmonate and ethylene also act antagonistically, such as in the regulation of apical hook development and wounding responses. Here, we highlight the recent breakthroughs in the understanding of jasmonate–ethylene co-actions and point out the potential power of studying protein–protein interactions for systematically exploring signal cross-talk.

Keywords: EIN3 • Ethylene • Jasmonate • MYC2 • Protein–protein interactions.

Abbreviations: bHLH, basic helix–loop–helix; CEND, C-terminal end; CTR1, CONSTITUTIVE TRIPLE RESPONSE 1; COI1, CORONATINE INSENSITIVE 1; EBF1, ETHYLENE BINDING FACTOR 1; EBF2, ETHYLENE BINDING FACTOR 2; EIL1, ETHYLENE INSENSITIVE 3 LIKE 1; EIN2, ETHYLENE INSENSITIVE 2; EIN3, ETHYLENE INSENSITIVE 3; ERF1, ETHYLENE RESPONSE FACTOR 1; GAI, GIBBERELLIC ACID INSENSITIVE; HDA6, HISTONE DEACETYLASE 6; HLS1, HOOKLESS 1; JAR1, JASMONATE RESISTANT 1; JAZ protein, JASMONATE ZIM-DOMAIN protein; NINJA, NOVEL INTERACTOR of JAZ; ORA59, OCTADECANOID-RESPONSIVE ARABIDOPSIS AP2/ERF 59; PIF3, PHYTOCHROME INTERACTING FACTOR3; PPI, protein–protein interaction; RGA: REPRESSOR OF GA1-3; SCF, Skip1–Cullin–F-box.

Introduction

Jasmonate and ethylene are two essential plant hormones. Both regulate a variety of growth and developmental events, such as seed germination, hypocotyl elongation, root elongation, root hair development, fertility and flowering time (Browse 2005, Zhu and Guo 2008). Besides these growth-related events, jasmonate and ethylene are both required for plant resistance to infections by necrotrophic fungi (Dong 1998). It has been shown that jasmonate and ethylene treatment synergistically and interdependently induce pathogen-responsive gene expression. Plants impaired in jasmonate and/or ethylene signaling fail to induce pathogen-responsive gene expression and are much more susceptible to fungal infections (Penninckx et al. 1998, Lorenzo et al. 2003). Jasmonate and ethylene are hence recognized as defense hormones for plant survival. In addition, jasmonate antagonizes ethylene’s effect in the promotion of the apical hook of etiolated seedling (Turner et al. 2002), while ethylene antagonizes jasmonate-dependent plant wounding responses (Rojo et al. 1999, Bodenhausen and Reymond 2007). Although these phenomena have been described more than one decade ago, their underlying molecular mechanisms were not made clear until very recently with the progress in the characterization of jasmonate and ethylene signal transduction pathways.

Jasmonate Signaling

After synthesis, jasmonate is conjugated with l-isoleucine by JASMONATE RESISTANT 1 (JAR1) to form the bioactive type of jasmonate, JA-Ile (Staswick and Tiryaki 2004, Suza and Staswick 2008, Fonseca et al. 2009). The Cyt P450 family protein CYP94B3 converts JA-Ile into 12OH-JA-Ile by 12-hydroxylation to inactivate JA-Ile (Kitaoka et al. 201, Koo et al. 2011, Heitz et al. 2012). These reversible steps maintain adequate concentrations of active jasmonate in plant cells. Coronatine is a virulence effector, secreted by the hemibiotrophic pathogen Pseudomonas syringae, which shares high similarity in chemical structure with JA-Ile (Bender et al. 1999). Coronatine treatment also inhibits plant root elongation and induces pathogen-responsive gene expression, mimicking the effects of JA-Ile.
Screening coronatine-insensitive mutants by forward genetic approaches reveals that the CORONATINE INSENSITIVE 1 (COI1) gene is required for almost all the coronatine- or JA-triggered responses (Xie et al. 1998). COI1 encodes an F-box protein, which interacts with ASK1 to assemble a functional Skip1–Cullin–F-box (SCF) type E3 ubiquitin ligase (SCFCOI1) (Dai et al. 2002, Devoto 2002, Xu et al. 2002). JASMONATE ZIM-DOMAIN (JAZ) proteins are the long sought target proteins of COI1, which are degraded in the presence of jasmonate (Chini et al. 2007, Thines et al. 2007). The interactions between COI1 and JAZ proteins are JA-ile or coronatine dependent (Katsir et al. 2008, Melotto et al. 2008). Crystallography studies reveal that JA-ile acts like ‘molecular glue’, which promotes COI1–JAZ interaction and further JAZ protein degradation (Sheard et al. 2010). COI1–JAZ is therefore recognized as a co-receptor for JA perception.

JAZ proteins are transcriptional repressors, which interact with several branches of transcription factors (Table 1) and inhibit their transcriptional activities (Song et al. 2014b). The basic helix–loop–helix (bHLH) transcription factor MYC2 is the first identified JAZ protein-interacting transcription factor in jasmonate signaling. MYC2 predominantly mediates jasmonate-controlled root growth inhibition and wounding responses for plant resistance to herbivores (Dombrecht et al. 2007). Once JAZ proteins are degraded, the repressions are released and these JAZ-interacting transcription factors are activated to elicit various jasmonate-related responses (Fig. 1A). Interestingly, it seems that each individual group of transcription factors governs specific physiological functions as listed in Table 1. Currently there are three reported mechanisms that explain JAZ protein repression: (i) JAZ proteins interact with the adaptor protein NOVEL INTERACTOR OF JAZ (NINJA) as a co-repressor, which further recruits the Groucho/Tup1-type repressor TOPLESS to fulfill transcriptional repression (Pauwels et al. 2010); (ii) JAZ8 directly interacts with TOPLESS through the EAR motif in JAZ8 (Shyu et al. 2012); and (iii) several JAZ proteins (such as JAZ1/3/9) interact with HISTONE DEACETYLASE 6 (HDA6) to de-acetylate histone for transcriptional repression (Zhu et al. 2011). Because TOPLESS triggered repression is primarily correlated to histone deacetylation (Long et al. 2006) and JAZ proteins tend to form homodimers and/or heterodimers through mutual interactions (Chini et al. 2009), these three mechanisms are probably coupled together to enforce repressions.

Taken together, the core of jasmonate signaling is the derepression mechanism. Without jasmonate, JAZ proteins recruit co-repressor proteins to inhibit their interacting transcription factors. After jasmonate perception, SCFCOI1 promotes the turnover of JAZ proteins to activate various downstream transcription factors to elicit adequate jasmonate responses (Fig. 1A).

### Table 1 JAZ-interacting DNA-binding transcription factors in Arabidopsis

<table>
<thead>
<tr>
<th>Transcription factor</th>
<th>Physiological functions</th>
<th>Reference</th>
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<tbody>
<tr>
<td>MYC2/MYC3/MYC4</td>
<td>Root elongation, wounding responses, defense, metabolism,</td>
<td>Chini et al. (2007); Thines et al. (2007);</td>
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<td></td>
<td>hook development</td>
<td>Cheng et al. (2011); Fernandez-Calvo et al.</td>
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<td>(2011); Niu et al. (2011)</td>
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<td>MYB21/MYB24</td>
<td>Stamen development</td>
<td>Song et al. (2011)</td>
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<td>TT8, GL3, EGL3, MYB7S</td>
<td>Anthocyanin synthesis and trichome development</td>
<td>Qi et al. (2011)</td>
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<td>and GL1</td>
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<tr>
<td>EIN3/EL1</td>
<td>Defense, root elongation, hook and root hair development</td>
<td>Zhu et al. (2011)</td>
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<tr>
<td>ICE1/ICE2</td>
<td>Freezing response</td>
<td>Hu et al. (2013)</td>
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<tr>
<td>WRKY57</td>
<td>Leaf senescence</td>
<td>Jiang et al. (2014)</td>
</tr>
<tr>
<td>bHLH003/bHLH013/bHLH014/bHLH017</td>
<td>Root elongation, defense, fertility, anthocyanin synthesis</td>
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**Fig. 1** Illustration of jasmonate–ethylene co-actions. (A) A simplified illustration of jasmonate signaling. JAZ proteins (JAZs) are central repressors in jasmonate signaling, and interact with various transcription factors (TFs) to inhibit their functions. After jasmonate production, it promotes the interaction between JAZs and COI1 (an F-box protein). COI1 causes the degradation of JAZs to relieve their repressions on TFs and activate various jasmonate responses. (B) Jasmonate–ethylene co-action model. Ethylene-activated transcription factor EIN3/EIL1 (EIN3 stands for EIN3/EIL1 in the model) are also activated by jasmonate. They integrate both ethylene and jasmonate signaling in the co-operative control of plant defense. Another jasmonate-activated transcription factor MYC2 interacts with EIN3 and inhibits EIN3 function, while EIN3 also represses MYC2 functions in the regulation of wounding responses. Moreover, MYC2 directly induces the expression of EBF1, which further targets EIN3 for degradation to provide an additional layer of repression on EIN3.
Ethylene is a simple gaseous hormone. Inhibition of ethylene production is commercially used to extend fruit or flower shelf time. Ethylene is also a necessary hormone for regulating plant growth, development and defense (Kieber and Ecker 1993). One characteristic ethylene response is the so-called ‘triple response’ where etiolated seedlings grown under ethylene treatment exhibit short hypocotyls, short roots and an exaggerated apical hook (Guzman and Ecker 1990). These easily observed phenotypes greatly facilitate the identification of ethylene signaling components through forward genetic approaches for identifying ethylene-insensitive mutants or constitutive ethylene-responsive mutants. Ethylene binds to endoplasmic reticulum-localized ethylene receptors and further inhibits their interacting Raf-like kinase CONSTITUTIVE TRIPLE RESPONSE 1 (CTR1) (Chang et al. 1993, Kieber et al. 1993, Hua and Meyerowitz, 1998, Chen et al. 2002). In the absence of ethylene, CTR1 directly phosphorylates another endoplasmic reticulum-localized transmembrane protein ETHYLENE INSENSITIVE 2 (EIN2) at its C-terminal end (CEND) (Ju et al. 2012). When CTR1 is deactivated, EIN2 is dephosphorylated. EIN2 is a positive regulator in ethylene signaling, shares sequence similarity with NRAMP family transmembrane proteins at its N-terminus and has an approximately 800 amino acid CEND (Alonso et al. 1999). It has been recently reported that when the CEND is dephosphorylated, it can be cleaved and transported to the nucleus (Qiao et al. 2012, Wen et al. 2012). In the nucleus, there are two transcription factors, ETHYLENE INSENSITIVE 3 (EIN3) and EIN3 LIKE 1 (EIL1), which control almost all the ethylene responses (Solano et al. 1998).

Without ethylene, two F-box proteins EIN3-BINDING F-BOX PROTEIN 1 (EBF1) and EBF2 interact with EIN3/EIL1 and trigger EIN3/EIL1 degradation (Guo and Ecker 2003, Potuschak et al. 2003, Cagne et al. 2004). Ethylene treatment promotes EBF1/2 turnover to maintain EIN3/EIL1 protein accumulation for stimulation of downstream ethylene responses (An et al. 2010). It is speculated that the nuclear-localized EIN2 CEND somehow inhibits EBF1/2 and stimulates EIN3/EIL1 accumulation (Ji and Guo 2013).

Jasmonate–Ethylene Synergistic Interactions

Both jasmonate and ethylene are required for plant resistance against infections by necrotrophic fungi. Jasmonate and ethylene treatment induce pathogen-responsive gene expression, and those gene-encoded products enhance plant tolerance against infections. The primary signaling factors, ETHYLENE RESPONSE FACTOR 1 (ERF1) and OCTADE CANOID-RESPONSIVE ARABIDOPSIS AP2/ERF59 (ORA59), are necessary for eliciting the downstream pathogen-responsive gene expression. Jasmonate and/or ethylene treatment induces the expression of both ERF1 and ORA59 (Lorenzo et al. 2003, Pre et al. 2008, Zarei et al. 2011); however, jasmonate and/or ethylene treatment could not induce their expression in either the coi1 or ein2 mutant background (Lorenzo et al. 2003, Pre et al. 2008). These observations imply that jasmonate and ethylene act in both synergistic and interdependent manners. Genetic analysis suggests that ERF1 and ORA59 could be the node for integrating jasmonate and ethylene signaling; however, the demonstration of a direct biochemical interaction with the established hormone signaling molecules is currently lacking. Because ERF1 is one of the direct target genes for EIN3 (Solano et al. 1998), it is thus speculated that EIN3 is the direct molecular link for jasmonate–ethylene synergistic interactions.

It was recently shown that JAZ proteins could physically interact with and repress EIN3 and EIL1, suggesting that EIN3/EIL1 belong to a new branch of JAZ-targeted transcription factors. Genetic analysis indicates that EIN3/EIL1 are required for some jasmonate responses, including root hair development, pathogen-responsive gene expression and root growth inhibition. Further studies revealed that JAZ proteins recruit HDA6 as a co-repressor for inhibiting EIN3/EIL1 functions. It was next shown that jasmonate treatment reduces the interaction between HDA6 and EIN3/EIL1 and enhances EIN3/EIL1 transcriptional activities (Zhu et al. 2011). The identification of EIN3/EIL1 as new JAZ target proteins demonstrates that EIN3/EIL1 are the direct molecular links for jasmonate–ethylene co-actions in modulating plant defense responses. Jasmonate treatment activates EIN3/EIL1 through the de-repression mechanism, while ethylene stimulates EIN3/EIL1 protein accumulation for their activation. These distinctive mechanisms work together to activate EIN3/EIL1 and trigger their downstream cascade to induce pathogen-responsive gene expression.

Jasmonate–Ethylene Antagonistic Interactions

In addition to the co-operative actions of jasmonate and ethylene, these hormones also act in antagonistic ways. JA inhibits ethylene-stimulated exaggerated apical hook formation, whereas ethylene suppresses jasmonate-mediated plant wounding responses (Rojo et al. 1999, Turner et al. 2002, Bodenhausen and Reymond 2007).

Two recent studies found that the molecular mechanisms for jasmonate–ethylene antagonistic interactions are based on the direct mutual inhibitions between two branches of JA-activated transcription factors (MYC2 and EIN3/EIL1). Ethylene promotes exaggerated hook formation through the induction of HOOKLESS 1 (HLS1) expression, which encodes a protein of unknown function that shares certain sequence similarity with N-acetyltransferase family proteins (Lehman et al. 1996). HLS1 loss-of-function mutants are impaired in exhibiting exaggerated apical hook under ethylene treatment, suggesting that HLS1 is a positive regulator for hook formation. It has been recently shown that jasmonate treatment reverses the induction effect of ethylene on HLS1 expression (Song et al. 2014a, Zhang et al. 2014), and HLS1 is a direct target gene for EIN3 (An et al. 2012, Chang et al. 2013). These results imply that the opposing regulation of EIN3 activity is key to the antagonistic physiological functions of these two hormones. There are currently two separate mechanisms for explaining how jasmonate suppresses ethylene’s effects: (i) jasmonate-activated transcription factor MYC2 directly binds to the promoter of EBF1 and...
induces EBF1 expression, which further triggers EIN3/EIL1 protein degradation to inhibit their activities (Zhang et al. 2014); and (ii) besides this layer of post-transcriptional regulation, MYC2 is able to interact physically with EIN3/EIL1 proteins and suppress their transcriptional activities (Song et al. 2014a, Zhang et al. 2014). Protein–DNA interaction experiments show that MYC2 directly abrogates the association between EIN3 and the HLS1 promoter (Zhang et al. 2014). Meanwhile, it has been observed that the physical interaction between MYC2 and EIN3/EIL1 also inhibits MYC2 activities, particularly for the MYC2-triggered wound responses. Taken together, these mutual repressions between MYC2 and EIN3/EIL1 cause the antagonism in jasmonate and ethylene signaling (Fig. 1B).

From a biological standpoint, this antagonism between jasmonate and ethylene signaling provides a fine-tuning mechanism that regulates both hormone signaling pathways.

Perspectives: Lessons from Jasmonate–Ethylene Co-Actions

It can be concluded that the co-operative or antagonistic interactions between jasmonate and ethylene are largely dependent on the interaction status of their downstream signaling components. When ethylene-activated transcription factors EIN3/EIL1 are trapped by the JAZ–HDA6 repressors, EIN3/EIL1 transcriptional activities are inhibited. After jasmonate-triggered JAZ protein degradation, JAZ–HDA6 proteins will no longer interact with EIN3/EIL1, allowing EIN3/EIL1 to induce their downstream transcriptional cascade. However, jasmonate treatment also activates another transcription factor, MYC2, through a similar derepression mechanism. Activated MYC2 interacts with EIN3/EIL1 and inhibits EIN3/EIL1 functions by abrogating their DNA binding abilities (Fig. 1B). Thus, the fate of EIN3/EIL1 is switched from activation to deactivation. In other words, the function of major transcription factors in one signaling pathway can be affected through physical interaction with other signaling proteins. This provides a possible entry for signal integration of different hormones. Nevertheless, the interaction frameworks among different proteins are more complicated than previously thought due to at least three major layers of complexity. To simplify the framework, we omit other jasmonate and ethylene signaling components and only include the above-discussed JAZ, EIN3 and MYC2 in the following case studies.

First, each protein has more than one interaction partner and exhibits mutual interactions to form a network (Fig. 2). For example, not only do JAZ proteins interact with MYC2 and EIN3, but also some JAZ proteins interact with DELLA proteins (Hou et al. 2010, Yang et al. 2012). In addition, MYC2 and EIN3 also interact with DELLA in the regulation of sesquiterpene synthesis and hook development (An et al. 2012, Hong et al. 2012). DELLA proteins are a family of transcriptional repressors in gibberellin signaling and are degraded upon gibberellin application. The degradation of DELLA causes the activation of DELLA-interacting transcription factors, such as PHYTOCHROME INTERACTING FACTOR3 (PIF3) and PIF4, to promote hypocotyl elongation (Sun 2008). The interactions between DELLA and JAZ mutually inhibit their repression activity and contribute to the jasmonate and gibberellin signaling interactions (Fig. 2).

Secondly, each protein family contains multiple members and these individual members form homodimers and heterodimers. EIN3/EIL1 have two members, whereas the JAZ protein family has 12 members (JAZ1–JAZ12) (Chini et al. 2007, Thines et al. 2007); MYC2 has two close homologs MYC3 and MYC4 (Cheng et al. 2011, Fernandez-Calvo et al. 2011, Niu et al. 2011), whereas the DELLA family contains five members [GIBBERELLIC ACID INSUSITIVE (GAI), REPRESSOR OF GA1-3 (RGA), RGA-LIKE 1 (RGL1), RGL2 and RGL3] (Sun 2008). It has been reported that JAZ proteins mutually interact with themselves to form homodimers and or heterodimers (Chini et al. 2009). EIN3 and EIL1 tend to form homodimers to facilitate their association with the palindromic DNA repeats located in the EIN3-binding sites (Solano et al. 1998). Likewise, MYC2/MYC3/MYC4 also form homodimers and heterodimers among themselves (Fernandez-Calvo et al. 2011). The dimerization capacity among these four categories of proteins (JAZ, EIN3, MYC2 and DELLA) makes their collective protein–protein interactions (PPIs) much more complicated. For instance, JAZ1 can interact with other JAZ family members along with multiple MYC2, EIN3 and DELLA partners. It has been experimentally demonstrated that JAZ1 resides in a large protein complex of more than one MegaDalton (Geerink et al. 2010), supporting that many PPIs exist. Since protein functions are highly correlated with their interaction status, it will be intriguing to characterize systematically each individual PPI pair under certain treatments to determine its underlying biological significance.

Thirdly, their native protein abundances are modulated by hormone treatment. As we described before, ethylene promotes EIN3 protein accumulation, while jasmonate accelerates EIN3 degradation. The abundances of JAZ and DELLA repressors are negatively regulated by jasmonate and gibberellin, respectively. In addition, it was recently reported that MYC2/MYC3/MYC4 are stabilized by jasmonate (Chico et al. 2014). Changes in protein abundance will certainly disturb their PPI statuses because the mole ratio between two proteins in one
A PPI pair will be changed. In other words, this simplified interaction network will be perturbed under certain hormone treatments because of changes in protein abundance (Fig. 2). It has been reported that the interaction between JAZ and MYC2 can be enhanced by gibberellin treatment. Gibberellin promotes DELLA degradation to weaken DELLÀ–JAZ interactions, thereby enhancing JAZ–MYC2 interactions (Hou et al. 2010).

The actual in vivo situation is much more complicated than suggested by the three above-mentioned layers of complexity. For instance, there is no doubt that dynamic protein compartmentation and modification will affect PPI. Thus, it will be challenging to estimate the interaction status of each component under certain treatment.

Though we only discuss the interactions of four protein families, it has recently been revealed that the actual PPI network is much more complicated at the Arabidopsis proteome level (Arabidopsis Interactome Mapping Consortium 2011, Jones et al. 2014). The Arabidopsis research community has already discovered plenty of PPI information and the many interaction possibilities among most Arabidopsis proteins. Moving forward, we believe that the next step will be to measure PPI strength quantitatively one by one with hormone treatments and determine the impact of PPI on each interaction partner. Taking EIN3 for example, we know that EIN3 is able to interact with JAZ, MYC2 and DELLÀ at the very least. However, under certain treatments, such as jasmonate plus gibberellin, what are the consequences? How do the interactions change? How is EIN3–DNA association affected? Although PPI strength used to be quantitatively measured in a yeast two-hybrid (Y2H) system, Y2H is not an ideal system for studying PPI under certain hormone treatments, especially in the presence of multiple native Arabidopsis proteins that will result in competitive interactions. The split luciferase system is better for this quantitative purpose because it is a sensitive in vivo analysis and the interaction results are easily quantified with a conventional luminometer machine (Chen et al. 2008). Moreover, given that the majority of the Arabidopsis genes are cloned and that the protoplast-based transient expression system is widely adopted (Yoo et al. 2007, Arabidopsis Interactome Mapping Consortium 2011), it is feasible to measure PPI strength under certain hormone treatments at a high throughput scale using the split luciferase system. This kind of study can provide more information if we include mutants or overexpression lines within protoplast preparations to see the impact on PPI in the absence or presence of additional proteins. With large sets of quantitative experimental data, there is no doubt that computational simulation can be applied to predict PPI intensity and even protein–DNA association through calculating individual association/dissociation ratios.

Concluding Remarks

In the new era of biological studies, systems biology and quantitative biology, supported by cutting edge omics technologies, are progressively becoming new directions for understanding the secrets of life. The studies of jasmonate and ethylene co-actions shed new light on the studies of hormone–hormone interactions. Although the concept is not completely novel, it again demonstrates the power of PPIs in integrating divergent signals. Without a doubt, the Arabidopsis research community will uncover more and more exciting results on signaling interaction studies, particularly with the help of systems biology approaches, to dissect the integration mechanisms through the top-down investigation of PPI dynamics.

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Disclosures

The authors have no conflicts of interest to declare.

References


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