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Article Addendum

Auxin Homeostasis in Plant Stress Adaptation Response

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Addendum to:

GH3-Mediated Auxin Homeostasis Links Growth Regulation with Stress Adaptation Response in Arabidopsis

Park JE, Park JY, Kim YS, Staswick PE, Jeon J, Yun J, Kim SY, Kim J, Lee YH, Park CM

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ABSTRACT

Auxin plays a wide range of regulatory roles in diverse aspects of plant growth and developmental processes through a complex network of signaling interactions. In the May issue of *Journal of Biological Chemistry*, we have demonstrated that auxin homeostasis directly links growth regulation with stress adaptation responses through interactions with salicylic acid (SA) and abscisic acid (ABA) signals. In this signaling network, the endogenous auxin content is coordinately regulated through negative feedback by a group of auxin-inducible *GH3* genes that encode auxin-conjugating enzymes. The Arabidopsis mutant *wes1-D* overexpressing a *GH3* gene *WES1* exhibits typical auxin-deficient traits, such as reduced growth and leaf curling, but is resistant to both biotic and abiotic stresses. In addition, various stress-regulated genes, including pathogenesis-related protein genes (*PR*s) and C-repeat/dehydration responsive element binding factor genes (*CBF*s), are up-regulated in the mutant. Consistent with these observations, *WES1* is activated by pathogenic infections and abiotic stresses as well as by exogenous SA and ABA. We therefore propose that the *WES1*-mediated growth suppression would underlie the commonly observed symptoms of infected or stressed plants and provide a mechanism for auxin action in the fitness costs of induced resistance in plants.

Numerous genes and signaling pathways have been characterized and the roles of several growth hormones, including SA, ABA, and jasmonic acid (JA), have been extensively studied in stress responses in plants. However, the underlying molecular mechanisms are largely unknown, primarily because of the complex interactions between multiple signaling pathways.[1-4]

Notably, it has recently been reported that auxin, which is widely recognized as a growth regulator in plants, also participates in stress responses.[5-7] Frequently observed symptoms in infected or stressed plants are growth reduction and altered metabolism. In addition, auxin-regulated genes are affected in plants infected with pathogens.[5,6] These observations suggest that auxin is involved in adaptive responses to biotic and abiotic stresses.

Auxin function is manifested, at least in part, at the transcriptional level by regulating a group of primary responsive genes, including *Aux/IAAs*, *GH3s*, and small auxin-up RNAs (*SAURs*).[8] Particularly, all *GH3*-overexpressing mutants characterized so far, such as *dfl1-D*,[9] *dfl2-D*,[10] *ydk1-D*[11] and *wes1-D*,[12] exhibit severely reduced growth. This phenotype is well consistent with the biochemical activities of the GH3 proteins. The GH3 enzymes conjugate indole-3-acetic acid (IAA) to amino acids.[13,14] We have recently demonstrated that the GH3-mediated growth regulation is closely linked to stress adaptation responses.[12] The *wes1-D* mutant exhibits enhanced resistance to both biotic and abiotic stresses, and the *PR* and *CBF* genes are up-regulated in the mutant, strongly supporting that the WES1-mediated growth reduction is intimately associated with adaptive responses to environmental stresses.

Another feature of the *WES1* gene is its light responsiveness. *WES1* is induced by end-of-day far-red (EOD-FR) light, suggesting that *WES1* may be regulated by the light-stable phytochrome B.[15] The *wes1-D* mutant is phenotypically similar to *axr2-1*, an auxin-resistant mutant with a mutation in domain II of IAA7. However, whereas *axr2-1* exhibits short hypocotyls in both light and darkness, the dwarfed phenotype of *wes1-D* occurs only in the light (Park et al., in preparation). When grown in darkness or under far-red light, the *wes1-D* hypocotyls were comparable to control hypocotyls. However, they were significantly shorter under red or blue light, suggesting a photomorphogenic role for *WES1*. In addition, all Arabidopsis *GH3* genes characterized so far are responsive to light of particular wavelengths,[9-11] suggesting that they are also involved in plant photomorphogenesis.

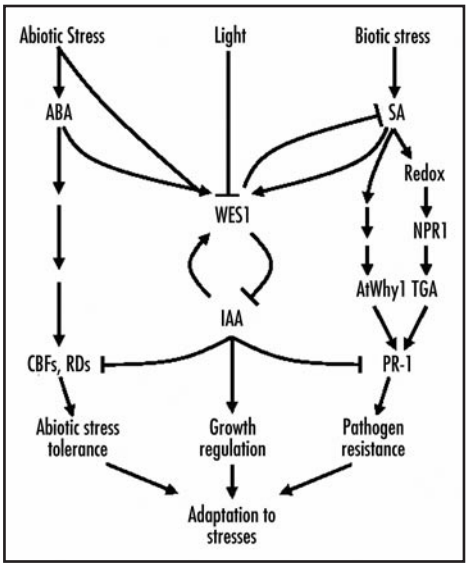

Figure 1. A proposed working model for WES1 function in stress adaptation responses. The endogenous auxin content is regulated by the WES1 enzyme through feedback regulation. *WES1* is upregulated by environmental stresses as well as by SA and ABA, causing reduction of endogenous IAA and resultant growth retardation under stress conditions. It seems that light signals are also incorporated into the *WES1*-mediated signaling pathways, which may provide an adaptive advantage on stressed plants.

It is well known that phytochrome-mediated light signals are essential for *PR* induction and hypersensitive reaction (HR).[16-18] These observations indicate that light also plays a critical role in SA-mediated disease resistance. This may be the reason why pathogen infection experiments are routinely carried out in darkness or in dim light. The *WES1*-mediated light signal is likely to be incorporated into the SA and ABA signaling pathways (Fig. 1). This idea is further supported by the repression of *PR*s and *CBF*s by auxin and the level of free SA in *wes1-D*.[12] Further work will be required to determine unequivocally the physiological importance of light-SA interactions in the *WES1*-mediated signaling network. It would be an adaptive strategy that is activated preferentially in the light. Alternatively, it may be related with the rapid acclimation that is essential for plant fitness to natural environment.[19]

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