Why are Seedlings of Large-Seeded Plants Considered to Withstand Drought Stresses Efficiently?

Lai-Sheng Meng* and Ji-Hong Jiang

The Key Laboratory of Biotechnology for Medicinal Plant of Jiangsu Province, School of Life Science, Jiangsu Normal University, Xuzhou, Jiangsu, 221116, People’s Republic of China.

Short Commentary

While seedlings of large-seeded plants are considered to withstand resource restrictions and abiotic stresses more efficiently, plants with small seed sizes are considered more efficient colonizers due to their ability to produce greater numbers of seeds. On the other hand, seed size can be altered intraspecifically in response to environmental cues [1]. Ecologists and evolutionary biologists had early observed this phenomenon [1-4]. However, it is largely unknown the mechanism underlying why are seedlings of large-seeded plants considered to withstand drought stresses efficiently. We found Arabidopsis ARF2 (Auxin Response Factor2), ANT(AINTEGUMENTA) and COR15A(COLD-REGULATED15A) are important regulators of both drought tolerance and seed mass, and the ARF2 transcription repressor negatively regulates the ANT gene through binding directly to its promoter, in turn, the ANT transcription factor positively modulates the expression of the COR15A gene by binding directly to its promoter. Genetic evidence indicates ARF2-ANT-COR15A forms ABA (abscisic acid) signaling-mediated gene cascade for regulation of both drought tolerance and seed mass, which has cross talk with the auxin signal pathway. Together, our proposed model provides a better understanding of seed mass and drought tolerance regulation, which may in turn lead to better increased crop yield and crop breeding [1].

In this model, what signaling molecule performs a key relationship between drought tolerance (stress) and seed mass (growth and development)? We found that the phytohormone abscisic acid (ABA) plays a key role. ABA is required for normal plant growth. Many genotypes with mutations in DNA replication present phenotypes that are hypersensitive to ABA during seed germination and seedling growth [5], suggesting that ABA signaling might repress cell division via modulating DNA replication-related proteins. ABA is also a key regulator of plant responses to environmental cues, including drought, cold, and salt [6]. Drought and salt stresses can lead to the accumulation of ABA, triggering many adaptive responses [7]. For example, under water-deprived conditions, ABA induces stomatal closure and can lead to decreased transpirational water loss [8]. Therefore, in plants, ABA is both endogenous developmental signaling and stress signaling. Under normal physiological condition, low concentrations of ABA can boost root growth through both the promotion of the quiescent center and the inhibition of stem cell differentiation. However, high concentrations of ABA can suppress root growth by suppressing cell division [9]. Our observation in leaf development also support this notion [1]. Together, in our model, ARF2 transcription repressor integrates ABA signals into regulatory of seed mass and drought tolerance by negatively modulating ANT-mediated regulation of COR15A genes.

The loss-of-function mutant of ARF2 presents a pleiotropic phenotype, showing an enhanced growth of aerial organs and seed size as a result of extra cell division, the inhibition of floral bud opening, and the delay in flowering, leaf senescence, floral organ abscission, and silique ripening [10-12]. ANT encodes a transcription factor of the AP2-domain family [13], and its loss-of-function mutant presents the decrease in the number and size of floral organs, as well as the defect in the initiation and growth of the integuments during ovule development. ANT regulates the mass of seeds and organs through cell proliferation by maintaining the meristematic competence of cells during organogenesis [14]. We also found that ANT is an important factor of regulating salt stress. Recently, our reports revealed that ARF2-ANT-COR15A form a ABA signal-mediated gene cascade for regulating both drought tolerance and seed mass. Together, this work presents a large body of work and provides a new insight on the link between auxin and ABA in seed size and abiotic tolerance regulation, and thus this is original and relevant study with important impacts for the basic science as well as agriculture [1].

Similar to this study, currently we reported that ANGSTIFOLIA3 (AN3), a transcription coactivator, regulates Arabidopsis plant drought tolerance by modifying root system and stomatal density via transrepressing YODA expression [15,16]. We also found that AN3 modulates seed mass (our unpublished data). Thus, AN3 integrates an unknown signal into seed mass and drought tolerance by negatively modulating YODA-mediated regulation.

Taken together, our current research not only demonstrated the first direct evidence for ARF2 mediated both seed mass and drought tolerance by negatively regulating the expression of ANT and COR15A, enriched the regulation network of both seed mass and drought tolerance, but also provided a novel insight in understanding the mechanism underlying why are seedlings of large-seeded plants considered to withstand drought stresses efficiently [1]. However, it remain unknown how ABA signaling induces the activity of ARF2 and ANT, and what relationships between ABA receptors and ARF2 or ANT. It is possible that ARF2 or ANT has a similar function to ABA receptors and its function is independent of ABA receptors. Thus, further research is necessary to discover the mystery of ARF2 or ANT in mediating both seed mass and drought tolerance with ABA signaling.

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References

*Corresponding author: Lai-Sheng Meng, The Key Laboratory of Biotechnology for Medicinal Plant of Jiangsu Province, School of Life Science, Jiangsu Normal University, Xuzhou, Jiangsu, 221116, People’s Republic of China. E-mail: menglaisheng@mail.kib.ac.cn

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