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Article Addendum

Cross-talk between gibberellins and salicylic acid in early stress responses in *Arabidopsis thaliana* seeds

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Salicylic acid (SA) is a plant hormone mainly associated with the induction of defense mechanism in plants, although in the last years there is increasing evidence on the role of SA in plant responses to abiotic stress. We recently reported that an increase in endogenous SA levels are able to counteract the inhibitory effects of several abiotic stress conditions during germination and seedling establishment of *Arabidopsis thaliana* and that this effect is modulated by gibberellins (GAs) probably through a member of the GASA (Giberellic Acid Stimulated in Arabidopsis) gene family, clearly showing the existence of a cross talk between these two plant hormones in Arabidopsis.

GAs and SA play important roles in many processes of plant growth and development, and despite the recent papers reporting the existence of a complex network of hormone interactions, evidences of a cross talk between these two plant hormones have been very scarce.[1,2] These authors indicate that GAs are able to regulate SA biosynthesis during plant responses to pathogens. Interestingly, ABA has recently been proved to negative regulate SA-mediated defenses by downregulating SA biosynthesis.[3] These data are consistent with the well known ABA/GAs antagonistic regulation of many aspects of plant development, such as seed dormancy or germination.[4,5] Thus, it seems clear that ABA and GAs are able to control plant immune responses by modulating the levels of salicylic acid and/or jasmonic acid.[1-3] In addition to the role of GAs in the regulation of plant responses to biotic stress, we have recently documented a role of GAs in early plant abiotic stress responses in Arabidopsis through modulation of SA levels,[6] hormone that been involved in responses to abiotic stress conditions.[7] For instance, it has been proved that SA has an important role in heat stress responses[8] or in the improved germination of *Arabidopsis thaliana* seeds under salt stress conditions.[9]

We showed that GAs and the overexpression of a GA-responsive gene were able to increase not only endogenous levels of SA, but also the expression of *ics1* and *npr1* genes, involved in SA biosynthesis and action, respectively.[6] In addition, we have also analyzed expression levels of other genes that have been reported as SA-regulated. For instance, isocitrate lyase, a key enzyme involved in lipid metabolism during seed germination[10] and a good marker of seed vigor under stress conditions,[11] was found to be induced by SA in germinated seeds of *Arabidopsis thaliana*.[9] Thus, we proved that the expression of isocitrate lyase was upregulated in *GASA4* overexpressing lines, and after exogenous application of GA_3 (Fig. 1), both situations increasing endogenous SA levels.[6] We have documented that SA may have a role in some of the physiological processes associated with GAs, since exogenous application of SA was able to both revert the inhibitory effect of PCB on seed germination and improve germination of the GA-deficient mutant *ga1-3*.[6] Thus, we can hypothesize that the GA-mediated induction of isocitrate lyase gene observed in *Arabidopsis thaliana* is the result of the increased levels of SA detected either after overexpression of the GA-induced *GASA4* gene in Arabidopsis or after exogenous application of gibberellic acid. In other words, GAs are able to induce the expression of isocitrate lyase gene in a SA-dependent manner, producing the establishment of a vigorous seedling.[9] These data support the idea that GAs may have an important role in SA biosynthesis and action, and that some of the physiological effects of this hormone may be mediate by SA. In summary, our results clearly show the existence of a cross talk between these two plant hormones during *Arabidopsis thaliana* seeds germination and early seedling growth under abiotic stress conditions, showing another junction in the complex mechanism of hormone interactions.

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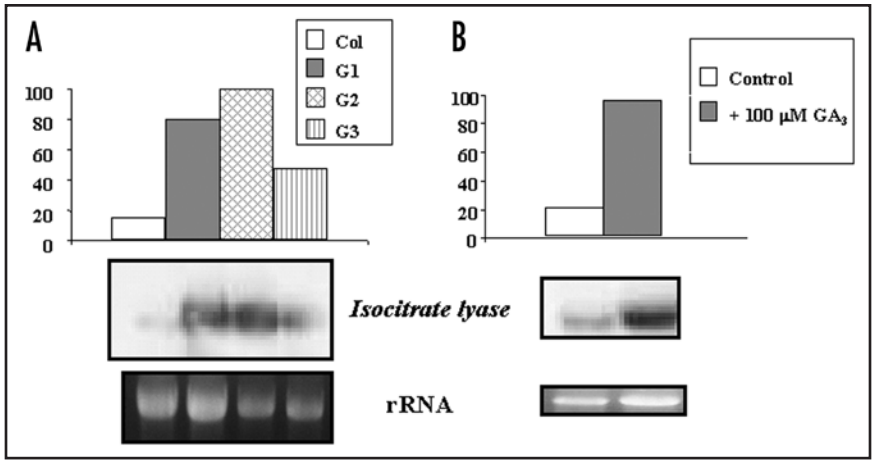

Figure 1. (A) Expression of the isocitrate lyase gene in *FsGASA*-overexpressing plants (G1 to G3) compared to Col-0. (B) Expression of the isocitrate lyase gene in Arabidopsis seedlings treated or not with 100 μM GA_3. mRNA levels were determined by northern blot analysis using total RNAs (10 μg/line) isolated from 7 d-old seedlings. Bottom, ethidium bromide stained gels showing rRNAs. Top: quantification of hybridization signals obtained by using a phosphoimage scanner. Data were normalized to the rRNA value. Blots were repeated twice and yielded similar results.

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References

1. Robert-Seilaniantz A, Navarro L, Bari R, Jones JD. Pathological hormone imbalances. Curr Opin Plant Biol 2007; 10:372-9.
2. Navarro L, Bari R, Achard P, Lison P, Nemri A, Harberd NP, Jones JDG. DELLAs control plant immune responses by modulating the balance of jasmonic acid and salicylic acid signaling. Curr Biol 2008; 18:650-5.
3. de Torres-Zabala M, Bennett MH, Truman WH, Grant MR. Antagonism between salicylic and abscisic acid reflects early host pathogen conflict and moulds plant defence. Plant J 2009; doi: 10.1111/j.1365-313X.2009.03875.x.
4. Kermode AR. Role of abscisic acid in seed dormancy. J Plant Growth Reg 2005; 24:319-44.
5. Kucera B, Cohn MA, Leubner-Metzger G. Plant hormone interactions during seed dormancy release and germination. Seed Sci Res 2005; 15:281-307.
6. Alonso-Ramírez A, Rodríguez D, Reyes D, Jiménez JA, Nicolás G, López-Climent M, et al. Evidence for a role of gibberellins in salicylic acid modulated early plant responses to abiotic stress in *Arabidopsis thaliana* seeds. Plant Physiol 2009; doi: 10.1104/pp.109.139352.
7. Horvath E, Szalai G, Janda T. Induction of abiotic stress tolerance by salicylic acid signaling. J Plant Growth Reg 2007; 26:290-300.
8. Clarke SM, Mur LAJ, Wood JE, Scott IM. Salicylic acid dependent signaling promotes basal thermotolerance but is not essential for acquired thermotolerance in *Arabidopsis thaliana*. Plant J 2004; 38:432-47.
9. Rajjou L, Belghazi M, Huguet R, Robin C, Moreau A, Job C, Job D. Proteomic investigation of the effect of salicylic acid on Arabidopsis seed germination and establishment of early defense mechanisms. Plant Physiol 2006; 141:910-23.
10. Eastmond PJ, Graham IA. Re-examining the role of glyoxylate cycle in oilseeds. Trends Plant Sci 2001; 6:72-8.
11. de los Reyes BG, Myers SJ, McGrath JM. Differential induction of glyoxylate cycle enzymes by stress as a marker for seedling vigor in sugar beet (*Beta vulgaris*). Mol Genet Genomics 2003; 269:692-8.