Commentary

AUXIN BINDING PROTEIN 1 (ABP1): A matter of fact

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Science is a global community enterprise. Collectively, we contribute to building the knowledge tower by using bricks and mortar that should be strong, solid, and long lasting. A brick with defects may lead this tower, or a part there of, to collapse. When that happens, the community suffers, laying waste countless dedicated hours of work, especially by students and postdoctoral scholars. This is the reason why the integrity of science is critically important. We thus should always strive to produce bricks as solid as possible, and we need discussion, and hopefully resolution, when an individual brick, as published data, becomes ambiguously inconsistent.

A recent paper by Gao et al. (2015) from Dr. Yunde Zhao's and Dr. Mark Estelle's labs at the University of California San Diego may have revealed some bricks that require plenty of discussions. The work concerns the AUXIN BINDING PROTEIN 1 (ABP1) that is known to the community for decades as a protein binding to auxin at low concentration *in vitro* (see review, Jones 1994). ABP1 was described as an essential protein as a putative homozygous null mutation conferred embryo lethality (Chen et al. 2001). More recently, based on additional studies, it was proposed as a pivotal auxin receptor responsible for various signaling pathways (Scherer 2011; Grones and Friml 2015). Gao et al. generated a null *abp1* mutant allele in *Arabidopsis thaliana* by using their recently developed stage-specific ribozyme-based CRISPR/Cas9 technology (Gao and Zhao 2014). Complementing this approach, they also identified an *abp1* null allele among the available *A. thaliana* T-DNA lines. Unexpectedly, the newly obtained *abp1* mutants are indistinguishable from wild-type plants in every tested assay, including growth, flowering time, and auxin responsiveness. The findings of Gao et al. (2015) call into question the conclusions of a series of publications that have attributed pronounced defects in embryogenesis, growth, cell division and expansion, and auxin signaling to compromised ABP1 function (Chen et al. 2001; Braun et al. 2008; Tromas et al. 2009; Roberts et al. 2010; Chen et al. 2010, 2014; Xu et al. 2010, 2014 Paque et al. 2014).

Given that the evidence of Gao et al. (2015) seems iron-clad that loss of ABP1 is inconsequential (at least under standard laboratory growth conditions), how can it be

possible that other groups found such strong defects in their T-DNA insertion and TILLING mutants, and in transgenic plants carrying targeted partial knock-downs of ABP1? It is plausible that the inducible expression of an anti-ABP1 monoclonal antibody sequence (Braun et al. 2008; Tromas et al. 2009; Paque et al. 2014) and over-expression of ABP1 antisense RNA (Braun et al. 2008; Chen et al. 2014) may have off-target effects. The previous embryo lethal T-DNA insertion line (*abp1-1*) (Chen et al. 2001) and the TILLING allele (*abp1-5*) (Xu et al. 2010, 2014; Chen et al. 2014) may contain background mutations that may account for the observed phenotypes, but such an interpretation is not reconcilable with the reported results that both *abp1-1* and *abp1-5* were complemented by the wild type ABP1 transgene (Chen et al. 2001; Xu et al. 2010). Regardless how it plays out, this is a good example to remind readers that hypotheses in biology can be disproven, but never proven. We wonder whether the scientists involved fell prey to that dangerous trap of believing a favorite hypothesis?

Gaining insight into nature through research is a trial-and-error process; hence, mistakes are unavoidable. Whereas results from Gao et al. (2015) may also need additional and independent examinations, we strongly encourage those colleagues involved in previous ABP1 studies to go back to their old seed stocks and research notebooks to re-examine what might have happened at the bench level when their experiments were performed. There seems to be a need for careful reexamination of data processing and interpretation, and possibly some tests to be revisited. It is also important that different ABP1-related experimental materials should be made available and exchanged among laboratories involved, to constructively explore possible causes for the discrepancy. No doubt, the ABP1 literature needs to be re-evaluated and, in the process, we might all gain invaluable insight as to how our experiments have the potential to lead us astray.

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