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RETRACTION
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Retraction: Mechanosensitive channel candidate MCA2 is involved in touch-induced root responses in *Arabidopsis*

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Keywords: mechanosensitive channel, Arabidopsis, root, skewing, waving, calcium, touch response, mechanical stress

A retraction of the Original Research Article:

Mechanosensitive channel candidate MCA2 is involved in touch-induced root responses in *Arabidopsis*
by Nakano, M., Samejima, R., and Iida, H. (2014). Front. Plant Sci. 5:421. doi: 10.3389/fpls.2014.00421

The authors and the journal wish to retract the 21 Aug 2014 article cited above in light of new experimental evidence.

Following the publication of the study, we performed a DNA microarray analysis to detect genes with expression levels that were specifically lower in the *mca2*-null mutant than in the Col-0 wild type, and found that the expression level of the *AXR4* gene (At1G54990), which encodes a protein required for the subcellular localization of the auxin influx carrier AUX1 (Dharmasiri et al., 2006), was significantly lower in the *mca2*-null mutant. To confirm this finding, we then performed a semi-quantitative reverse transcription-PCR analysis using the primers axr4-f1 and axr4-Cr1 (**Figure 1A**), and found that the RT-PCR product was detectable in some *mca2*-null seedlings at wild-type levels but not in other *mca2*-null seedlings at all. This result suggested that some *mca2*-null seedlings have a certain lesion in the *AXR4* locus, and a PCR-based genomic deletion analysis (**Figures 1B,C**) followed by DNA sequencing confirmed this speculation. Our conclusion is that most of the *mca2*-null seedlings used in the study presented in the above paper had a homozygous 2592-bp deletion that started from the intron between exons 1 and 2 of the *AXR4* gene and reached the intron between exons 1 and 2 of the adjacent gene *AT1G55000* (**Figure 1A**). Therefore, the phenotypes presented in the above paper may be ascribable to the *axr4* mutation, the *at1g55000* mutation, or both or even triple mutations, but not to the *mca2*-null mutation. The *AT1G55000* gene encodes the peptidoglycan-binding LysM domain-containing protein involved in a macromolecule catabolic process in the cell wall[1].

Our phenotypic study revealed that none of the *mca2*-null *AXR4*$^{+}$ *AT1G55000*$^{+}$ seedlings showed all the abnormal phenotypes reported in the above paper, regarding the skewing, waving, and bending responses of the root. In contrast, the seedlings of the *axr4-1* (Ws-2 background) and *axr4-2* (Col background) single mutants obtained from the Arabidopsis Biological Resource Center (ABRC germplasm names CS8018 and CS8019, respectively)

[1]Database of The Arabidopsis Information Resource (TAIR) at http://www.arabidopsis.org/servlets/TairObject?type=locus&name=AT1G55000

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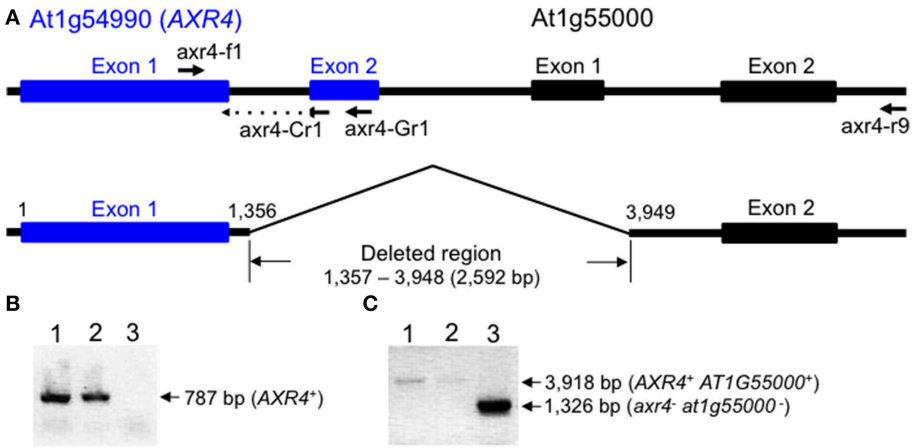

FIGURE 1 | Schematic representation of the *AXR4* locus and PCR data to check its integrity. (A) The *AXR4* locus and its adjacent locus At1g55000 in *Arabidopsis thaliana* (ecotype Col-0). The upper line drawing shows intact loci, while the lower one shows loci with the 2592-bp deletion found in some *mca2*-null mutants. The position and orientation of the primers (axr4-f1, axr4-Cr1, axr4-Gr1, and axr4-r9) are presented by arrows. Nucleotide numbering starts from the 5′-end of exon 1 in the *AXR4* gene. **(B)** An example of PCR data with the primers axr4-f1 and axr4-Gr1. **(C)** An example of PCR data with the primers arx4-f1 and axr4-r9. Genomic DNA was isolated from an *mca2*-null mutant with the $mca2^{-}/mca2^{-}$ $AXR4^{+}/AXR4^{+}$ genotype (lane 1), the wild type with the $MCA2^{+}/MCA2^{+}$ $AXR4^{+}/AXR4^{+}$ genotype (lane 2), and another *mca2*-null mutant with the $mca2^{-}/mca2^{-}$ $axr4^{-}/axr4^{-}$ genotype (lane 3), and subjected to PCR followed by agarose gel electrophoresis. The nucleotide sequences of the primers axr4-f1, axr4-Cr1, axr4-Gr1, and axr4-r9 were 5′-GCAACGTGTAGCTAAGGCTCTTCC-3′, 5′-TCGCCGGATTT GCTTTCCTGAG-3′, 5′-GTAGTAGTCCATTCCTCACCAAG-3′, and 5′-AGCTCCATCTTCGTGTCTAG-3′, respectively, where the underlined nucleotide sequence is complementary to the coding strand of the 3′-terminal region of the exon 1 of the *AXR4* gene. The PCR conditions used were as follows: initial activation of Taq DNA polymerase (Quick Taq™ HS DyeMix, TOYOBO, Osaka, Japan) at 94°C for 2 min, followed by 30 cycles of denaturation at 94°C for 30 s, annealing at 55°C for 30 s, and extension at 68°C for 2 min.

showed the same abnormal phenotypes as those described in the above paper. We also confirmed that the abnormal phenotypes for the skewing, waving, and bending responses of the *mca2*-null *arx4 at1g55000* triple mutant were identical to those of the *axr4-1* and *axr4-2* single mutants. These findings clearly demonstrated that the abnormal phenotypes described in the above paper were ascribed solely to the mutation in the *AXR4* gene.

An important question is why did some of our *mca2*-null germplasms have the $axr4^{-}/axr4^{-}$ $at1g55000^{-}/at1g55000^{-}$ allele? We had never used *axr4* mutants in our laboratory before the above paper was published. We speculated that some of the seeds of the *mca2*-null mutant (germplasm name: SALK_129208) obtained from the ABRC 12 years ago were heterozygous for the *AXR4* and *AT1G55000* loci (i.e., $AXR4^{+}/axr4^{-}$ $AT1G55000^{+}/at1g55000^{-}$), and multiple self-pollinations performed by us to maintain seed viability produced seed stocks with $axr4^{-}/axr4^{-}$ $at1g55000^{-}/at1g55000^{-}$ as well as $AXR4^{+}/AXR4^{+}$ $AT1G55000^{+}/AT1G55000^{+}$ and $AXR4^{+}/axr4^{-}$ $AT1G55000^{+}/at1g55000^{-}$, although all of the 20 seeds of the *mca2*-null mutant (germplasm name: SALK_129208C), which were newly obtained from the ABRC and tested, had the genotype of $AXR4^{+}/AXR4^{+}$ $AT1G55000^{+}/AT1G55000^{+}$. As for the *MCA2* locus, 19 out of the 20 seeds had the $mca2^{-}/mca2^{-}$ genotype and one had the $MCA2^{+}/mca2^{-}$ genotype. The *mca1*-null *mca2*-null double mutant and *mca2/MCA2* complementation lines used in the above paper were $AXR4^{+}/AXR4^{+}$ $AT1G55000^{+}/AT1G55000^{+}$. Furthermore, the *mca2*-null single and *mca1*-null *mca2*-null lines used in our previous study (Yamanaka et al., 2010) were also $AXR4^{+}/AXR4^{+}$ $AT1G55000^{+}/AT1G55000^{+}$.

We deeply regret any scientific misconceptions that have been caused by the above paper and apologize to the scientific community for any adverse consequences.

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Conflict of Interest Statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.