Correction to: Structures of plant resistosome reveal how NLR immune receptors are activated

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Published online: 28 February 2020

Correction to: aBIOTECH

<https://doi.org/10.1007/s42994-019-00012-y>

In the article by Shi et al., published online on December 06, 2019, the figure of the stepwise activation of ZAR1 unexpectedly resembled Fig. 1 of the article of Adachi et al. (Adachi H, Kamoun S, Maqbool A. *Nat Plants*, 2019, 5(5):457–458). To avoid any confusion, the authors replaced Fig. 1 with a revised version and amended the figure caption.

The original article can be found online at <https://doi.org/10.1007/s42994-019-00012-y>.

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Fig. 1 The stepwise activation and proposed working model of ZAR1. In the absence of AvrAC and PBL2, ZAR1 binds an ADP and forms a complex with RKS1 (resting state). Upon pathogen infection, the effector AvrAC secreted by *Xanthomonas campestris* pv. *campestris* (*Xcc*) uridylylates PBL2, enabling the uridylated form of PBL2 (PBL2^{UMP}) to be recruited by ZAR1–RKS1 complex through direct interaction with RKS1. Binding of PBL2^{UMP} promotes ADP release and primes the ZAR1–RKS1–PBL2^{UMP} complex in an intermediate state (priming state). After binding of ATP/dATP, the tertiary complexes undergo a structural remodeling and form a pentamer called resistosome (activation state). The resistosome is proposed to disturb the integrity of plasma membrane (PM) through the funnel-shaped structure formed by the very N-terminal helices ($\alpha 1$) of ZAR1 proteins (membrane insertion)