Thigmoimmunity: mechanical signals prime the quantitative disease resistance of plant.

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Every year, agriculture suffers millions of euros losses due to damage by pests around the world. To meet a growing global food demand, the identification of the genetics basis underlying plant sustainable resistance to pests is a central task. *Sclerotinia sclerotiorum* is the causal fungal pathogen of white mold disease and infects more than 400 plants species such as oilseed rape. *Arabidopsis thaliana* exhibits a quantitative disease resistance (QDR) to *S. sclerotiorum* (Roux et al. 2014). This type of resistance is achieved by multiple intricate genes networks, not always specific to immunity (Peyraud et al. 2017). As of now, QDR is thought to be triggered by the perception of pathogen associated molecules. Interestingly, plant-fungus interactions involve also mechanical signaling intrinsic to the penetration of host tissue by pathogens (Nezhad et al. 2013, Jayaraman et al. 2014). Thigmoimmunity, the interplay between mechanical signals and plant immunity, was evidenced in the interaction of *A. thaliana* with *Botrytis cinerea* (Chehab et al. 2012, Benikhlef et al. 2013). However, the molecular mechanisms underlying thigmoimmunity and the impact of mechanical signal intensity remains enigmatic (Mbengue et al. 2016). Here, we investigated thigmoimmunity in natural accessions and mutant lines of *A. thaliana* submitted to a range of mechanical signal intensities, to which we applied strains from 10^{-3}% to 10% at different frequencies. Leaves submitted to mechanical signals were then inoculated with *S. sclerotiorum*. The analysis of infection phenotypes revealed that mechanical signal increased significantly plant resistance by limiting strongly the spreading speed of infection in tissue. First results indicate that gain of resistance was not linearly correlated with intensity of mechanical signal. Connections between mechanotransduction and QDR pathways are currently studied on the basis of transcriptome analysis.

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