Regulation of differentiation of nitrogen-fixing bacteria by microsymbiont targeting of plant thioredoxin s1

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Résumé en anglais	Legumes associate with rhizobia to form nitrogen (N2)-fixing nodules, which is important for plant fitness [1, 2]. Medicago truncatula controls the terminal differentiation of Sinorhizobium meliloti into N2-fixing bacteroids by producing defensin-like nodule-specific cysteine-rich peptides (NCRs) [3, 4]. The redox state of NCRs influences some biological activities in free-living bacteria, but the relevance of redox regulation of NCRs in planta is unknown [5, 6], although redox regulation plays a crucial role in symbiotic nitrogen fixation [7, 8]. Two thioredoxins (Trx), Trx s1 and s2, define a new type of Trx and are expressed principally in nodules [9]. Here, we show that there are four Trx s genes, two of which, Trx s1 and s3, are induced in the nodule infection zone where bacterial differentiation occurs. Trx s1 is targeted to the symbiosomes, the N2-fixing organelles. Trx s1 interacted with NCR247 and NCR335 and increased the cytotoxic effect of NCR335 in S. meliloti. We show that Trx s silencing impairs bacteroid growth and endoreduplication, two features of terminal bacteroid differentiation, and that the ectopic expression of Trx s1 in S. meliloti partially complements the silencing phenotype. Thus, our findings show that Trx s1 is targeted to the bacterial endosymbiont, where it controls NCR activity and bacteroid terminal differentiation. Similarly, Trxs are critical for the activation of defensins produced against infectious microbes in mammalian hosts. Therefore, our results suggest the Trx-mediated regulation of host peptides as a conserved mechanism among symbiotic and pathogenic interactions.
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