Small non-coding RNAs (sRNAs) are ubiquitous components of regulatory networks underlying virtually any adaptive response in bacteria. As regulators of gene expression, sRNAs accumulate differentially in the cell in response to external cues. Their main activity mechanism involves protein-assisted base-pairing interactions with short and discontinuous stretches of complementary nucleotides usually mapping to the translation initiation region of multiple trans-encoded target mRNAs. In most cases, this antisense interaction promotes post-transcriptional gene silencing by blocking translation and/or degradation of the target mRNAs by cellular RNases (reviewed in Jiménez-Zurdo et al. 2013).

In our laboratory, we are interested in the characterization of the non-coding transcriptome of the *Medicago sativa* (alfalfa) symbiont *Sinorhizobium meliloti*. Deletion of one of the hundreds of sRNA genes identified in this bacterium alters nodule organogenesis and compromises nitrogen-fixation efficiency on alfalfa roots (Robledo et al, 2017). According to this symbiotic phenotype, this sRNA was named NfeR1 (Nodule Formation Efficiency RNA). NfeR1 uses redundantly three identical anti-Shine Dalgarno (aSD) motifs for targeting and down-regulation of multiple mRNAs coding for the periplasmic component of ABC transport systems. Northern blot probing and transcription tracking with promoter-eGFP fusions uncovered maximum NfeR1 accumulation upon an osmotic upshift in free-living bacteria and during all steps of the symbiotic interaction with *M. sativa*. The strength and differential regulation of nfeR1 transcription is conferred by a motif that is conserved in the promoter regions of its α-proteobacterial homologs. Pull-down assays revealed that the NfeR1 promoter might bind the global nitrogen regulator NtrC and the LysR type transcriptional regulator LsrB (LysR Symbiotic Regulator).

In the communication, we will provide novel insights into the transcriptional regulation, targeting potential, activity mechanism and symbiotic function of NfeR1.


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