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Plant Small RNA Species Direct Gene Silencing in Pathogenic Bacteria as well as Disease Protection

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Abstract

Plant small RNAs (sRNAs) and/or double-stranded RNAs (dsRNAs) trigger RNA interference (RNAi) in interacting eukaryotic pathogens or parasites. However, it is unknown whether this phenomenon could operate in bacterial phytopathogens, which lack a eukaryotic-like RNAi machinery. Here, we first show that Arabidopsis-encoded inverted repeat transgenes trigger silencing of *Pseudomonas syringae* heterologous reporter and endogenous virulence-associated genes during infection. Antibacterial Gene Silencing (AGS) of the latter was associated with a reduced pathogenesis, which was also observed upon application of corresponding plant-derived RNAs onto wild-type plants prior to infection. We additionally demonstrate that sRNAs directed against virulence factor transcripts were causal for silencing and pathogenesis reduction, while cognate long dsRNAs were inactive. Overall, this study provides the first evidence that plant sRNAs can directly reprogram gene expression in a phytopathogenic bacterium and may have wider implications in the understanding of how plants regulate transcriptome, community composition and genome evolution of associated bacteria.

Introduction

RNAi is a conserved gene regulatory mechanism that has been characterized as an antiviral defense response by repressing translation, accumulation and/or replication of viral RNAs¹. In plants, RNAi has also been shown to control resistance against bacterial, fungal and oomycete pathogens partly by fine-tuning the expression of immune-responsive genes^{2,3}. The core mechanism of RNAi involves the processing of dsRNAs or single-stranded RNAs (ssRNAs) carrying stem loop structures (e.g. primary microRNA –miRNA– transcripts) by DCL proteins leading to the production of 20-25 nt long short interfering RNAs (siRNAs) or miRNAs. siRNAs or miRNAs are then loaded into Argonaute (AGO) proteins to direct post-transcriptional silencing of sequence complementary mRNA targets through endonucleolytic

cleavage and/or translational inhibition⁴.

An important feature of plant sRNAs, and particularly of siRNAs, is their ability to trigger non-cell autonomous silencing in adjacent cells as well as in distal tissues^{5,6}. This phenomenon is essential to prime antiviral response ahead of the infection front but also to translocate silencing signals between plant cells and their non-viral eukaryotic interacting (micro)organisms^{7,8}. For example, plant sRNAs were previously found to be exported in the fungal pathogens *Verticillium dahliae*⁹ and *Botrytis cinerea*¹⁰ as well as in the oomycete pathogen *Phytophthora capsici*¹¹, leading to the silencing of pathogenicity factors. On the other hand, fungal siRNAs from *B. cinerea* were shown to be translocated into plant cells to silence defense genes¹², highlighting bidirectional RNAi in a natural plant-fungal interaction. In addition, a very recent report provides evidence that rhizobial tRNA-derived sRNAs can silence legume symbiotic genes¹³. However, whether plant sRNAs can in turn directly reprogram gene expression in plant-associated bacteria remains unknown.

Artificial trans-kingdom RNAi has long been employed to direct Host-Induced Gene Silencing (HIGS), a technology used to characterize the function of fungal and oomycete genes or to engineer disease resistance in plants. HIGS notably relies on *in planta* expression of dsRNAs bearing homologies to essential and/or virulence genes and can operate in insects, nematodes, parasitic plants, oomycete and fungi. For example, HIGS confers full protection against *Fusarium graminearum*¹⁴ and *B. cinerea*¹⁰, a phenotype which can be recapitulated by spraying antifungal dsRNAs and/or siRNAs onto Arabidopsis wild type (WT) plants prior to infection^{15,10}. The latter phenomenon is referred to as Spray-Induced Gene Silencing (SIGS) and is reminiscent of ‘environmental RNAi’, a process involving the uptake of RNAs from the environment to trigger RNAi^{16,17}. However, so far, HIGS and SIGS have only been shown to be functional in eukaryotic (micro)organisms possessing canonical RNA silencing factors. Indeed, there is currently no evidence indicating that host-encoded dsRNAs and/or sRNAs

could direct gene silencing in interacting phytopathogenic bacteria, which lack conventional eukaryotic-like RNAi factors. It is also unknown whether external application of any of these RNA entities could trigger environmental RNAi in pathogenic prokaryotic cells and if such hypothetical RNA-based regulatory process could be used to protect plants towards bacterial diseases. Here, we wanted to test these intriguing ideas using *Pseudomonas syringae* pv. *tomato* strain DC3000 (*Pto* DC3000) as an experimental model system.

Results

Pto DC3000 is a Gram-negative bacterium that is the causal agent of bacterial speck disease in tomato and can also infect *Arabidopsis thaliana*¹⁸. This bacterium enters into leaf tissues through stomata or wounds and further multiplies in the apoplast¹⁹. To assess whether plant sRNAs and/or dsRNAs could reprogram *Pto* DC3000 gene expression, we first targeted a chromosomally integrated *Photobacterium luminescens luxCDABE* reporter driven by the constitutive kanamycin promoter²⁰. This *lux*-tagged *Pto* DC3000 (*Pto* WT LUC) strain spontaneously emits luminescence because it co-expresses the luciferase catalytic components *luxA* and *luxB* genes along with the genes required for substrate production, namely *luxC*, *luxD* and *luxE*²¹ (Fig.1a). Two independent *Arabidopsis* transgenic lines that constitutively express a chimeric inverted repeat which carries sequence homology with the coding regions of *luxA* and *luxB* were selected on the basis of their ability to produce anti-*luxA* and anti-*luxB* siRNAs (Fig.1b). They were subsequently syringe-infiltrated with the *lux*-tagged *Pto* DC3000 strain and the levels of luminescence activity were monitored at 24 hours post-inoculation (hpi). A significant reduction in luminescence activity was found in IR-*LUXA/LUXB*-compared to control Col-0-infected plants (Fig.1c). By contrast, the growth of the bacterial reporter strain remained unchanged in the apoplast of IR-*LUXA/LUXB* lines compared to Col-0 plants (Fig.1d), indicating that the above effects were not due to a decreased bacterial titer

in these transgenic lines. Furthermore, a significant reduction in the accumulation of both *luxA* and *luxB* mRNAs was found in IR-*LUXA/LUXB*- compared to control Col-0-infected plants, while the levels of the non-targeted *proC* bacterial transcripts remained unchanged (Fig.1e). These data indicate that an Arabidopsis-encoded inverted repeat can trigger silencing of *Pto* DC3000 heterologous reporter genes during infection.

Pto DC3000 possesses a large repertoire of pathogenicity factors, among them the type III secretion system (TTSS) and the phytotoxin coronatine (COR), which mimics the most active isoleucine conjugate of the phytohormone Jasmonic Acid (JA), both being critical for pathogenesis²²⁻²⁵. To investigate whether Antibacterial Gene Silencing (AGS) could be additionally effective against endogenous *Pto* DC3000 genes that are relevant for pathogenesis, we next generated Arabidopsis transgenic plants that constitutively express a chimeric inverted repeat carrying sequence homology with the coding regions of the *coronafacic acid polyketide synthase I (cfa6)* gene and of the *hrpL* alternative sigma factor of *Pto* DC3000 (Fig.2a). The former bacterial factor is essential for the biosynthesis of coronafacic acid (CFA), which is a major structural component of COR²⁶, while the latter controls directly the expression of type III-secretion system associated genes and indirectly the expression of COR biosynthesis genes^{27, 28}. As a negative control, we have generated Arabidopsis transgenic lines overexpressing an inverted repeat, which does not exhibit sequence homology with the *Pto* DC3000 genome, but instead targets three cytochrome P450 lanosterol C-14 α -demethylase (*CYP51*) genes of the fungal pathogen *F. graminearum*¹⁴. These stable transgenic lines are referred to as IR-*CFA6/HRPL* and control vector (CV) plants, respectively, and do not exhibit any developmental defect compared to Col-0 plants, despite high accumulation of artificial siRNAs (Fig.2b, 2c; Supplementary Fig.2). Additional characterization of the reference IR-*CFA6/HRPL*#4 transgenic line by sRNA sequencing, revealed high and comparable levels of anti-*cfa6* and anti-*hrpL* siRNAs, with a bias towards

21 nt siRNAs (Fig.2d). Furthermore, siRNAs were produced along the *CFA6* and *HRPL* regions of the chimeric inverted repeat (Fig.2e), which is consistent with a processive cleavage of the *CFA6/HRPL* dsRNA precursors by DCL proteins. An sRNA target prediction analysis against the *Arabidopsis thaliana* and *Pto* DC3000 annotated genes also indicated that an off-target effect seems unlikely (Supplementary Fig.1, Table S1, S2).

We further dip-inoculated these transgenic plants with *Pto* DC3000 and monitored *cfa6* and *hrpL* mRNA levels by RT-qPCR analyses at 3 days-post infection (dpi). While the *cfa6* mRNA levels were moderately altered in two out of three independent IR-*CFA6/HRPL* lines compared to Col-0 plants, the levels of *hrpL* transcripts were reproducibly and significantly reduced in all the three IR-*CFA6/HRPL* lines compared to Col-0 plants (Fig.2f). By contrast, the down-regulation of *cfa6* or *hrpL* mRNAs was not observed in CV- *versus* Col-0-infected plants (Fig.2f), supporting a specific effect of these antibacterial RNAs. In addition, the mRNA level of the non-targeted *proC* gene was unchanged in IR-*CFA6/HRPL*-infected lines compared to Col-0- or CV-infected plants (Fig.2f). We conclude that the Arabidopsis-encoded IR-*CFA6/HRPL* transgene can at least trigger silencing of the *Pto* DC3000 *hrpL* gene during infection.

It has been previously reported that *Pto* DC3000 triggers the reopening of stomata as a counter-defense towards plant-induced stomatal closure, which is rapidly orchestrated upon bacterial detection²⁹. This virulence response is critical for *Pto* DC3000 pathogenesis as it allows bacterial cells to reach the apoplast and to colonize inner leaf tissues. Previous studies have also shown that COR plays a major role in *Pto* DC3000-induced stomatal reopening³⁰. Accordingly, the COR-deficient *Pto* DC3118 strain, which is specifically mutated in the *cfa6* gene and referred to here as *Pto* DC3000 $\Delta cfa6$ (*Pto* $\Delta cfa6$), is not able to reopen stomata at 3 hpi (Fig.3a, Supplementary Fig.3a). Similarly, we found that the *Pto* DC3000 $\Delta hrpL$ (*Pto* $\Delta hrpL$) strain, which is also deficient in COR biosynthesis²⁴, was not capable of

reopening stomata (Fig.3a, Supplementary Fig.3a). On the contrary, and as shown previously³⁰, a normal stomatal reopening phenotype was observed during infection with the type III secretion-defective *Pto* DC3000*hrcC* mutant (*Pto* Δ *hrcC*) (Fig.3a; Supplementary Fig.3a), indicating that at this infection timepoint, type III effectors appear to be dispensable for this response.

We next monitored *Pto* DC3000-induced stomatal reopening response in IR-*CFA6*/*HRPL* transgenic lines. Significantly, these plants were fully insensitive to the stomatal reopening response typically triggered by *Pto* DC3000 at 3 hpi (Fig.3b; Supplementary Fig.3b), thereby mimicking the phenotypes observed during infection of Col-0 plants with the *Pto* Δ *cfa6* or the *Pto* Δ *hrpL* strains (Fig.3a, 3b; Supplementary Fig.3a, 3b). By contrast, *Pto* DC3000-triggered stomatal reopening events were unaltered in CV- compared to Col-0-infected plants (Fig.3c; Supplementary Fig.3c), supporting a specific effect of antibacterial RNAs in this process. In addition, the altered stomatal reopening phenotype detected in IR-*CFA6*/*HRPL* plants infected with the WT *Pto* DC3000 strain, and in Col-0 plants challenged with the *Pto* Δ *cfa6* or the *Pto* Δ *hrpL* strains, was fully rescued upon exogenous application of COR (Fig.3a, 3b; Supplementary Fig.3a, 3b). These data provide pharmacological evidence that the reduced *Pto* DC3000 pathogenesis manifested at infected IR-*CFA6*/*HRPL* stomata is likely caused by an altered ability of the associated and/or surrounding bacterial cells to produce COR.

We next assessed the impact that AGS could have on the ability of *Pto* DC3000 to colonize the apoplast of surface-inoculated leaves, a phenotype which is dependent on both type III effectors and COR²². To this end, we dip-inoculated Col-0, CV and IR-*CFA6*/*HRPL* plants with *Pto* DC3000 and subsequently monitored bacterial titer at 2 dpi. We found that *Pto* DC3000 was less effective in colonizing the apoplast of IR-*CFA6*/*HRPL* lines compared to Col-0 and CV-infected plants, a phenotype that was comparable to the growth defect of the *Pto* Δ *cfa6* strain in Col-0 plants (Fig.4a). Nevertheless, this phenotype was less pronounced

than the one observed with the *Pto* Δ *hrpL* strain grown on Col-0 plants (Supplementary Fig.4a), which might be due to the partial silencing of the targeted virulence factors and/or to the known RNA silencing suppression activity triggered by *Pto* DC3000 (Fig.2f)^{31, 32}. In addition, we noticed that IR-*CFA6*/*HRPL* plants dip-inoculated with *Pto* DC3000 showed reduced water-soaking symptoms at 1 dpi (Fig.4b), thereby phenocopying Col-0 plants dip-inoculated with the *Pto* Δ *cfa6* or the *Pto* Δ *hrpL* strains (Supplementary Fig.4b). Therefore, AGS can alter the ability of *Pto* DC3000 to trigger water-soaking and to multiply in the apoplast of Arabidopsis leaves, which are both critical steps of bacterial pathogenesis³³.

Next, we assessed whether AGS could interfere with the capacity of *Pto* DC3000 to colonize xylem vessels, which is the main route used by this bacterium to propagate in the leaf vasculature^{34, 35}. This *Pto* DC3000 virulence phenotype has been recently characterized and found here to be dependent on both type III effectors and COR (Supplementary Fig.4c, 4d). For this purpose, we wound-inoculated the leaf midvein of IR-*CFA6*/*HRPL* plants with a GFP-tagged *Pto* DC3000 (*Pto* DC3000-GFP) and further scored the number of bacterial propagation from inoculation sites. We found a strong decrease in *Pto* DC3000-GFP vascular propagation in the IR-*CFA6*/*HRPL* lines compared to Col-0- and CV-infected plants (Fig.4c, 4d), thereby mimicking the impaired spreading phenotype of the *Pto* Δ *cfa6*-GFP and *Pto* Δ *hrpL*-GFP strains on Col-0 plants (Supplementary Fig.4c). Altogether, these data demonstrate that AGS can limit pathogenesis of *Pto* DC3000 in different tissues of Arabidopsis transgenic plants.

Environmental RNAi is a phenomenon by which (micro)organisms uptake external RNAs from the environment, resulting in the silencing of genes containing sequence homologies to the RNA triggers¹⁷. This process has been initially characterized in *Caenorhabditis elegans*³⁶⁻³⁹ and was further found to operate in other nematodes but also in insects, plants and fungi^{15,40}. However, this approach has never been used against a bacterial phytopathogen that

lacks a canonical eukaryotic-like RNAi machinery such as *Pto* DC3000. To test this possibility, we first assessed whether RNA extracts from IR-*CFA6/HRPL* plants could trigger silencing of *cfa6* and *hrpL* genes in *in vitro* conditions. For this purpose, we incubated total RNAs from CV and IR-*CFA6/HRPL* plants with *Pto* DC3000 cells, and further analyzed by RT-qPCR the levels of *cfa6* and *hrpL* mRNAs. Results from these analyses revealed a reduction in the accumulation of both virulence factor mRNAs upon treatment with RNA extracts from IR-*CFA6/HRPL* plants (Fig.5a). By contrast, the level of the non-targeted *proC* and *rpoB* mRNAs remained unaltered in the same conditions (Fig.5a). These data imply that plant antibacterial RNAs are likely taken-up by *Pto* DC3000 cells and subsequently trigger gene silencing in these prokaryotic cells. It also suggests that exogenous application of these antibacterial RNAs could be used as a strategy to dampen pathogenesis. To test this idea, we pre-treated Arabidopsis Col-0 leaf sections with total RNAs from IR-*CFA6/HRPL* plants for one hour, subsequently challenged them with *Pto* DC3000, and further monitored stomatal reopening events at 3 hpi. Strikingly, we found that RNA extracts from IR-*CFA6/HRPL* plants fully suppressed the ability of *Pto* DC3000 to reopen stomata (Fig.5b; Supplementary Fig.6a), thereby mimicking the phenotype observed in infected IR-*CFA6/HRPL* leaves (Fig.3b; Supplementary Fig.3b). We additionally investigated whether this approach could be used to control the growth of *Pto* DC3000 *in planta*. For this purpose, we pre-treated for one hour Arabidopsis Col-0 plants with total RNAs from IR-*CFA6/HRPL* plants and further dip-inoculated them with *Pto* DC3000. We found that these RNA extracts triggered a decreased *Pto* DC3000 titer at 2 dpi, a phenotype that was comparable to the ones observed in IR-*CFA6/HRPL* and Col-0 plants inoculated with the *Pto*Δ*cfa6* strain (Fig.4a, 5c). By contrast, application of total RNAs from CV plants did not alter growth of *Pto* DC3000 in the same conditions (Fig.5c), supporting a specific effect of antibacterial RNAs in this process. To assess whether such RNA-based biocontrol approach could also be effective in cultivated

plants, we repeated the same assay on tomato (*Solanum lycopersicum*), which is the natural host of *Pto* DC3000. Pre-treatment of WT tomato leaves for one hour with RNA extracts from IR-*CFA6/HRPL* plants led to compromised *Pto* DC3000-induced necrotic disease symptoms and to a reduction in bacterial content compared to leaves pre-treated with RNA extracts from CV plants (Fig.5d, 5e, 5f; Supplementary Fig.5a, 5b, 5c). Collectively, these data demonstrate that external application of plant antibacterial RNAs can trigger AGS and disease protection against *Pto* DC3000 in both Arabidopsis and tomato plants.

Next, we interrogated which RNA entities are responsible for AGS upon external application of antibacterial RNAs. We first introduced the *dcl2-1*, *dcl3-1* and *dcl4-2* mutations in the IR-*CFA6/HRPL*#4 reference line. These resulting IR-*CFA6/HRPL*#4 *dcl234* plants revealed high accumulation of IR-*CFA6/HRPL* transcripts, despite probable transcriptional repression of the 35S_{pro}:IR-*CFA6/HRPL* transgene due to the use of T-DNA insertional mutant carrying the 35S promoter (Fig.6a)^{41, 42}. Furthermore, anti-*cfa6* and anti-*hrpL* siRNAs were undetectable in those plants (Fig.6b), which is consistent with a role of DCL2, DCL3 and DCL4 in the processing of IR-*CFA6/HRPL*. We subsequently extracted total RNAs from these plants, incubated them with *Pto* DC3000 cells, and further monitored *cfa6* and *hrpL* mRNA levels by RT-qPCR analysis. Using this *in vitro* assay, we found that RNA extracts from IR-*CFA6/HRPL*#4 *dcl234* plants were no longer able to trigger down-regulation of *cfa6* and *hrpL* mRNAs (Fig.6c), despite high accumulation of *CFA6/HRPL* dsRNA precursor transcript (Fig.6a). By contrast, RNA extracts from the IR-*CFA6/HRPL*#4 plants, which contain high levels of anti-*cfa6* and anti-*hrpL* siRNAs (Fig.2c, 6b), triggered reduced accumulation of these bacterial mRNAs (Fig.5a, 6c).

We next decided to analyze the antibacterial activity of the above RNA extracts by monitoring their effects on stomatal reopening of Col-0-infected plants. A prerequisite for this assay was to verify that Col-0 leaf sections would not trigger the processing of *CFA6/HRPL*

dsRNAs, and thus the production of anti-*cfa6* and anti-*hrpL* siRNAs, upon incubation with RNA extracts from IR-*CFA6/HRPL#4 dcl234* plants, which was validated by northern blot analysis (Fig.6d, Supplementary Fig.6b). Interestingly, when we further monitored stomatal aperture in Col-0-infected leaf tissues pre-treated for one hour with these RNA entities, we found that *Pto* DC3000 triggered normal stomata reopening, thereby mimicking the effect observed with control RNAs from Col-0 or *dcl234* plants (Fig.6e; Supplementary Fig.6c). By contrast, RNA extracts from IR-*CFA6/HRPL#4* plants, which contain abundant anti-*cfa6* and anti-*hrpL* siRNAs, triggered full suppression of stomatal reopening in the same conditions (Fig.6d, 6e). Collectively, these data indicate that *CFA6/HRPL* long dsRNAs are neither involved in AGS nor in pathogenesis reduction. They rather suggest that corresponding siRNAs are likely the RNA entities responsible for these phenotypes. To verify this assumption, we further purified sRNA species from IR-*CFA6/HRPL* plant total RNAs using a glass fiber filter-based method (Supplementary Fig.7a, 7b), and subjected them to stomatal reopening assay. We found that these sRNA species suppressed *Pto* DC3000-triggered stomatal reopening, to the same extent as IR-*CFA6/HRPL* plant total RNA extracts (Fig.6f; Supplementary Fig.7c). By contrast, long RNA species, which were not filtered through the above columns, were inactive (Fig.6f; Supplementary Fig.7c), further supporting that *CFA6/HRPL* long dsRNAs are not involved in this response. We have then synthesized *CFA6/HRPL* long dsRNAs and cognate double-stranded siRNAs *in vitro* to further test their antibacterial activity (Fig.6g). The *in vitro*-synthesized *CFA6/HRPL* long dsRNAs did not alter the ability of *Pto* DC3000 to reopen stomata, nor did *CYP51* long dsRNAs used as controls (Fig.6h; Supplementary Fig.7d). By contrast, *in vitro*-synthesized anti-*cfa6* and anti-*hrpL* siRNAs fully suppressed stomatal reopening (Fig.6h; Supplementary Fig.7d), and accordingly, triggered silencing of *cfa6* and *hrpL* genes (Fig.6i). Altogether, these data

provide evidence that siRNAs directed against *cfa6* and *hrpL* genes are critical for AGS and pathogenesis reduction, while cognate long dsRNAs are ineffective for both processes.

Although the above findings indicate that siRNAs can trigger AGS and antibacterial activity, they do not firmly demonstrate that these RNA entities are causal for these phenomena. To address this, we decided to generate recombinant bacteria expressing a siRNA-resilient version of the *hrpL* gene. To this end, we complemented the *PtoΔhrpL* mutant with either a WT *hrpL* transgene or a mutated version that contains as many silent mutations as possible in the siRNA targeted region, which were predicted to alter the binding of siRNAs with the *hrpL* mRNA but to produce the same protein sequence (Fig.7a, 7b, Supplementary Table3). Both transgenes were expressed under the constitutive neomycin phosphotransferase II (*NPTII*) promoter (Fig.7c). The resulting recombinant bacteria, referred to as *PtoΔhrpL* WT *hrpL* and *PtoΔhrpL* mut *hrpL*, were found to restore the ability of the *PtoΔhrpL* strain to reopen stomata when inoculated on Col-0 leaf sections (Fig.7f; Supplementary Fig.9a, 9b), indicating that both transgenes are functional in a stomatal reopening assay. We further assessed the sensitivity of each recombinant bacterium to AGS. For this purpose, we incubated *PtoΔhrpL* WT *hrpL* and *PtoΔhrpL* mut *hrpL* strains with total RNA extracts from CV and IR-*CFA6/HRPL#4* plants and further monitored *hrpL* transgene mRNA levels by RT-qPCR analysis. We found a significant decrease in the accumulation of *hrpL* mRNAs expressed from the *PtoΔhrpL* WT *hrpL* strain, which was not detected upon treatment with RNA extracts from CV plants (Fig.7d). These data indicate that the WT *hrpL* transgene expressed from the *PtoΔhrpL* WT *hrpL* strain is sensitive to AGS despite its constitutive expression driven by the *NPTII* promoter. By contrast, the accumulation of *hrpL* mRNAs expressed from the *PtoΔhrpL* mut *hrpL* strain was unaltered in response to RNA extracts from IR-*CFA6/HRPL#4* plants (Fig.7d), indicating that siRNAs no longer exert their AGS effect

towards this recombinant bacterium. Collectively, these findings demonstrate that anti-*hrpL* siRNAs are causal for silencing of the *hrpL* gene in *Pto* DC3000 cells.

We finally investigated the responsiveness of each recombinant bacterial strain to siRNA-directed pathogenesis reduction by exploiting the *Pto* DC3000-induced stomatal reopening assay. To assess the specific effect of siRNAs towards suppression of *hrpL*-mediated stomatal reopening function, we cloned an IR-*HRPL* inverted repeat targeting the same *hrpL* sequence region than the one targeted by the IR-*CFA6/HRPL* hairpin, and further validated its capacity to produce anti-*hrpL* siRNAs upon Agrobacterium-mediated transient expression in *Nicotiana benthamiana* leaves (Fig.7e). *N. benthamiana* total RNAs containing anti-*hrpL* siRNAs were found to fully suppress the ability of *Pto* DC3000 to reopen stomata (Fig.7f, Supplementary Fig.8). Importantly, similar results were obtained when *N. benthamiana* RNA extracts containing anti-*hrpL* siRNAs were incubated with the *Pto*Δ*hrpL* WT *hrpL* strain (Fig.7f; Supplementary Fig.9a, 9b), supporting a sensitivity of this bacterial strain to siRNA action. By contrast, the *Pto*Δ*hrpL* mut *hrpL* strain was fully competent in reopening stomata (Fig.7f; Supplementary Fig.9a, 9b), indicating that anti-*hrpL* siRNAs no longer exert their antibacterial effects towards this recombinant bacterium. Similar results were also observed in Arabidopsis stable IR-*HRPL* transgenic lines overexpressing anti-*hrpL* siRNAs (Fig.7g, 7h; Supplementary Fig.9c). This indicates that the suppression of stomatal reopening phenotype is not due to potential off-target effects of these host-encoded siRNAs but rather caused by their targeting effects over the *hrpL* transcript sequence. Altogether, these data provide sound evidence that anti-*hrpL* siRNAs are causal for the suppression of *hrpL*-mediated stomatal reopening function. They also further validate a novel role of *hrpL* in bacterial-induced stomatal reopening, indicating that AGS can be employed as a tool to characterize bacterial gene function.

Discussion

We show here that Arabidopsis-encoded siRNAs trigger the silencing of *Pto* DC3000 virulence-associated genes, resulting in the dampening of pathogenesis. In particular, we found that anti-*cfa6* and/or anti-*hrpL* siRNAs fully suppressed *Pto* DC3000-induced stomatal reopening, a virulence response that is dependent on the production of COR by epiphytic bacterial cells⁴⁶. Our data therefore indicate that antibacterial siRNAs can act at the pre-invasive stage of the infection, presumably by preventing COR biosynthesis in *Pto* DC3000 cells that come in contact with the leaf surface. In addition, we found that AGS was capable of reducing the ability of *Pto* DC3000 to mount water-soaking symptoms, to multiply in the leaf apoplast and to propagate in the leaf vasculature of Arabidopsis. Therefore, siRNAs additionally act at a post-invasive stage of the infection by targeting endophytic bacterial cells present in the apoplast and in xylem vessels. These observations imply that siRNAs must be externalized from plant cells towards the leaf surface, the apoplast and xylem vessels in order to reach epiphytic and endophytic bacterial populations. One siRNA trafficking mechanism might involve plant extracellular vesicles (EVs), whose secretion is enhanced during antibacterial defense and which contain diverse species of sRNAs^{43, 44}. Such a hypothesis would be consistent with recent findings showing that EVs ensure the movement of siRNAs from plant cells to fungal or oomycete cells^{7,11}. It would also be congruent with a recent report showing that plant-derived exosome-like nanoparticles ingested by mice deliver plant miRNAs into specific commensal bacteria to reprogram their gene expression⁴⁵. In accordance with these studies, we found that EVs isolated from the Arabidopsis IR-*CFA6/HRPL#4* leaf apoplast, which were in a size range between 50 to 300 nm⁴⁴ (Fig.8a; Supplementary Fig.10a), were fully competent in suppressing stomatal reopening, as observed with the apoplastic fluid from these transgenic plants (Fig.8b; Supplementary Fig.10b, 10d,

10f). This suggests that apoplastic EVs may, at least in part, contribute to the trafficking of sRNAs from plant cells towards *Pto* DC3000 cells.

We additionally showed that incubating RNA extracts containing antibacterial siRNAs with *Pto* DC3000 cells was sufficient to trigger AGS. However, corresponding dsRNAs were ineffective for this process, suggesting that the latter RNA entities are either not taken-up by, or not properly processed in, *Pto* DC3000 cells. This is a major distinction from environmental RNAi in *C. elegans* and plant herbivores, which specifically relies on long dsRNAs^{37-40,46}, or in filamentous pathogens, which is triggered by both dsRNAs and siRNAs^{15,10}. Instead, we found that plant siRNA species were causal for environmental RNAi in *Pto* DC3000, and this regulatory process was even, intriguingly, recapitulated in the presence of *in vitro*-synthesized siRNA duplexes. These data imply that *Pto* DC3000 must additionally be capable of taking-up –passively and/or actively– free sRNAs, despite the presence of a cell wall and an intricate double membrane structure. The data also suggest that *Pto* DC3000 must have evolved a machinery to take charge of the internalized sRNAs and direct gene silencing in bacterial cells. It will thus be appealing to identify such prokaryotic factors and to elucidate the principles of sRNA target recognition and mode of action in bacterial cells. Investigating whether our findings also apply to endogenous plant sRNAs, and assessing their possible implications in the regulation of the transcriptome, community composition and genome evolution of plant-associated bacteria will represent exciting directions for future studies.

Material and Methods

Plasmid construction

The IR-*CFA6/HRPL* construct is composed of 250 bp regions of *Pto* DC3000 genes, *cfa6* (1-250 nt) and *hrpL* (99-348 nt), aligned in sense and antisense directions with the intron of the petunia chalcone synthase gene (*CHSA*) in between. The control vector (CV) construct IR-*CYP51* is composed of described conserved regions from *F. graminearum* *CYP51A*, *CYP51B* and *CYP51C*¹³ and the IR-*LUXA/LUXB* is composed regions from *luxA* and *luxB* genes of the *luxCDABE* operon and they are aligned in sense and antisense directions with the same intron sequence as described above. The IR-*CFA6/HRPL*, IR-*CYP51* and IR-*LUXA/LUXB* constructs containing *EcoRI* and *Sall* sites at both extremities were synthesized by GenScript® and inserted by restriction enzymatic digestion into a modified pDON221-P5-P2 vector carrying additional *EcoRI* and *Sall* sites to facilitate the insertion of these long-inverted repeats. The plasmids containing the 35S_{pro}:IR-*CFA6/HRPL* and 35S_{pro}:IR-*CYP51* were obtained by a double recombination between pDON221-P5-P2 carrying the inverted repeat sequences and pDON221-P1-P5r carrying the Cauliflower Mosaic Virus (CaMV) 35S promoter sequence, in the the pB7WG Gateway destination vector using LR clonase plus (Life Technologies). These plasmids were then introduced into the Agrobacterium C58C1 strain. The IR-*HRPL* construct, which is composed of the same 250 bp region of *hrpL* as in the IR-*CFA6/HRPL* construct, was recombined by using GreenGate technology⁴⁷ to constitute the plasmid 35S_{pro}:IR-*HRPL*, which was then transformed in the Agrobacterium C58C1 strain. To generate the WT *hrpL* and the mut *hrpL* plasmids, the wild type *hrpL* sequence was amplified from the genomic DNA isolated from *Pto* DC3000, while the mutant *hrpL* sequence was amplified from a mutated sequence synthesized by GenScript®. These two sequences were further cloned into pDON207 vector using BP clonase (Life Technologies) and then introduced by recombination using LR clonase (Life Technologies) into the pBS0046 destination vector, which carries a

constitutive NPTII promoter. Specific primers used for the purpose of cloning are listed in Supplementary table 4.

Plant material and growth conditions

Stable transgenic lines expressing IR-*LUXA/LUXB*, IR-*CFA6/HRPL*, IR-*CYP51* (CV) and IR-*HRPL* constructs were generated by transforming Arabidopsis (accession Col-0) plants using Agrobacterium mediated-floral dip method⁴⁸. Two independent Arabidopsis T2 transgenic lines of IR-*LUXA/LUXB*, #18 and #20; three independent Arabidopsis T4 transgenic lines of IR-*CFA6/HRPL*, #4, #5 and #10; two independent Arabidopsis T2 transgenic lines of IR-*HRPL*, #1 and #4 and one reference Arabidopsis T4 transgenic line for IR-*CYP51* #2 (CV) were generated and used in our experiments. For genetic analysis, *dcl2-1 dcl3-1 dcl4-2* (*dcl234*) triple mutant was crossed with the reference line IR-*CFA6/HRPL*#4 and the F3 plants were genotyped to select homozygous *dcl234* mutant containing the IR-*CFA6/HRPL* transgene. Sterilized seeds of Arabidopsis Col-0 and the selected homozygous transgenic lines were first grown for 12-14 days at 22°C on plates containing ½ x MS medium (Duchefa), 1% sucrose and 0.8 % agar (with or without antibiotic selection) in 8h photoperiod. Seedlings were then pricked out to soil pots and grown in environmentally controlled conditions at 22°C/ 19°C (day/night) with an 8h photoperiod under light intensity of 100 µE/m²/s. Four- to five-week-old plants were used for all the experiments. Seeds of tomato (*Solanum lycopersicum* ‘Moneymaker’) and *Nicotiana benthamiana* were directly germinated on soil pots and grown in environmentally controlled conditions at 22 °C/ 19°C (day/night) with a 16h photoperiod under light intensity of 100 µE/m²/s. Four- to five-week old plants were used for all the experiments.

Bacterial strains

The *PtoΔcfa6*-GFP (*Pto* DC3118-GFP) strain is a gift from S. Y. He, while the *PtoΔhrpL* strain is a gift from C. Ramos⁴⁹. The *PtoΔhrpL* strain expressing the *GFP* reporter gene was generated by transformation with the GFP-pPNpt Green plasmid by electroporation and then plated on NYGB medium (5 g L⁻¹ bactopectone, 3 g L⁻¹ yeast extract, 20 ml L⁻¹ glycerol) containing gentamycin (1 μg mL⁻¹) for selection at 28 °C. To generate the *Pto* DC3000 WT *hrpL* and mut *hrpL* strains, the *PtoΔhrpL* strain was transformed with the plasmids *NPTII_{pro}:WT-hrpL* and *NPTII_{pro}:mut-hrpL*, respectively, by electroporation and then plated in NYGB medium with gentamycin (1 μg mL⁻¹) at 28°C for two days. The colonies containing the plasmid were selected by PCR using specific primers to *hrpL* gene. The lux-tagged *Pto* DC3000 strain has been previously described²¹.

Bacterial infection assays in plants

(a) Bacterial dipping assay: Three hours after the beginning of the night cycle in growth chamber, three plants per condition were dip-inoculated with *Pto* DC3000 at 5 x 10⁷ cfu mL⁻¹ supplemented with 0.02 % Silwet L-77 (Lehle seeds). Plants were then immediately placed in chambers with high humidity. Water-soaking symptoms were observed 24 hours post-infection and pictures of representative leaves were taken. Two days post-inoculation, bacterial titer was measured for individual infected leaves (n=8). To quantify bacterial transcripts in infected plants, pool of infected leaves from three plants was collected three days post-inoculation.

(b) Wound-inoculation assay: Bacterial propagation in the mid-veins was assessed as described previously³⁴. Around 15 leaves from three plants per condition were inoculated with a toothpick dipped in *GFP*-tagged bacteria at a concentration of 5 x 10⁶ cfu mL⁻¹ and then the plants were covered for 3 days. Bacterial propagation was then analyzed by monitoring GFP

signal under a UV light using an Olympus MV 10 × Macrozoom with a GFP filter and representative pictures of bacterial propagation were taken with a CCD camera AxioCam Mrc Zeiss.

(c) Plant protection assay: Prior to bacterial infection, four leaves of three Col-0 plants were dipped with mock solution (water) or 20 ng μl^{-1} of specific total RNAs, both supplemented with 0.02% of Silwet L-77. After one hour, leaves were dip-inoculated with *Pto* DC3000 WT or *Pto* Δ *cfa6* at 5×10^7 cfu ml^{-1} with 0.02% of Silwet L-77. Bacterial titers were monitored two days post-inoculation, as specified earlier. Similar assay was performed using tomato plants but with a *GFP*-tagged *Pto* DC3000. Representative pictures of bacterial disease symptoms observed at 3 dpi were depicted.

(d) Bacterial luminescence quantification: Three plants per condition were syringe-infiltrated with the *lux*-tagged *Pto* DC3000 strain at 10^6 cfu ml^{-1} . Plants were placed in a chamber with high humidity to facilitate proper infection. After 24 hours, leaf discs were prepared and placed in individual wells of a 96 well plate to quantify the luminescence using Berthold Centro LB 960 Microplate Luminometer. Four leaves per plant were taken into consideration. Leaf discs from individual leaves were collected and pooled to monitor bacterial titers as described in (a).

Tomato infection quantification

(a) GFP loci quantification: Tomato leaves infected with *Pto* DC3000-GFP strain were analyzed under a UV light using an Olympus MV 10x Macrozoom with a GFP filter and pictures were taken with a CCD camera AxioCam Mrc Zeiss. Number of GFP loci was quantified with ImageJ software for at least 10 pictures per condition. Individual leaves were collected to extract genomic DNA.

(b) Bacterial genomic DNA quantification: To quantify bacterial infection in the infected tomato plants, the amount of bacterial genomic DNA (gDNA) was measured relative to plant gDNA⁵⁰. Genomic DNA was isolated from tomato leaf samples infected with *Pto* DC3000-GFP using the DNeasy plant mini kit (QIAGEN, Germany) according to the manufacturer's instructions. Using 1 ng of gDNA, qPCR was performed using Takyon SYBR Green Supermix (Eurogentec®) and GFP-specific primers. Amount of bacterial gDNA was normalized to that of tomato using *Ubiquitin*-specific primers. Primer sequences are listed in Supplementary table 4.

Agrobacterium-mediated transient expression of inverted repeats in *N. benthamiana*

To express the IR-*HRPL* hairpin and the IR-*CFA6/HRPL* chimeric hairpin transiently, the *A. tumefaciens* C58C1 strains carrying these plasmids were grown overnight in LB medium at 28°C. Cells were resuspended in a solution containing 10 mM MES, pH 5.6, 10 mM MgCl₂ and 200 µM acetosyringone at a final concentration of 0.5 OD₆₀₀. Cultures were incubated in the dark at room temperature for 5-6 hours before Agrobacterium-mediated infiltration in 4-week old *N. benthamiana*. After 3 days, leaves were harvested for total RNA extraction and molecular analyses.

Stomatal aperture measurements

Using intact leaves from three plants, sections were dissected and immersed in mock solution (water) or bacterial suspension at 10⁸ cfu ml⁻¹. After 3 hours, unpeeled leaf sections were stained with 10 µg ml⁻¹ propidium iodide (Sigma) and abaxial surface was observed under SP5 laser scanning confocal microscope. The stomatal aperture (width relative to length) was measured using ImageJ software for at least 50-70 stomata per condition. For RNA and vesicle treatments, the leaf sections were incubated with total RNAs or APF and P40 fraction

extracted from specified genotypes for one hour before incubation with the bacteria. In specified experiments, 1 μM of Coronatine (COR; Sigma) was supplemented to the bacterial suspension.

***In vitro* AGS assay**

To assess whether the transcripts of *Pto* DC3000 *cfa6* and *hrpL* can be directly targeted by the dsRNA and/or the siRNAs generated by the hairpin IR-*CFA6/HRPL*, 2 ml cultures of *Pto* DC3000 WT, *Pto* Δ *hrpL* WT *hrpL* and *Pto* Δ *hrpL* mut *hrpL* at 10^7 cfu ml⁻¹ were treated for 4 and/or 8 hours, with 20 ng μl^{-1} of specified total RNA extracted from CV or IR-*CFA6/HRPL*#4 transgenic plants. For each condition, bacteria were harvested and processed for molecular analyses.

Quantitative RT-PCR Analyses

To monitor plant-encoded transcripts, total RNA was extracted from plant samples using Nucleospin RNA plant kit (Macherey Nagel). 0.5 μg of DNase-treated RNA was reverse transcribed using qScript cDNA Supermix (Quanta Biosciences®). cDNA was then amplified by real time PCR reactions using Takyon SYBR Green Supermix (Eurogentec®) and gene-specific primers. Expression was normalized to that of *Arabidopsis Ubiquitin*. To monitor bacterial transcripts, total RNA was extracted from bacteria-infected plant samples or from *in vitro* treated bacteria using Nucleospin RNA kit. After DNase treatment, 250 ng of total RNA was reverse transcribed using random hexamer primers and qScript Flex cDNA kit (Quanta Biosciences®) and then amplified by real time PCR reaction using gene-specific primers. Expression was normalized to that of *gyrA*. Real time PCR was performed in 384-well optical reaction plates heated at 95°C for 10 min, followed by 45 cycles of denaturation at 95°C for 15s, annealing at 60°C for 20s, and elongation at 72°C for 40s. A melting curve was

performed at the end of the amplification by steps of 1°C (from 95°C to 50°C). Primer sequences are listed in Supplementary table 3.

RNA Gel Blot Analyses

Accumulation of low molecular weight RNAs was assessed by Northern blot analysis as previously described³¹. Total RNA was extracted using TriZOL reagent and stabilized in 50% formamide and 30 µg of total RNAs were used. To generate specific ³²P-radiolabelled probes, regions of 150 bp to 300 bp were amplified from the plasmids using gene specific primers (Supplementary Table 4) and the amplicons were labeled by random priming (Prime-a-Gene® Labeling System, Promega). U6 was monitored as an equal loading control.

Separation of long and small RNA fractions

From 100 µg of TriZOL-extracted total RNA, long and small RNA fractions were separated using the mirVana miRNA isolation kit (Ambion®) according to the manufacturer's instructions. The long and small RNA fractions were visualized using agarose gel electrophoresis and further analyzed using Bioanalyzer 2100 (Agilent Technologies).

***In vitro* synthesis of Inverted Repeat (IR) RNAs**

In vitro synthesized RNAs were generated following the instruction of the MEGAscript® RNAi Kit (Life Technologies, Carlsbad, CA). Templates were amplified by PCR using gene specific primers containing the T7 promoter. PCR amplification was done in two steps with two different annealing temperatures to increase the specificity of primers annealing. After the amplification step, PCR products were purified by gel extraction using the PCR Clean-up kit (Macherey-Nagel) to eliminate any unspecific PCR products. To produce dsRNAs, the purified PCR products were used as templates for *in vitro* transcription performed according

to the MEGAscript RNAi Kit instructions (Life Technologies). After purification with the filter cartridges, the corresponding dsRNAs were processed in 18-25 nt siRNAs by ShortCut® RNase III (NEB, Ipswich, MA) for 40 min at 37°C. siRNAs were then specifically purified by using the mirVana™ miRNA Isolation Kit (Life Technologies, Carlsbad, CA). Each step of this process was followed by gel electrophoresis (TAE 1X, 1% agarose gel for DNA amplification and 2% agarose gel for RNAs) to verify the quality of RNAs.

Apoplastic Fluid (APF) and Extracellular Vesicles (EVs) extraction

Extraction was done as previously described¹⁷. Briefly, sixty leaves of 5 week-old CV or IR-*CFA6/HRPL* plants were syringe-infiltrated with vesicle isolation buffer (20 mM MES, 2 mM CaCl₂, 0.01 M NaCl, pH 6.0). Leaves were then placed inside a 20 ml needleless syringe that was inserted in a 50 ml Falcon and centrifuged at 900 g for 15 minutes at 4°C. The apoplastic fluid (APF) was collected and, to get rid of any cell debris, filtered through 0.45 µm filter before to be centrifuged at 10,000 g for 30 minutes at 4°C. The APF was subjected to an ultracentrifugation step at 40,000 g for 60 min at 4°C to pellet EV fraction (P40) that was washed with vesicle isolation buffer and pelleted again. The pellet was further resuspended in 2 ml of 20 µM Tris buffer pH=7.5 and stored at 4°C. The size and concentration of EVs from both the APF and P40 fractions were analyzed using a LM10 Nanoparticle Tracking Analysis device (Malvern)⁵¹.

Bioinformatic analysis

Small RNA libraries were constructed and sequenced from four- to five-week old leaves of IR-*CFA6/HRPL*#4. Raw reads have been deposited at the NCBI SRA under Bioproject (PRJNA587213). Libraries were mapped against the *Arabidopsis thaliana* genome (v TAIR10.1 GCF_000001735.4) and the IR-*CFA6/HRPL* sequence using ShortStack (default

parameters)⁵². Coverage of mapped loci was obtained with the Genomic Alignments package in R⁵³. Unique reads mapping to *cfa6* and *hrpL* in both replicates were extracted and quantified from ShortStack mapping results using samtools⁵⁴. Each unique read was aligned against annotated Arabidopsis and *Pto* DC3000 coding sequences (CDS) using BLAST⁵⁵ (-e-value 10, -word_size 4, -ungapped, -reward 1, -penalty -1), and the top target (with lowest e-value) was kept for each read in each target set. Binding free energy was calculated for each read/target pair using RNAup (-interaction pairwise)⁵⁶.

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Author contributions

M. S-R., M.C., O.T. and A.R. performed the experiments, A.E.F. and V.M. generated some constructs, A.P-Q conducted bioinformatic analyses, M. S-R., O.T., and L.N. analyzed the data; M. S-R., O.T. and L.N. designed the experiments, M. S-R. and L.N. wrote the manuscript, L.N. conceived the overall study.

Competing interests

Two patents have been applied for (PCT/EP2019/072169, PCT/EP2019/072170) on the AGS technology on which M. S-R and L.N. are listed as inventors.

Figure legends

Fig. 1. Stable expression of the IR-*LUXA/LUXB* inverted repeat transgene in *Arabidopsis* triggers silencing of *Pto* DC3000 *luxA* and *luxB* genes during infection, which is accompanied by a decrease in luminescence activity

a. Schematic representation of the *luxCDABE* operon chromosomally integrated in the genome of *Pto* DC3000²⁰. The 250 bp regions of *luxA* (1-250 nt; highlighted region in pink) and *luxB* (1-250 nt; highlighted region in violet) were used to generate the chimeric hairpin construct under the control of the constitutive 35S promoter, hereby designated as 35S_{pro}:IR-*LUXA/LUXB*. **b.** Accumulation levels of anti-*luxA* and anti-*luxB* siRNAs in the two independent *Arabidopsis* transgenic lines expressing 35S_{pro}:IR-*LUXA/LUXB* were detected by low molecular weight northern blot analysis. U6 was used as a loading control. **c.** Luminescence activity of *lux*-tagged *Pto* DC3000 (*Pto* WT LUC) is decreased in the infected *Arabidopsis* transgenic lines carrying the 35S_{pro}:IR-*LUXA/LUXB* transgene compared to the infected Col-0 plants. Col-0 plants and IR-*LUXA/LUXB* #18 and #20 transgenic lines were syringe-infiltrated with *Pto* WT LUC at 10⁶ cfu ml⁻¹. The bacterial luminescence and titers were monitored at 1 day post-infection (dpi). Four leaves from three plants per condition and from three independent experiments (n) are considered for the analysis. The luminescence activity was estimated by calculating the ratio of bacterial luminescence recorded from individual leaf sample and the number of bacteria (data presented in d.) counted for the corresponding sample. **d.** The IR-*LUXA/LUXB* transgenic lines, #18 and #20, do not exhibit significantly reduced *Pto* WT LUC titer when compared to Col-0-infected plants. Bacterial titers were monitored for the same samples used in c. **e.** Accumulation level of both *luxA* and *luxB* mRNAs are significantly decreased on IR-*LUXA/LUXB*-infected plants compared to Col-0-infected plants. Using the same samples as described in c. and d., bacterial transcript levels of *proC*, *luxA* and *luxB* were monitored by quantitative RT-PCR analysis. These

mRNA levels were quantified relative to the levels of bacterial *gyrA* transcript. Error bars indicate the standard deviations of mRNA values obtained in three independent experiments. Statistically significant differences were assessed using ANOVA test (ns: p-value>0.05; *: p-value<0.05, **: p-value<0.01, ****: p-value<0.0001).

Fig. 2. Stable expression of the IR-*CFA6*/*HRPL* inverted repeat transgene in *Arabidopsis* triggers silencing of *Pto* DC3000 *cfa6* and *hrpL* genes during infection

a. Schematic representation of the *Pto* DC3000 genes *cfa6* (in blue) and *hrpL* (in green). The 250 bp regions of *cfa6* (1-250 nt) and *hrpL* (99-348 nt) were used to generate the chimeric hairpin construct under the control of the constitutive 35S promoter. **b.** Representative pictures of five-week old *Arabidopsis* Col-0 plants and of independent homozygous transgenic plants expressing the 35S_{pro}:IR-*CYP51* (Control vector: CV) or the 35S_{pro}:IR-*CFA6*/*HRPL* transgenes. **c.** Accumulation level of anti-*cfa6* and anti-*hrpL* siRNAs for the *Arabidopsis* plants depicted in b. was detected by low molecular weight northern blot analysis. U6 was used as a loading control. **d.** Size distribution and abundance of 20-24 nt long sRNA reads from IR-*CFA6*/*HRPL*#4 transgenic line that were mapped to the *cfa6* (upper panel) and *hrpL* (lower panel) regions of the IR-*CFA6*/*HRPL* sequence. Data from two biological replicates (rep1 and rep2) are presented. **e.** The coverage of sRNA reads which is computed as total count of mapped reads across the IR-*CFA6*/*HRPL* inverted repeat is depicted and include both the plus and minus strand of the construct. **f.** *Pto* DC3000 *hrpL* mRNA accumulation is significantly decreased in the three independent IR-*CFA6*/*HRPL*-infected transgenic plants compared to Col-0- and CV-infected plants. *Arabidopsis* plants depicted in b. were dip-inoculated with 10⁸ cfu ml⁻¹ *Pto* DC3000 WT strain and at 3 days post-infection (dpi), bacterial transcript levels of *proC*, *cfa6* and *hrpL* were monitored by quantitative RT-PCR analysis. These mRNA levels are quantified relative to the level of

bacterial *gyrA* transcripts. Error bars indicate the standard deviations of mRNA values obtained in three independent experiments. Statistically significant differences were assessed using ANOVA test (ns: p-value>0.05; *: p-value<0.05, **: p-value<0.01, ***: p-value<0.001).

Fig. 3. Stable expression of the IR-*CFA6*/*HRPL* inverted repeat transgene in *Arabidopsis* suppresses *Pto* DC3000-induced stomatal reopening

a. The *Pto* Δ *cfa6* and *Pto* Δ *hrpL* strains, but not the *Pto* Δ *hrcC* strain, were impaired in their ability to reopen stomata and these phenotypes were rescued upon addition of exogenous COR. Sections of unpeeled leaves of Col-0 plants were incubated with mock solution (water) or 10^8 cfu ml⁻¹ of *Pto* DC3000 WT, Δ *cfa6*, Δ *hrpL* or Δ *hrcC* strains for 3 hours. Stomatal aperture was assessed by computing the ratio width over length using ImageJ software. **b.** *Pto* DC3000 WT no longer induced stomatal reopening in *Arabidopsis* transgenic lines overexpressing the IR-*CFA6*/*HRPL* inverted repeat transgene. Stomatal aperture measurement was conducted in Col-0 and IR-*CFA6*/*HRPL*#4, #5, #10 transgenic lines infected with *Pto* WT strain as described in a. **c.** The *Pto* DC3000-induced stomatal reopening response was unaltered in CV compared to Col-0 plants. Stomatal aperture measurement was conducted in Col-0 and CV plants incubated with *Pto* WT strain as described in a. For all these experiments, n = number of stomata analyzed per condition and statistical significance was assessed using the ANOVA test (ns: p-value > 0.05; ****: p-value < 0.0001). Similar results were obtained from at least three independent experiments (two other biological replicates are presented in Supplementary Fig.3). Pictures at the bottom of the graph depict the representative stomatal aperture phenotypes corresponding to the mean value for each condition/genotype.

Fig. 4. Stable expression of the IR-*CFA6*/*HRPL* inverted repeat transgene in *Arabidopsis* inhibits the ability of *Pto* DC3000 to multiply in the apoplast, to mount water-soaking symptoms and to spread in the leaf vasculature

a. *Arabidopsis* IR-*CFA6*/*HRPL* transgenic lines exhibit a significantly reduced *Pto* WT titer when compared to Col-0 and CV-infected plants. Col-0, CV and IR-*CFA6*/*HRPL*#4, #5 and #10 plants were dip-inoculated with *Pto* WT-GFP and Col-0 plants were dip-inoculated with the *Pto*Δ*cfa6*-GFP strain. Bacterial titers were monitored at 2 days post-infection (dpi). Four leaves from three plants per condition and from three independent experiments (n) were considered for the comparative analysis. Statistical significance was assessed using the ANOVA test (ns: p-value>0.05; ****: p-value<0.0001). **b.** IR-*CFA6*/*HRPL*#4, #5 and #10 transgenic plants exhibit reduced water-soaking symptoms in comparison to Col-0 and CV plants. Representative leaf pictures of water-soaking symptoms were taken 1 day after dip-inoculation with *Pto* WT-GFP or *Pto*Δ*cfa6*-GFP strain. **c. & d.** IR-*CFA6*/*HRPL*#4, #5 and #10-infected plants exhibit reduced vascular spreading of *Pto* WT compared to Col-0- and CV-infected plants. Plants were wound-inoculated in midveins with *Pto* WT-GFP and Col-0 was wound-inoculated with *Pto*Δ*cfa6*-GFP. White circles indicate the site of wound-inoculation in the midvein. To index the spreading of bacteria from the inoculation sites, GFP fluorescence signal was observed under UV light and pictures were taken at 3 dpi. When the bacteria propagated away from any of the three inoculation sites, it was indexed as propagation with 4 corresponding to the highest propagation index. Pictures from three biological replicates were taken into consideration and one representative picture is depicted.

Fig. 5. Exogenously applied total RNA extracts from IR-*CFA6*/*HRPL* plants reduce *Pto* DC3000 pathogenesis in both *Arabidopsis* and tomato plants

a. *In vitro* AGS assay showing that total RNA extract from IR-*CFA6/HRPL#4* plants triggers silencing of both *cfa6* and *hrpL* genes. *Pto* WT cells were incubated *in vitro* for 4 and 8 hours with 20 ng μl^{-1} of total RNAs from CV or IR-*CFA6/HRPL#4* plants. Significant reduction of the bacterial transcripts *cfa6* and *hrpL* was observed by RT-qPCR at both timepoints, while accumulation of *proC* and *rpoB* transcripts remained unaffected. *gyrA* was used as an internal control to quantify the accumulation of bacterial transcripts. Error bars indicate the standard deviations of values from three independent experiments. **b.** The ability of *Pto* WT to reopen stomata was altered upon exogenous application of total RNA extracts from IR-*CFA6/HRPL* plants compared to CV plants. Col-0 leaves were treated for 1 hour with water or 20 ng μl^{-1} of total RNAs extracted from CV or IR-*CFA6/HRPL#4* plants and were incubated with *Pto* WT for 3 hours. Stomatal aperture was measured and analyzed as described in Fig.3a. Two other biological replicates are presented in Supplementary Fig.6a. **c.** Pretreatment with IR-*CFA6/HRPL#4* total RNAs reduced the ability of *Pto* DC3000 to multiply in the apoplast of leaves when compared to pretreatment with CV total RNAs. Col-0 leaves were treated with 20 ng μl^{-1} of total RNAs from CV or IR-*CFA6/HRPL#4* plants for 1 hour, followed by dip-inoculation with *Pto* WT. Bacterial titers were monitored at 2 dpi as in Fig.4a. The number of leaves (n) corresponds to collective values from three independent biological replicates. Statistically significant differences were assessed using ANOVA test (ns: p-value>0.05; **: p-value<0.01; ***: p-value<0.001). **d.** The tomato leaves treated with CV total RNAs displayed more necrotic symptoms as compared to the leaves treated with IR-*CFA6/HRPL#4* total RNAs. The experiment was conducted as in c. but using five-week-old tomato (*Solanum lycopersicum*, cultivar ‘Moneymaker’) plants. Representative pictures of infected leaves in the two conditions are depicted. **e.** A reduced number of *Pto* WT-GFP foci was observed in tomato leaves treated with total RNA extracts from IR-*CFA6/HRPL#4* vs CV plants. Infected-leaves were observed at 3 dpi under UV light to estimate the number of GFP loci. Left panel:

Dot plot representing the number of GFP loci analyzed using ImageJ software from 3-4 different leaves per condition with at least 4 pictures per leaf. The values used for the analysis are from two different independent experiments. Right panel: Representative picture of the tomato leaves described in d. **f.** *Pto* WT-GFP DNA content is decreased in tomato leaves treated with total RNA extracts from IR-*CFA6/HRPL*#4 vs CV plants. The level of bacterial DNA content was analyzed by qPCR using tomato *Ubiquitin* gene as a control. For e and f, student's t-test was performed for the comparative analysis (ns: p-value>0.05; **: p-value<0.01; ***: p-value<0.001; ****: p-value<0.0001). Results of another biological replicate for tomato infection assay are presented in Supplementary Fig.5.

Fig. 6. Anti-*cfa6* and anti-*hrpL* small RNA species, but not corresponding unprocessed *CFA6/HRPL* dsRNA precursors, are the RNA entities responsible for AGS and for the suppression of stomatal reopening

a. Accumulation level of IR-*CFA6/HRPL* transcripts in Col-0, *dcl2-1 dcl3-1 dcl4-2 (dcl234)*, IR-*CFA6/HRPL*#4 (#4) and IR-*CFA6/HRPL*#4 in *dcl234* mutant background (#4 x *dcl234*) was assessed by RT-qPCR. *Ubiquitin* was used as a control. The mean and standard deviation of three independent experiments are represented here. **b.** Accumulation level of anti-*cfa6* and anti-*hrpL* siRNAs in the same genotypes presented in a. were assessed by low molecular weight northern blot analysis. U6 was used as a loading control. **c.** Total RNA extract from #4 x *dcl234* plants does not alter the transcript accumulation levels of *cfa6* and *hrpL*. *Pto* WT cells were incubated *in vitro* for 8 hours with 20 ng μl^{-1} of total RNAs extracted from the same genotypes described in a. Accumulation levels of *cfa6* and *hrpL* transcripts was assessed by RT-qPCR analysis using *gyrA* as a control. Error bars indicate the standard deviations of values from three independent experiments. **d.** Accumulation level of anti-*cfa6* and anti-*hrpL* siRNAs in the indicated genotypes along with Col-0 plants incubated with total RNAs derived

from the same genotypes for 1 hour were assessed by low molecular weight northern blot analysis. U6 was used as a loading control. Another biological replicate is presented in Supplementary Fig.6b. **e.** Total RNA extract from #4 x *dcl234* plants does not suppress *Pto* DC3000-induced stomatal reopening response. Col-0 leaves were treated with water or 20 ng μl^{-1} of total RNA extracts from the same genotypes used in a. and incubated with *Pto* WT for 3 hours. Stomatal aperture was measured and analyzed as described in Fig.3a. Two other biological replicates are presented in Supplementary Fig.6c. **f.** Small RNA species, but not the corresponding long RNA species, from IR-*CFA6/HRPL#4* plants suppress stomatal reopening to the same extent as total RNAs extracted from these plants. The experiment was conducted as in d. but with total, long (> 200 nt) or small (< 200 nt) RNA fractions, which were separated from total RNAs of IR-*CFA6/HRPL#4* plants. Two other biological replicates are presented in Supplementary Fig.7c. **g.** Agarose gel picture of ethidium bromide stained *in vitro* synthesized (IVS) long and small RNA duplexes. **h.** *In vitro*-synthesized (IVS) sRNAs, but not the corresponding long dsRNAs derived from *CFA6/HRPL* chimeric construct, suppressed stomatal reopening. The experiment was conducted as in d. but with *in vitro* synthesized- (IVS), -long or -small RNA species. Long and small RNAs derived from *CYP51* were used as negative controls. Two other biological replicates are presented in Supplementary Fig.7d. **i.** *In vitro* synthesized (IVS) antibacterial siRNAs significantly alter the transcript accumulation levels of *cfa6* and *hrpL*. Accumulation levels of *cfa6* and *hrpL* transcripts were assessed by RT-qPCR analysis using *gyrA* as a control. Error bars indicate the standard deviations of values from three independent experiments. For all the experiments, statistically significant differences were assessed using ANOVA test (ns: p-value>0.05; *: p-value<0.05, **: p-value<0.01, ****: p-value < 0.0001).

Fig. 7. Anti-*hrpL* siRNAs are causal for the silencing of *Pto* DC3000 *hrpL* gene and for the suppression of stomatal reopening

a. Sequence of WT *hrpL* (99-348 nt) selected to generate the inverted repeat transgene (highlighted in yellow) was aligned with the sequence of mutated *hrpL* (mut *hrpL*) designed to contain as many silent mutations as possible in the siRNA targeted region (highlighted in grey). **b.** Unique reads mapping to *hrpL* gene of *Pto* DC3000 in the two sRNA libraries presented in Fig.2d were used for BLAST analysis. The 500 most abundant anti-*hrpL* siRNA reads produced by IR-*CFA6/HRPL*#4 plants were selected and blasted against the WT and mut *hrpL* sequences depicted in a. The free energy of binding for each read paired with its top target (lowest *e-value*) was calculated using RNAup and is presented here. Few siRNA-target pairs were above the blast threshold (*e-value* <10) when the anti-*hrpL* siRNA reads were blasted against the mut *hrpL* sequence (right panel). Thermodynamic energy analysis of these few siRNA-target interactions revealed a mean free energy of binding that was roughly two times higher than the one obtained for the 500 siRNA-target pairs retrieved with the WT *hrpL* sequence (left panel), supporting an impaired binding of the anti-*hrpL* siRNAs towards the mut *hrpL* sequence. **c.** Schematic representation of the *PtoΔhrpL* strain along with the complementation strains generated upon transformation with the plasmids encoding WT *hrpL* or mut *hrpL*, respectively under the control of the constitutive promoter *NPTII*. **d.** *In vitro* AGS assay showing that the *PtoΔhrpL* WT *hrpL* strain is sensitive to antibacterial RNAs while the *PtoΔhrpL* mut *hrpL* is refractory to these RNA entities. *PtoΔhrpL* WT *hrpL* (left panel) and *PtoΔhrpL* mut *hrpL* (right panel) bacterial strains were incubated with total RNAs extracted from CV or IR-*CFA6/HRPL* #4 plants for 8 hours. Accumulation of WT *hrpL* and mut *hrpL* transcripts was analyzed by RT-qPCR. The mRNA levels were quantified relative to the level of *gyrA* transcripts. Error bars indicate the standard deviations of values from three independent experiments. **e.** Accumulation of anti-*cfa6* and anti-*hrpL* siRNAs was

assessed by low molecular weight northern analysis using total RNA extracts from *N. benthamiana* plants transiently expressing 35S_{pro}:IR-HRPL, 35S_{pro}:IR-CFA6/HRPL and from non-transformed *N. benthamiana* leaves. U6 was used as a loading control. **f.** Both the WT *hrpL* and mut *hrpL* constructs fully complemented *PtoΔhrpL* for its ability to reopen stomata and only the *PtoΔhrpL* mut *hrpL* strain is refractory to anti-*hrpL* siRNA action. Col-0 leaves were treated with total RNAs extracted either from *N. benthamiana* alone or from *N. benthamiana* expressing the inverted repeat IR-HRPL and stomatal reopening response was assessed upon incubation with *PtoΔhrpL* WT *hrpL* and *PtoΔhrpL* mut *hrpL* bacterial strains. Stomatal reopening response was assessed as described previously. Two other biological replicates are presented in Supplementary Fig.9a. **9b g.** Accumulation of anti-*hrpL* siRNAs was assessed by low molecular weight northern analysis using total RNA extracts from Arabidopsis stable transgenic lines expressing the 35S_{pro}:IR-HRPL transgene. U6 was used as a loading control. **h.** Col-0 and IR-HRPL#1 and #4 leaves were treated with water as Mock or indicated bacterial strains for 3 hours prior to stomata aperture measurements. Stomatal reopening response was assessed as described previously. Another biological replicate is presented in Supplementary Fig.9c. For all the stomata experiments, n = number of stomata analyzed per condition. Statistical significance for all the experiments was assessed using the ANOVA test (ns: p-value>0.05; **: p-value<0.001; ****: p-value<0.0001).

Fig. 8. Apoplastic fluid and apoplastic extracellular vesicles (EVs) from Arabidopsis IR-CFA6/HRPL plants trigger suppression of *Pto* DC3000-induced stomatal reopening

a. Apoplastic EVs from the Arabidopsis transgenic lines IR-CYP51 (CV) and IR-CFA6/HRPL #4 (#4) were isolated by collecting the apoplastic fluids (APF) from these plants and subjecting them to ultracentrifugation at 40,000g as previously described⁴⁴. The population of particles from the recovered P40 fractions were analyzed using Nanoparticle Tracking

Analysis (NTA). The number of particles, the size of particles according to their diameter (in nm), and the median size of the particles are depicted for each sample. **b.** Both the APF and the P40 fractions triggered full suppression of stomatal reopening. Stomatal aperture was measured in Col-0 leaves that were incubated for 1 hour with the above APF or P40 fractions before being inoculated with *Pto* WT for 3 hours. Results from another biological replicate are presented in Supplementary Fig.10e, 10f. For the stomata experiment, n = number of stomata analyzed per condition and statistical significance was assessed using the ANOVA test (ns: p-value>0.05; ****: p-value<0.0001).

Fig. 1. Stable expression of the IR-LUXA/LUXB inverted repeat transgene in Arabidopsis triggers silencing of *Pto* DC3000 *luxA* and *luxB* genes during infection, which is accompanied by a decrease in luminescence activity

a. Schematic representation of the *luxCDABE* operon chromosomally integrated in the genome of inserted in the genome of *Pto* DC3000²⁰. The 250 bp regions of *luxA* (1-250 nt; highlighted region in pink) and *luxB* (1-250 nt; highlighted region in violet) were used to generate the chimeric hairpin construct under the control of the constitutive 35S promoter, hereby designated as 35S_{pro}:IR-LUXA/LUXB. **b.** Accumulation levels of anti-*luxA* and anti-*luxB* siRNAs in the two independent Arabidopsis transgenic lines expressing 35S_{pro}:IR-LUXA/LUXB were detected by low molecular weight northern blot analysis. U6 was used as a loading control. **c.** Luminescence activity of *lux*-tagged *Pto* DC3000 (*Pto* WT LUC) is decreased in the infected Arabidopsis transgenic lines carrying the 35S_{pro}:IR-LUXA/LUXB transgene compared to the infected Col-0 plants. Col-0 plants and IR-LUXA/LUXB #18 and #20 transgenic lines were syringe-infiltrated with *Pto* WT LUC at 10⁶ cfu ml⁻¹. The bacterial luminescence and titers were monitored at 1 day post-infection (dpi). Four leaves from three plants per condition and from three independent experiments (n) are considered for the analysis. The luminescence activity was estimated by calculating the ratio of bacterial luminescence recorded from individual leaf sample and the number of bacteria (data presented in d.) counted for the corresponding sample. **d.** The IR-LUXA/LUXB transgenic lines, #18 and #20, do not exhibit significantly reduced *Pto* WT LUC titer when compared to Col-0-infected plants. Bacterial titers were monitored for the same samples used in c. **e.** Accumulation level of both *luxA* and *luxB* mRNAs are significantly decreased on IR-LUXA/LUXB-infected plants compared to Col-0-infected plants. Using the same samples as described in c. and d., bacterial transcript levels of *proC*, *luxA* and *luxB* were monitored by quantitative RT-PCR analysis. These mRNA levels were quantified relative to the levels of bacterial *gyrA* transcript. Error bars indicate the standard deviations of mRNA values obtained in three independent experiments. Statistically significant differences were assessed using ANOVA test (ns: p-value>0.05; *: p-value<0.05; **: p-value<0.01; ****: p-value<0.0001).

Fig. 2. Stable expression of the IR-CFA6/HRPL inverted repeat transgene in Arabidopsis triggers silencing of *Pto* DC3000 *cfa6* and *hrpL* genes during infection
a. Schematic representation of the *Pto* DC3000 genes *cfa6* (in blue) and *hrpL* (in green). The 250 bp regions of *cfa6* (1-250 nt) and *hrpL* (99-348 nt) were used to generate the chimeric hairpin construct under the control of the constitutive 35S promoter. **b.** Representative pictures of five-week old Arabidopsis Col-0 plants and of independent homozygous transgenic plants expressing the 35S_{pro}:IR-CYP51 (Control vector: CV) or the 35S_{pro}:IR-CFA6/HRPL transgenes. **c.** Accumulation level of anti-*cfa6* and anti-*hrpL* siRNAs for the Arabidopsis plants depicted in b. were detected by low molecular weight northern blot analysis. U6 was used as a loading control. **d.** Size distribution and abundance of 20-24 nt long sRNA reads from IR-CFA6/HRPL#4 transgenic line that mapped to the *cfa6* (upper panel) and *hrpL* (lower panel) regions of the IR-CFA6/HRPL sequence. Data from two biological replicates (rep1 and rep2) are presented. **e.** The coverage of sRNA reads which is computed as total count of mapped reads across the IR-CFA6/HRPL inverted repeat is depicted and include both the plus and minus strand of the construct. **f.** *Pto* DC3000 *hrpL* mRNA accumulation is significantly decreased in the three independent IR-CFA6/HRPL-infected transgenic plants compared to Col-0- and CV-infected plants. Arabidopsis plants depicted in b. were dip-inoculated with 10⁸ cfu ml⁻¹ *Pto* DC3000 WT strain and at 3 days post-infection (dpi), bacterial transcript levels of *proC*, *cfa6* and *hrpL* were monitored by quantitative RT-PCR analysis. These mRNA levels are quantified relative to the level of bacterial *gyrA* transcripts. Error bars indicate the standard deviations of mRNA values obtained in three independent experiments. Statistically significant differences were assessed using ANOVA test (ns: p-value>0.05; *: p-value<0.05, **: p-value<0.01, ***: p-value<0.001).

Fig. 3. Stable expression of the IR-*CFA6/HRPL* inverted repeat transgene in *Arabidopsis* suppresses *Pto* DC3000-induced stomatal reopening
a. The *Pto* $\Delta cfa6$ and *Pto* $\Delta hrpL$ strains, but not the *Pto* $\Delta hrpC$ strain, were impaired in their ability to reopen stomata and these phenotypes were rescued upon addition of exogenous COR. Sections of unpeeled leaves of Col-0 plants were incubated with mock solution (water) or 10^8 cfu ml⁻¹ of *Pto* DC3000 WT, $\Delta cfa6$, $\Delta hrpL$ or $\Delta hrpC$ strains for 3 hours. Stomatal aperture was assessed by computing the ratio width over length using ImageJ software. **b.** *Pto* DC3000 WT no longer induced stomatal reopening in *Arabidopsis* transgenic lines overexpressing the IR-*CFA6/HRPL* inverted repeat transgene. Stomatal aperture measurement was conducted in Col-0 and IR-*CFA6/HRPL* #4, #5, #10 transgenic lines infected with *Pto* WT strain as described in a. **c.** The *Pto* DC3000-induced stomatal reopening response was unaltered in CV compared to Col-0 plants. Stomatal aperture measurement was conducted in Col-0 and CV plants incubated with *Pto* WT strain as described in a. For all these experiments, n = number of stomata analysed per condition and statistical significance was assessed using the ANOVA test (ns: p-value > 0.05; ****: p-value < 0.0001). Similar results were obtained from at least three independent experiments (two other biological replicates are presented in Fig. S3). Pictures at the bottom of the graph depict the representative stomatal aperture phenotypes corresponding to the mean value for each condition/genotype.

Fig. 4. Stable expression of the IR-CFA6/HRPL inverted repeat transgene in Arabidopsis inhibits the ability of *Pto* DC3000 to multiply in the apoplast, to mount water-soaking symptoms and to spread in the leaf vasculature

a. Arabidopsis IR-CFA6/HRPL transgenic lines exhibit a significantly reduced *Pto* WT titer when compared to Col-0 and CV-infected plants. Col-0, CV and IR-CFA6/HRPL#4, #5 and #10 plants were dip-inoculated with *Pto* WT-GFP and Col-0 plants were dip-inoculated with the *Pto* $\Delta cfa6$ -GFP strain. Bacterial titers were monitored at 2 days post-infection (dpi). Four leaves from three plants per condition and from three independent experiments (n) were considered for the comparative analysis. Statistical significance was assessed using the ANOVA test (ns: p-value>0.05; ****: p-value<0.0001). **b.** IR-CFA6/HRPL#4, #5 and #10 transgenic plants exhibit reduced water-soaking symptoms in comparison to Col-0 and CV plants. Representative leaf pictures of water-soaking symptoms were taken 1 day after dip-inoculation with *Pto* WT-GFP or *Pto* $\Delta cfa6$ -GFP strain. **c. & d.** IR-CFA6/HRPL#4, #5 and #10-infected plants exhibit reduced vascular spreading of *Pto* WT compared to Col-0- and CV-infected plants. Plants were wound-inoculated in midveins with *Pto* WT-GFP and Col-0 was wound-inoculated with *Pto* $\Delta cfa6$ -GFP. White circles indicate the site of wound-inoculation in the midvein. To index the spreading of bacteria from the inoculation sites, GFP fluorescence signal was observed under UV light and pictures were taken at 3 dpi. When the bacteria propagated away from any of the three inoculation sites, it was indexed as propagation with 4 corresponding to the highest propagation index. Pictures from three biological replicates were taken into consideration and one representative picture is depicted.

Fig. 5. Exogenously applied total RNA extracts from IR-*CFA6/HRPL* plants reduce *Pto* DC3000 pathogenesis in both Arabidopsis and tomato plants

a. *In vitro* AGS assay showing that total RNA extract from IR-*CFA6/HRPL* plants triggers silencing of both *cfa6* and *hrpL* genes. *Pto* WT cells were incubated *in vitro* for 4 and 8 hours with 20 ng μl^{-1} of total RNAs from CV or IR-*CFA6/HRPL* #4 plants. Significant reduction of the bacterial transcripts *cfa6* and *hrpL* was observed by RT-qPCR at both the timepoints, while accumulation of *proC* and *rpoB* transcripts remained unaffected. *gyrA* was used as an internal control to quantify the accumulation of bacterial transcripts. Error bars indicate the standard deviations of values from three independent experiments. Statistically significant differences were assessed using ANOVA test (ns: p-value > 0.05; **: p-value < 0.01, ***: p-value < 0.001). **b.** The ability of *Pto* WT to reopen stomata was altered upon exogenous application of total RNA extracts from IR-*CFA6/HRPL* plants compared to CV plants. Col-0 leaves were treated for 1 hour with water or 20 ng μl^{-1} of total RNAs extracted from CV or IR-*CFA6/HRPL* #4 plants and were incubated with *Pto* WT for 3 hours. Stomatal aperture was measured and analyzed as described in Fig. 3a. Two other biological replicates are presented in Supplementary Fig. 5a. **c.** Pretreatment with IR-*CFA6/HRPL* #4 total RNAs reduced the ability of *Pto* DC3000 to multiply in the apoplast of leaves when compared to pretreatment with CV total RNAs. Col-0 leaves were treated with 20 ng μl^{-1} of total RNAs from CV or IR-*CFA6/HRPL* #4 plants for 1 hour, followed by dip-inoculation with *Pto* WT. Bacterial titers were monitored at 2 dpi as in Fig. 4a. The number of leaves (n) corresponds to collective values from three independent biological replicates. **d.** The tomato leaves treated with CV total RNAs displayed more necrotic symptoms as compared to the leaves treated with IR-*CFA6/HRPL* #4 total RNAs. The experiment was conducted as in c. but using five-week-old tomato (*Solanum lycopersicum*, cultivar 'MoneyMaker') plants. Representative pictures of infected leaves in the two conditions are depicted. **e.** A reduced number of *Pto* WT-GFP foci was observed in tomato leaves treated with total RNA extracts from IR-*CFA6/HRPL* #4 vs CV plants. Infected-leaves were observed at 3 dpi under UV light to estimate the number of GFP loci. Left panel: Dot plot representing the number of GFP loci analyzed using ImageJ software from 3-4 different leaves per condition with at least 4 pictures per leaf. The values used for the analysis are from two different independent experiments. Right panel: Representative picture of the tomato leaves described in d. **f.** *Pto* WT-GFP DNA content is decreased in tomato leaves treated with total RNA extracts from IR-*CFA6/HRPL* #4 vs CV plants. The level of bacterial DNA content was analyzed by qPCR using tomato *Ubiquitin* gene as a control. For e and f, student's t-test was performed for the comparative analysis (ns: p-value > 0.05; **: p-value < 0.01; ***: p-value < 0.001; ****: p-value < 0.0001). Results of another biological replicate for tomato infection assay are presented in Supplementary Fig. 5.

a. Accumulation level of IR-*CFA6*/HRPL transcripts in Col-0, *dcl2-1 dcl3-1 dcl4-2 (dcl234)*, IR-*CFA6*/HRPL#4 (#4) and IR-*CFA6*/HRPL#4 in *dcl234* mutant background (#4 x *dcl234*) was assessed by RT-qPCR. *Ubiquitin* was used as a control. The mean and standard deviation of three independent experiments are represented here. **b.** Accumulation level of anti-*cfa6* and anti-*hrpL* siRNAs in the same genotypes presented in a. was assessed by low molecular weight northern blot analyses. U6 was used as a loading control. **c.** Total RNA extract from #4 x *dcl234* plants does not alter the transcript accumulation levels of *cfa6* and *hrpL*. *Pto* WT cells were incubated *in vitro* for 8 hours with 20 ng μl^{-1} of total RNAs extracted from the same genotypes described in a. Accumulation levels of *cfa6* and *hrpL* transcripts was assessed by RT-qPCR analysis using *gyrA* as a control. Error bars indicate the standard deviations of values from three independent experiments. **d.** Accumulation level of anti-*cfa6* and anti-*hrpL* siRNAs in the indicated genotypes along with Col-0 plants incubated with total RNAs derived from the same genotypes for 1 hour was assessed by low molecular weight northern blot analysis. U6 was used as a loading control. Other biological replicate is presented in Supplementary Fig. 6b. **e.** Total RNA extract from #4 x *dcl234* plants does not suppress *Pto* DC3000-induced stomatal reopening response. Col-0 leaves were treated with water or 20 ng μl^{-1} of total RNA extracts from the same genotypes used in a. and incubated with *Pto* WT for 3 hours. Stomatal aperture was measured and analyzed as described in Fig. 3a. Two other biological replicates are presented in Supplementary Fig. 6c. **f.** Small RNA species, but not the corresponding long RNA species, from IR-*CFA6*/HRPL#4 plants suppress stomatal reopening to the same extent as total RNAs extracted from these plants. The experiment was conducted as in d. but with total, long (> 200 nt) or small (< 200 nt) RNA fractions, which were separated from total RNAs of IR-*CFA6*/HRPL#4 plants. Two other biological replicates are presented in Supplementary Fig. 7c. **g.** Agarose gel picture of ethidium bromide stained *in vitro* synthesized (IVS) long and small RNA duplexes. **h.** *In vitro*-synthesized (IVS) sRNAs, but not the corresponding long dsRNAs derived from *CFA6*/HRPL chimeric construct, suppressed stomatal reopening. The experiment was conducted as in d. but with *in vitro* synthesized- (IVS), -long or -small RNA species. Long and small RNAs derived from *CYP51* were used as negative controls. Two other biological replicates are presented in Supplementary Fig. 7e. **i.** *In vitro* synthesized (IVS) antibacterial siRNAs significantly alter the transcript accumulation levels of *cfa6* and *hrpL*. Accumulation level of *cfa6* and *hrpL* transcripts was assessed by RT-qPCR analysis using *gyrA* as a control. Error bars indicate the standard deviations of values from three independent experiments. For all the experiments, statistically significant differences were assessed using ANOVA test (ns: p-value>0.05; *: p-value<0.05, **: p-value<0.01, ****: p-value < 0.0001).

Fig. 7. Anti-*hrpL* siRNAs are causal for the silencing of *Pto* DC3000 *hrpL* gene and for the suppression of stomatal reopening

a. Sequence of WT *hrpL* (99-348 nt) selected to generate the inverted repeat transgene (highlighted in yellow) was aligned with the sequence of mutated *hrpL* (mut *hrpL*) designed to contain as many silent mutations as possible in the siRNA targeted region (highlighted in grey). **b.** Unique reads mapping to *hrpL* gene of *Pto* DC3000 in the two siRNA libraries presented in Fig. 2c were used for BLAST analyses. The 500 most abundant anti-*hrpL* siRNA reads produced by IR-CFA6/HRPL#4 plants were selected and blasted against the WT *hrpL* and mut *hrpL* sequences depicted in a. The free energy of the top targets with lowest e-values for each read/target pair was calculated using RNAup and is presented here. Few siRNA-target pairs were above the blast threshold when the anti-*hrpL* siRNA reads were blasted against the mut *hrpL* sequence (right panel). Thermodynamic energy analysis of these few siRNA-target interactions revealed a mean free energy that was roughly two times higher than the one obtained for the 500 siRNA-target pairs retrieved with the WT *hrpL* sequence (left panel), supporting an impaired binding of the anti-*hrpL* siRNAs towards the mut *hrpL* sequence. **c.** Schematic representation of the *Pto* Δ *hrpL* strain along with the complementation strains generated upon transformation with the plasmids encoding WT *hrpL* or mut *hrpL*, respectively under the control of the constitutive promoter NPTII. **d.** *In vitro* AGS assay showing that the *Pto* Δ *hrpL* WT *hrpL* strain is sensitive to antibacterial RNAs while the *Pto* Δ *hrpL* mut *hrpL* is refractory to these RNA entities. *Pto* Δ *hrpL* WT *hrpL* (left panel) and *Pto* Δ *hrpL* mut *hrpL* (right panel) bacterial strains were incubated with total RNAs extracted from CV or IR-CFA6/HRPL#4 plants for 8 hours. Accumulation of WT *hrpL* and mut *hrpL* transcripts was analyzed by RT-qPCR. The mRNA levels were quantified relative to the level of *gyrA* transcripts. Error bars indicate the standard deviations of values from three independent experiments. **e.** Accumulation of anti-*cfa6* and anti-*hrpL* siRNAs was assessed by low molecular weight northern analysis using total RNA extracts from *N. benthamiana* plants transiently expressing 35S_{pro}:iR-HRPL, 35S_{pro}:iR-CFA6/HRPL and from non-transformed *N. benthamiana* leaves (Nb). U6 was used as a loading control. **f.** Both the WT *hrpL* and mut *hrpL* constructs fully complemented *Pto* Δ *hrpL* for its ability to reopen stomata and only the *Pto* Δ *hrpL* mut *hrpL* strain is refractory to anti-*hrpL* siRNA action. Col-0 leaves were treated with total RNAs extracted either from *N. benthamiana* alone or from *N. benthamiana* expressing the inverted repeat IR-HRPL and stomatal reopening response was assessed upon incubating with *Pto* Δ *hrpL* WT *hrpL* and *Pto* Δ *hrpL* mut *hrpL* bacterial strains. Stomatal reopening response was assessed as described previously. Two other biological replicates are presented in Supplementary Fig. 9a. **g.** Accumulation of anti-*hrpL* siRNAs was assessed by low molecular weight northern analysis using total RNA extracts from Arabidopsis stable transgenic lines expressing the 35S_{pro}:iR-HRPL transgene. U6 was used as a loading control. **h.** Col-0 and IR-HRPL, #1 and #4 leaves were treated with water as Mock or indicated bacterial strains for 3 hours prior to stomata aperture measurements. Stomatal reopening response was assessed as described previously. Another biological replicate is presented in Supplementary Fig. 9b. For all the stomata experiments, n = number of stomata analysed per condition. Statistical significance for all the experiments was assessed using the ANOVA test (ns: p-value>0.05; **: p-value<0.001; ****: p-value<0.0001).

Fig 8. Apoplastic fluid and apoplastic extracellular vesicles (EVs) from Arabidopsis IR-CFA6/HRPL plants trigger suppression of *Pto* DC3000-induced stomatal reopening

a. Apoplastic EVs from the Arabidopsis transgenic lines IR-CYP51 (CV) and IR-CFA6/HRPL #4 (#4) were isolated by collecting the apoplastic fluids (APF) from these plants and subjected them to ultracentrifugation at 40,000g as previously described (ref). The population of particles from the recovered P40 fractions were analysed using Nanoparticle Tracking Analysis (NTA). The number of particles, the size of particles according to their diameter (in nm), and the median size of the particles are depicted for each sample. **b.** Both the APF and the P40 fractions triggered full suppression of stomatal reopening. Stomatal aperture was measured in Col-0 leaves that were incubated for 1 hour with the above APF or P40 fractions before being inoculated with *Pto* WT for 3 hours. Results of another biological replicate is presented in Supplementary Fig 10e and 10f. For the stomata experiment, n = number of stomata analysed per condition and statistical significance was assessed using the ANOVA test (ns: p-value>0.05; ****: p-value<0.0001).

SUPPLEMENTARY INFORMATION

Plant Small RNA Species Direct Gene Silencing in Pathogenic Bacteria as well as
Disease Protection

Supplementary Fig. 1. Target prediction of sequenced anti-*cfa6* and anti-*hrpL* siRNAs in Arabidopsis and *Pto* DC3000

Unique reads mapping to *cfa6* and *hrpL* genes of *Pto* DC3000 from the two siRNA libraries presented in Fig. 2d were used for BLAST analyses. The 500 most abundant anti-*cfa6* and anti-*hrpL* siRNA reads were selected and blasted against annotated Arabidopsis genes (TAIR10) and *Pto* DC3000 coding sequences (CDS). The free energy of the top targets with lowest e-values for each read/target pair (500 pairs) was calculated using RNAup and is presented here. Thermodynamic energy analysis revealed that the free energy of siRNA-*Pto* DC3000 target interactions (Kcal/mol), which are almost exclusively composed of siRNA/*cfa6* and siRNA/*hrpL* pairs (Table S2), is significantly lower than the one from siRNA-Arabidopsis transcript interactions (Table S1), indicating that off-targets in Arabidopsis seems to be unlikely (Table S2 depicting the high e-values for all the interactions between the anti-*cfa6* and anti-*hrpL* siRNAs and Arabidopsis genes).

Supplementary Fig. 2. Accumulation level of anti-*cyp51* siRNAs in Arabidopsis IR-*CYP51* transgenic lines.

Accumulation level of anti-*cyp51A/B/C* siRNAs in Col-0 and in the transgenic T3 homozygous 35S_{pro}-IR-*CYP51*#1 and #2 plants was assessed by low molecular weight northern blot analysis. U6 was used as a loading control. IR-*CYP51*#2 was used as a control transgenic line (CV) for the assays presented in this study.

Supplementary Fig. 3. Stable expression of the IR-CFA6/HRPL inverted repeat transgene in Arabidopsis suppresses *Pto* DC3000-induced stomatal reopening
a. Other two biological replicates of Fig. 3a: The *Pto* $\Delta cfa6$ and *Pto* $\Delta hrpL$ strains, but not the *Pto* $\Delta hrpC$ strain, were impaired in their ability to reopen stomata and these phenotypes were rescued upon addition of COR. Sections of unpeeled leaves of Col-0 plants were incubated with mock solution (water) or 10^8 cfu ml $^{-1}$ of *Pto* DC3000 WT, $\Delta cfa6$, $\Delta hrpL$ or $\Delta hrpC$ strains for 3 hours. Stomatal aperture was assessed by computing the ratio of the width and length using ImageJ software. **b.** Other two biological replicates of Fig. 3b: *Pto* DC3000 WT no longer induced stomatal reopening in Arabidopsis transgenic lines overexpressing the IR-CFA6/HRPL inverted repeat transgene. Stomatal aperture measurement was conducted in Col-0 and IR-CFA6/HRPL#4, #5, #10 transgenic lines infected with *Pto* WT strain as described in a. **c.** Other two biological replicates of Fig. 3c: The *Pto* DC3000-induced stomatal reopening response was unaltered in CV compared to Col-0 plants. Stomatal aperture measurement was conducted in Col-0 and CV plants incubated with *Pto* WT strain as described in a. For all these experiments, n = number of stomata analyzed per condition and statistical significance was assessed using the ANOVA test (ns: p-value > 0.05; ****: p-value < 0.0001).

Supplementary Fig. 4. Pathogenicity phenotypes of the *Pto* DC3000 strains deleted of either *cfa6* or *hrpL*

a. The *Pto* Δ *cfa6* and *Pto* Δ *hrpL* strains are impaired in leaf apoplast multiplication in Arabidopsis. Bacterial titers in Col-0 plants dip-inoculated with *Pto* WT, *Pto* Δ *cfa6* and *Pto* Δ *hrpL* strains were monitored 2 days-post inoculation (dpi). Data from two independent replicates is presented here. **b.** The *Pto* Δ *cfa6* and *Pto* Δ *hrpL* strains are impaired in their ability to induce water-soaking symptoms when dip-inoculated on Arabidopsis Col-0 plants. Representative pictures of water-soaking symptoms were taken at 1 dpi. **c.** & **d.** The *Pto* Δ *cfa6*, *Pto* Δ *hrpL* and *Pto* Δ *hrcC* mutant strains are compromised in leaf vascular propagation. Col-0 leaves were wound-inoculated in midveins with *Pto* WT-GFP, *Pto* Δ *cfa6*-GFP, *Pto* Δ *hrpL*-GFP or *Pto* Δ *hrcC*-GFP strains. White circles indicate the sites of wound-inoculation in the midveins. To index the spreading of bacteria from the inoculation sites, GFP fluorescence was observed under UV light and pictures were taken at 3 dpi. When the bacteria propagated away from any of the three inoculation sites, it was indexed as propagation with 4 corresponding to the highest propagation index. Pictures from three biological replicates were taken into consideration and one representative picture is depicted. n = number of pictures analyzed per condition and statistical significance was assessed using the ANOVA test (****: p-value < 0.0001).

Supplementary Fig. 5. Assessing the effect of total RNAs from IR-*CFA6*/*HRPL* Arabidopsis transgenic lines applied exogenously on tomato leaves before *Pto* DC3000 infection

a. Additional biological replicate of Fig. 5d: The tomato leaves treated with CV total RNAs displayed more necrotic symptoms as compared to the leaves treated with IR-*CFA6*/*HRPL*#4 total RNAs. The experiment was conducted as in Fig 5c. but using five-week-old tomato (*Solanum lycopersicum*, cultivar 'Moneymaker') plants. Representative pictures of infected leaves in the two conditions are depicted. **b.** Additional biological replicate of Fig. 5e: A reduced number of *Pto* DC3000-GFP foci was observed in tomato leaves treated with total RNA extracts from IR-*CFA6*/*HRPL*#4 vs CV plants. Infected-leaves were observed at 3 dpi under UV light to estimate the number of GFP loci. Left panel: Dot plot representing the number of GFP loci analyzed using ImageJ software from 3-4 different leaves per condition with at least 4 pictures per leaf. The values used for the analysis are from two different independent experiments. Right panel: Representative picture of the tomato leaves described in Fig 5d. **c.** Additional biological replicate of Fig. 5f: *Pto* WT-GFP DNA content is decreased in tomato leaves treated with total RNA extracts from IR-*CFA6*/*HRPL*#4 vs CV plants. The level of bacterial DNA content was analyzed by qPCR using tomato *Ubiquitin* gene as a control. For b and c, student's t-test was performed for the comparative analysis (ns: p-value>0.05; **, p-value<0.01, ***, p-value<0.001).

Supplementary Fig. 6. Assessing the effects of exogenously applied total RNAs from different genotypes on stomata reopening events

a. Other two biological replicates of Fig. 5b: The ability of *Pto* WT to reopen stomata was altered upon exogenous application of total RNA extracts from IR-*CFA6*/*HRPL* plants compared to CV plants. Col-0 leaves were treated for 1 hour with water (Mock) or 20 ng μl^{-1} of total RNAs extracted from CV or IR-*CFA6*/*HRPL* plants and were incubated with *Pto* WT for 3 hours. Stomatal aperture was measured and analyzed as described previously. **b.** Another biological replicates of Fig. 6d: Accumulation level of anti-*cfa6* and anti-*hrpL* siRNAs in the indicated genotypes along with Col-0 plants incubated with total RNAs derived from the same genotypes for 1 hour was assessed by low molecular weight northern blot analysis. U6 was used as a loading control. **c.** Other two biological replicates of Fig. 6e: Total RNA extract from #4 x *dcl234* plants does not suppress *Pto* DC3000-induced stomatal reopening response. Col-0 leaves were treated with water or 20 ng μl^{-1} of total RNA extracts from the same genotypes used in a. and incubated with *Pto* WT for 3 hours. Stomatal aperture was measured and analyzed as described previously. For all the stomata experiments, n = number of stomata analyzed per condition and statistical significance was assessed using the ANOVA test (ns: p-value > 0.05; ****: p-value < 0.0001).

Fig. S7. Characterization of the long and small RNA fractions separated from IR-CFA6/HRPL#4 total RNAs or *in vitro* synthesized (IVS)

a. Size distribution of total, long and small RNAs from IR-CFA6/HRPL#4 plants was determined with an agilent Bioanalyzer 2100 equipped with an RNA Nano chip. Low molecular weight RNA fractions are encircled for each sample. 18S and 25S ribosomal peaks are highlighted. **b.** Agarose gel picture of ethidium bromide stained total, long and small RNAs used in a. **c.** Other two biological replicates of Fig. 6f: Small RNA species, but not the corresponding long RNA species, from IR-CFA6/HRPL#4 plants suppress stomatal reopening to the same extent as total RNAs extracted from these plants. The experiment was conducted as in Fig. 6d. but with total, long (> 200 nt) or small (< 200 nt) RNA fractions, which were separated from total RNAs of IR-CFA6/HRPL#4 plants. **d.** Other two biological replicates of Fig. 6g: *In vitro* synthesized (IVS) sRNAs, but not the corresponding long dsRNAs derived from CFA6/HRPL chimeric construct, suppressed stomatal reopening. The experiment was conducted as in Fig. 6d. but with *in vitro* synthesized (IVS), long or small RNA species. Long and small RNAs derived from CYP51 were used as negative controls. For all the stomata experiments, n = number of stomata analyzed per condition. For all the experiments, statistically significant differences were assessed using ANOVA test (ns: p-value>0.05; *: p-value<0.05, **: p-value<0.01, ****: p-value<0.0001).

Supplementary Fig. 8. *N. benthamiana* total RNAs containing anti-*hrpL* siRNAs trigger suppression of *Pto* DC3000-induced stomatal reopening
 Col-0 leaves were treated with total RNAs extracted either from *N. benthamiana* alone or from *N. benthamiana* expressing the inverted repeat IR-*CFA6/HRPL* or IR-*HRPL*. Stomatal reopening response was assessed by incubating Col-0 leaves with specified total RNAs before incubating with *Pto* DC3000 as described previously. Two biological replicates are presented here.

Supplementary Fig. 9. Both exogenously applied and plant-encoded anti-*hrpL* siRNAs are causal for the the suppression of stomatal reopening events

a. & b. Other two biological replicates of Fig. 7f. Both the WT *hrpL* and mut *hrpL* constructs fully complemented *Pto* $\Delta hrpL$ for its ability to reopen stomata and only the *Pto* $\Delta hrpL$ mut *hrpL* strain is refractory to anti-*hrpL* siRNA action. Col-0 leaves were treated or not with total RNAs extracted either from *N. benthamiana* alone or from *N. benthamiana* expressing the inverted repeat IR-HRPL and stomatal reopening response was assessed upon incubating with *Pto* $\Delta hrpL$ WT *hrpL* and *Pto* $\Delta hrpL$ mut *hrpL* bacterial strains for 3 hours. Stomatal reopening response was assessed as described previously. **b.** Other two biological replicate of Fig. 7h: Col-0 and IR-HRPL#1 and #4 leaves were treated with water as Mock or indicated bacterial strains for 3 hours prior to stomata aperture measurements. Stomatal reopening response was assessed as described previously. For all the experiments, statistically significant differences were assessed using ANOVA test (ns: p-value>0.05; ****: p-value<0.0001).

Fig. S10. Apoplastic fluid and P40 vesicles isolated from Arabidopsis IR-CFA6/HRPL leaves trigger suppression of *Pto* DC3000-induced stomatal reopening

a. & c. Apoplastic EVs from the Arabidopsis transgenic lines IR-CYP51 (CV) and IR-CFA6/HRPL#4 (#4) were isolated by collecting the apoplastic fluids (APF) from these plants and analysed using Nanoparticle Tracking Analysis (NTA). The number of particles, the size of particles according to their diameter (in nm), and the median size of the particles are depicted for each sample from two independent replicates. **b. & d.** The ability of *Pto* WT to reopen stomata was altered to similar levels upon exogenous application of APF as compared to total RNAs derived from IR-CFA6/HRPL#4 plants. Stomata aperture was measured in Col-0 leaves incubated for 1 hour with water (Mock) or total RNAs (20 ng μl^{-1}) or APF extracted from CV or IR-CFA6/HRPL#4 transgenic lines before being incubated for 3 hours with *Pto* WT bacteria. Stomatal aperture was measured and analyzed as described previously. **e.** Additional biological replicate from Fig. 8a: Apoplastic EVs from the Arabidopsis transgenic lines IR-CYP51 (CV) and IR-CFA6/HRPL#4 (#4) were isolated by collecting the apoplastic fluids (APF) and subjected them to ultracentrifugation at 40,000g (P40) as previously described (ref). The population of particles from the recovered P40 fractions were analysed using Nanoparticle Tracking Analysis (NTA). The number of particles, the size of particles according to their diameter (in nm), and the median size of the particles are depicted for each sample. **f.** Additional biological replicate from Fig. 8b: Both the APF and the P40 fractions triggered full suppression of stomatal reopening. Stomatal aperture was measured in Col-0 leaves that were incubated for 1 hour with the above APF or P40 fractions before being inoculated with *Pto* WT for 3 hours. Stomatal aperture was measured and analyzed as described previously. For all the stomata experiments, n = number of stomata analyzed per condition and statistical significance was assessed using the ANOVA test (ns: p-value>0.05; ****: p-value<0.0001).

Supplementary Table legend

Supplementary Table 1. Targets of 500 most abundant siRNAs specific to *cfa6* or *hrpL* derived from IR-*CFA6/HRPL* in the *Pto* DC3000 genome

Supplementary Table 2. Targets of 500 most abundant siRNAs specific to *cfa6* or *hrpL* derived from IR-*CFA6/HRPL* in the *Arabidopsis thaliana* Col-0 genome

Supplementary Table 3. Targets of 500 most abundant anti-*hrpL* siRNAs derived from IR-*CFA6/HRPL* in the WT or mutated *hrpL* versions

Supplementary Table 4. List of primer sequences used in this study

sRNA_source	Genome ID	Targeted.by. (Unique reads)	Targeted.by (Total reads)	lowest_e_value	locus	description
cfa6	Pst DC3000 NC_004578.1_cds_NP_794435.1_4579	450	7075728	0,0000258	gene=cfa6	coronafacic acid polyketide synthase I
cfa6	Pst DC3000 NC_004578.1_cds_NP_790362.1_499	15	720356	0,17	gene=pqqF	coenzyme PQQ synthesis protein F
cfa6	Pst DC3000 NC_004578.1_cds_NP_791928.1_2070	15	117477	0,17	locus_tag=PSPTO_2105	thiamine biosynthesis lipoprotein
cfa6	Pst DC3000 NC_004578.1_cds_NP_791231.1_1369	7	77391	0,057	gene=hrpL	RNA polymerase sigma factor HrpL
cfa6	Pst DC3000 NC_004578.1_cds_NP_790641.1_777	3	68426	0,51	locus_tag=PSPTO_0794	hypothetical protein
cfa6	Pst DC3000 NC_004578.1_cds_NP_795316.1_5464	2	10724	1,5	gene=atpC	F0F1 ATP synthase subunit epsilon
cfa6	Pst DC3000 NC_004578.1_cds_NP_794924.1_5072	2	8838	1,5	locus_tag=PSPTO_5193	RND family efflux transporter MFP subunit
cfa6	Pst DC3000 NC_004578.1_cds_NP_791957.1_2100	1	7949	1,5	gene=pvsA	pyoverdine chromophore precursor synthetase
cfa6	Pst DC3000 NC_004578.1_cds_NP_791186.1_1324	2	3478	0,51	locus_tag=PSPTO_1359	ABC transporter permease
cfa6	Pst DC3000 NC_004578.1_cds_NP_791667.1_1809	2	2781	1,5	locus_tag=PSPTO_1843	aspartate kinase
cfa6	Pst DC3000 NC_004578.1_cds_NP_793378.1_3523	1	1548	0,51	locus_tag=PSPTO_3599	hypothetical protein
hrpL	Pst DC3000 NC_004578.1_cds_NP_791231.1_1369	451	6698293	0,0000258	gene=hrpL	RNA polymerase sigma factor HrpL
hrpL	Pst DC3000 NC_004578.1_cds_NP_794435.1_4579	4	11897	0,019	gene=cfa6	coronafacic acid polyketide synthase I
hrpL	Pst DC3000 NC_004578.1_cds_NP_793956.1_4100	1	1715	0,51	locus_tag=PSPTO_4195	hypothetical protein
hrpL	Pst DC3000 NC_004578.1_cds_NP_793436.1_3579	1	1641	1,5	locus_tag=PSPTO_3662	xanthine dehydrogenase accessory factor XdhC

sRNA_source	Genome	ID	Targeted.by. (Unique reads)	Targeted.by (Total reads)	lowest_e_value	locus	description
cfa6	Ath TAIR10	NC_003076.8_cds_NP_001032042.1_44625	41	2028283		1,9 gene=OVA2	tRNA synthetase class I (I, L, M and V) family protein
cfa6	Ath TAIR10	NC_003076.8_cds_NP_197110.1_39886	14	1236028		5,7 locus_tag=AT5G16060	Cytochrome c oxidase biogenesis protein Cmc1-like protein
cfa6	Ath TAIR10	NC_003070.9_cds_NP_563804.4_1481	30	946535		5,7 gene=TPST	tyrosylprotein sulfotransferase
cfa6	Ath TAIR10	NC_003076.8_cds_NP_001032057.1_45070	2	637167		17 gene=LPA2	low psii accumulation2
cfa6	Ath TAIR10	NC_003071.7_cds_NP_001031385.1_15060	16	278379		5,7 locus_tag=AT2G21300	ATP binding microtubule motor family protein
cfa6	Ath TAIR10	NC_003074.8_cds_NP_190127.1_26372	10	197128		5,7 locus_tag=AT3G45410	Concanavalin A-like lectin protein kinase family protein
cfa6	Ath TAIR10	NC_003075.7_cds_NP_192119.2_30164	11	186313		17 locus_tag=AT4G02100	Heat shock protein DnaJ with tetratricopeptide repeat-containing protein
cfa6	Ath TAIR10	NC_003076.8_cds_NP_974885.1_43802	23	175169		1,9 gene=OSB3	organellar single-stranded DNA binding protein 3
cfa6	Ath TAIR10	NC_003070.9_cds_NP_171667.1_137	20	174998		5,7 gene=GPAT4	glycerol-3-phosphate acyltransferase 4
cfa6	Ath TAIR10	NC_003070.9_cds_NP_001320772.1_8776	12	158983		17 gene=ARL2	ARG1-like 2
cfa6	Ath TAIR10	NC_003076.8_cds_NP_001190462.1_43500	14	145895		17 gene=PHT1;2	phosphate transporter 2
cfa6	Ath TAIR10	NC_003074.8_cds_NP_191458.1_28849	6	116106		17 gene=IPMI1	isopropylmalate isomerase 1
cfa6	Ath TAIR10	NC_037304.1_cds_YP_009472115.2_48166	12	86972		1,9 gene=mttB	transport membrane protein
cfa6	Ath TAIR10	NC_003070.9_cds_NP_173075.3_3102	4	86230		5,7 locus_tag=AT1G16250	Galactose oxidase/kelch repeat superfamily protein
cfa6	Ath TAIR10	NC_003071.7_cds_NP_179907.1_15421	4	69276		17 gene=UGT84B1	UDP-glucosyl transferase 84B1
cfa6	Ath TAIR10	NC_003074.8_cds_NP_001030896.1_29006	13	68649		5,7 gene=SAC9	sacI homology domain-containing protein / VW domain-containing protein
cfa6	Ath TAIR10	NC_003070.9_cds_NP_849898.1_11877	11	67561		1,9 locus_tag=AT1G77122	Uncharacterized protein family UPF0090
cfa6	Ath TAIR10	NC_003074.8_cds_NP_188618.3_23966	19	59612		1,9 gene=PRP40C	pre-mRNA-processing protein 40C
cfa6	Ath TAIR10	NC_003071.7_cds_NP_001324342.1_16771	9	56478		5,7 locus_tag=AT2G30480	hypothetical protein
cfa6	Ath TAIR10	NC_003076.8_cds_NP_196827.1_39354	8	55911		17 locus_tag=AT5G13230	Tetratricopeptide repeat (TPR)-like superfamily protein
cfa6	Ath TAIR10	NC_003075.7_cds_NP_001328069.1_35815	2	50735		5,7 gene=EDA39	calmodulin-binding family protein
cfa6	Ath TAIR10	NC_003074.8_cds_NP_191259.1_28493	4	47805		17 locus_tag=AT3G57000	nucleolar essential protein-like protein
cfa6	Ath TAIR10	NC_003074.8_cds_NP_191274.1_28522	4	45371		5,7 gene=NAP57	homologue of NAP57
cfa6	Ath TAIR10	NC_003076.8_cds_NP_198474.1_42307	9	40224		17 gene=PP2C74	Protein phosphatase 2C family protein
cfa6	Ath TAIR10	NC_003074.8_cds_NP_001118685.1_24672	5	37320		5,7 gene=HGL1	heteroglycan glucosidase 1
cfa6	Ath TAIR10	NC_003070.9_cds_NP_177125.3_10577	3	34049		17 gene=CUL3B	cullin 3B
cfa6	Ath TAIR10	NC_003076.8_cds_NP_001332094.1_43015	6	25828		5,7 gene=SDH2-2	succinate dehydrogenase 2-2
cfa6	Ath TAIR10	NC_003070.9_cds_NP_172353.1_1631	2	24103		5,7 locus_tag=AT1G08760	GPI-anchored adhesin-like protein, putative (DUF936)
cfa6	Ath TAIR10	NC_003075.7_cds_NP_193283.2_32248	4	23034		17 gene=UGT84A1	UDP-Glycosyltransferase superfamily protein
cfa6	Ath TAIR10	NC_003076.8_cds_NP_201271.1_47530	4	22359		17 gene=CMPG2	CYS, MET, PRO, and GLY protein 2
cfa6	Ath TAIR10	NC_003075.7_cds_NP_001328078.1_34834	6	20963		17 locus_tag=AT4G28150	hypothetical protein (DUF789)
cfa6	Ath TAIR10	NC_003076.8_cds_NP_680446.1_46045	7	18650		17 locus_tag=AT5G56890	Protein kinase superfamily protein
cfa6	Ath TAIR10	NC_003076.8_cds_NP_195740.1_37189	3	18617		17 locus_tag=AT5G01200	Duplicated homeodomain-like superfamily protein
cfa6	Ath TAIR10	NC_003074.8_cds_NP_191052.2_28089	6	18268		1,9 gene=PMR6	Pectin lyase-like superfamily protein
cfa6	Ath TAIR10	NC_003076.8_cds_NP_196750.1_39181	4	17244		5,7 gene=EMB3135	harpin-induced protein
cfa6	Ath TAIR10	NC_003075.7_cds_NP_192855.1_31308	2	15932		17 locus_tag=AT4G11170	Disease resistance protein (TIR-NBS-LRR class) family
cfa6	Ath TAIR10	NC_003071.7_cds_NP_973476.1_14262	2	14056		5,7 locus_tag=AT2G16980	Major facilitator superfamily protein
cfa6	Ath TAIR10	NC_003074.8_cds_NP_187514.1_21808	6	13338		17 locus_tag=AT3G09020	alpha 1,4-glycosyltransferase family protein
cfa6	Ath TAIR10	NC_003075.7_cds_NP_194585.1_34910	5	12910		17 gene=RIC7	ROP-interactive CRIB motif-containing protein 7
cfa6	Ath TAIR10	NC_003071.7_cds_NP_178535.3_13300	2	11429		17 gene=LpxB	transferases, transferring glycosyl groups
cfa6	Ath TAIR10	NC_003074.8_cds_NP_188705.1_24146	2	11358		17 locus_tag=AT3G20690	F-box and associated interaction domains-containing protein
cfa6	Ath TAIR10	NC_003074.8_cds_NP_001078165.1_23419	2	8555		5,7 gene=LPD1	lipoamide dehydrogenase 1
cfa6	Ath TAIR10	NC_003070.9_cds_NP_178014.2_12198	2	8335		17 locus_tag=AT1G78930	Mitochondrial transcription termination factor family protein
cfa6	Ath TAIR10	NC_003076.8_cds_NP_196118.1_37890	2	8327		17 gene=SUN1	SAD1/UNC-84 domain protein 1
cfa6	Ath TAIR10	NC_003071.7_cds_NP_181050.1_17673	2	8258		17 locus_tag=AT2G35050	kinase superfamily with octicosapeptide/Phox/Bem1p domain-containing protein
cfa6	Ath TAIR10	NC_003074.8_cds_NP_001325857.1_24967	2	7883		17 locus_tag=AT3G25430	Polynucleotidyl transferase, ribonuclease H-like superfamily protein
cfa6	Ath TAIR10	NC_003074.8_cds_NP_566817.1_25386	1	7762		17 locus_tag=AT3G27420	bromodomain testis-specific protein
cfa6	Ath TAIR10	NC_003076.8_cds_NP_001190624.1_47754	1	7529		17 gene=BAM1	Leucine-rich receptor-like protein kinase family protein
cfa6	Ath TAIR10	NC_003074.8_cds_NP_190058.1_26252	2	7112		1,9 gene=ADT4	arogenate dehydratase 4
cfa6	Ath TAIR10	NC_003074.8_cds_NP_188938.1_24538	2	6927		1,9 locus_tag=AT3G22980	Ribosomal protein S5/Elongation factor G/III/V family protein
cfa6	Ath TAIR10	NC_003070.9_cds_NP_001321840.1_4928	4	6811		5,7 gene=NRT1.6	nitrate transporter 1.6
cfa6	Ath TAIR10	NC_003076.8_cds_NP_196331.2_38305	2	6788		17 locus_tag=AT5G07140	Protein kinase superfamily protein
cfa6	Ath TAIR10	NC_003076.8_cds_NP_201215.1_47418	4	6654		1,9 locus_tag=AT5G64100	Peroxidase superfamily protein
cfa6	Ath TAIR10	NC_003074.8_cds_NP_001189846.1_21882	2	6588		17 gene=PLL3	pol-like 3
cfa6	Ath TAIR10	NC_003074.8_cds_NP_189335.2_25302	2	6516		17 locus_tag=AT3G26990	ENTH/VHS family protein
cfa6	Ath TAIR10	NC_003070.9_cds_NP_849610.2_1509	1	6269		5,7 gene=CHX6B	cation/H ⁺ exchanger 6B
cfa6	Ath TAIR10	NC_003070.9_cds_NP_849573.1_124	2	5042		5,7 locus_tag=AT1G01540	Protein kinase superfamily protein

cfa6	Ath TAIR10 NC_003076.8_cds_NP_199754.2_44698	3	5007	17 locus_tag=AT5G49430 WD40/YVTN repeat and Bromo-WDR9-I-like domain-containing protein
cfa6	Ath TAIR10 NC_003074.8_cds_NP_001190123.1_28699	2	4966	17 locus_tag=AT3G58110 hypothetical protein
cfa6	Ath TAIR10 NC_003076.8_cds_NP_200458.1_45976	2	4921	17 gene=GulLO7 FAD-dependent oxidoreductase family protein
cfa6	Ath TAIR10 NC_003074.8_cds_NP_187718.1_22205	1	4920	17 locus_tag=AT3G11070 Outer membrane OMP85 family protein
cfa6	Ath TAIR10 NC_003070.9_cds_NP_564584.1_7408	2	4854	17 gene=UCN Protein kinase superfamily protein
cfa6	Ath TAIR10 NC_003071.7_cds_NP_180929.1_17431	2	4600	17 locus_tag=AT2G33730 P-loop containing nucleoside triphosphate hydrolases superfamily protein
cfa6	Ath TAIR10 NC_003074.8_cds_NP_188014.1_22820	2	4232	17 locus_tag=AT3G13980 SKI/DACH domain protein
cfa6	Ath TAIR10 NC_003074.8_cds_NP_566579.1_23509	2	4095	17 gene=RPL3P ribosomal protein L3 plastid
cfa6	Ath TAIR10 NC_003076.8_cds_NP_196567.2_38816	2	3861	5,7 locus_tag=AT5G10050 NAD(P)-binding Rossmann-fold superfamily protein
cfa6	Ath TAIR10 NC_003074.8_cds_NP_566381.1_22144	2	3613	17 locus_tag=AT3G10770 Single-stranded nucleic acid binding R3H protein
cfa6	Ath TAIR10 NC_003075.7_cds_NP_001328421.1_34712	2	3478	17 locus_tag=AT4G27595 WEB family protein (DUF827)
cfa6	Ath TAIR10 NC_003071.7_cds_NP_179618.1_14823	2	3411	5,7 locus_tag=AT2G20280 Zinc finger C-x8-C-x5-C-x3-H type family protein
cfa6	Ath TAIR10 NC_003074.8_cds_NP_001189932.1_23836	2	3310	17 gene=PREP1 presequence protease 1
cfa6	Ath TAIR10 NC_003075.7_cds_NP_567998.3_36468	2	3209	5,7 locus_tag=AT4G36190 Serine carboxypeptidase S28 family protein
cfa6	Ath TAIR10 NC_003076.8_cds_NP_200190.1_45491	2	3063	5,7 locus_tag=AT5G53790 F-box protein, putative (DUF295)
cfa6	Ath TAIR10 NC_003075.7_cds_NP_193070.1_31729	1	2856	17 gene=LRX3 Leucine-rich repeat (LRR) family protein
cfa6	Ath TAIR10 NC_003075.7_cds_NP_195043.1_35837	2	2854	1,9 locus_tag=AT4G33170 Tetratricopeptide repeat (TPR)-like superfamily protein
cfa6	Ath TAIR10 NC_003076.8_cds_NP_200667.2_46366	2	2781	17 gene=RANBP1 RAN binding protein 1
cfa6	Ath TAIR10 NC_003075.7_cds_NP_001327989.1_35860	2	2608	5,7 locus_tag=AT4G33280 AP2/B3-like transcriptional factor family protein
cfa6	Ath TAIR10 NC_003076.8_cds_NP_568893.1_46438	1	1309	5,7 locus_tag=AT5G58950 Protein kinase superfamily protein
hrpL	Ath TAIR10 NC_003070.9_cds_NP_171980.1_780	4	976729	5,7 locus_tag=AT1G04880 HMG (high mobility group) box protein with ARID/BRIGHT DNA-binding domain-containing protein
hrpL	Ath TAIR10 NC_003070.9_cds_NP_001319035.1_3550	6	829463	5,7 locus_tag=AT1G18660 zinc finger (C3HC4-type RING finger) family protein
hrpL	Ath TAIR10 NC_003076.8_cds_NP_198624.1_42592	11	688488	5,7 locus_tag=AT5G38080 transmembrane protein
hrpL	Ath TAIR10 NC_003076.8_cds_NP_001331990.1_46021	10	397983	17 gene=HAG2 histone acetyltransferase of the GNAT family 2
hrpL	Ath TAIR10 NC_003076.8_cds_NP_198101.2_41844	21	344917	1,9 locus_tag=AT5G27490 Integral membrane Yip1 family protein
hrpL	Ath TAIR10 NC_003074.8_cds_NP_001326732.1_27241	9	253780	5,7 locus_tag=AT3G50380 vacuolar protein sorting-associated protein, putative (DUF1162)
hrpL	Ath TAIR10 NC_003074.8_cds_NP_188706.1_24147	8	200098	1,9 locus_tag=AT3G20700 F-box associated ubiquitination effector family protein
hrpL	Ath TAIR10 NC_003074.8_cds_NP_187423.2_21619	8	175936	0,071 locus_tag=AT3G07660 flocculation protein (DUF1296)
hrpL	Ath TAIR10 NC_003076.8_cds_NP_200787.1_46589	3	129003	17 locus_tag=AT5G59790 UPSTREAM OF FLC protein (DUF966)
hrpL	Ath TAIR10 NC_003070.9_cds_NP_001320578.1_12238	8	112138	17 locus_tag=AT1G79103 hypothetical protein
hrpL	Ath TAIR10 NC_003074.8_cds_NP_191318.4_28609	14	108905	5,7 locus_tag=AT3G57590 F-box and associated interaction domains-containing protein
hrpL	Ath TAIR10 NC_003071.7_cds_NP_565351.1_13749	1	105824	17 gene=CRCK3 calmodulin-binding receptor-like cytoplasmic kinase 3
hrpL	Ath TAIR10 NC_003076.8_cds_NP_196819.1_39336	14	102855	1,9 gene=EXO70C1 exocyst subunit exo70 family protein C1
hrpL	Ath TAIR10 NC_003076.8_cds_NP_197841.1_41358	9	100643	5,7 gene=DMR6 2-oxoglutarate (2OG) and Fe(II)-dependent oxygenase superfamily protein
hrpL	Ath TAIR10 NC_003074.8_cds_NP_001325725.1_22115	6	96366	1,9 gene=CAT7 cationic amino acid transporter 7
hrpL	Ath TAIR10 NC_003071.7_cds_NP_973476.1_14262	6	87960	5,7 locus_tag=AT2G16980 Major facilitator superfamily protein
hrpL	Ath TAIR10 NC_003076.8_cds_NP_001119157.1_37478	7	85801	5,7 gene=GSTL1 glutathione transferase lambda 1
hrpL	Ath TAIR10 NC_003071.7_cds_NP_001118273.1_13417	2	83985	5,7 gene=GRP3S glycine-rich protein 3 short isoform
hrpL	Ath TAIR10 NC_003076.8_cds_NP_199757.1_44702	4	83790	17 gene=ACLB-2 ATP citrate lyase subunit B 2
hrpL	Ath TAIR10 NC_003075.7_cds_NP_195464.2_36670	7	75027	5,7 locus_tag=AT4G37480 Chaperone DnaJ-domain superfamily protein
hrpL	Ath TAIR10 NC_003075.7_cds_NP_193506.2_32749	12	71605	1,9 gene=HDG4 homeodomain GLABROUS 4
hrpL	Ath TAIR10 NC_003074.8_cds_NP_001326496.1_22571	5	62061	5,7 gene=HAC5 histone acetyltransferase of the CBP family 5
hrpL	Ath TAIR10 NC_003076.8_cds_NP_201169.1_47342	6	57212	17 locus_tag=AT5G63640 ENTH/VHS/GAT family protein
hrpL	Ath TAIR10 NC_003071.7_cds_NP_001324812.1_18586	9	55676	5,7 gene=DAR2 DA1-related protein 2
hrpL	Ath TAIR10 NC_003076.8_cds_NP_001330670.1_43329	5	54997	17 gene=SDG25 SET domain protein 25
hrpL	Ath TAIR10 NC_003076.8_cds_NP_200784.2_46584	4	50514	17 locus_tag=AT5G59760 hypothetical protein (DUF1635)
hrpL	Ath TAIR10 NC_003071.7_cds_NP_181704.1_18998	2	46678	5,7 locus_tag=AT2G41730 calcium-binding site protein
hrpL	Ath TAIR10 NC_003075.7_cds_NP_567330.1_30812	5	44978	5,7 gene=ACS11 1-aminocyclopropane-1-carboxylate synthase 11
hrpL	Ath TAIR10 NC_003074.8_cds_NP_567118.1_29406	6	44216	5,7 locus_tag=AT3G61790 Protein with RING/U-box and TRAF-like domain
hrpL	Ath TAIR10 NC_003074.8_cds_NP_001327831.1_20318	6	39036	1,9 locus_tag=AT3G01310 Phosphoglycerate mutase-like family protein
hrpL	Ath TAIR10 NC_003070.9_cds_NP_001321735.1_3199	3	37902	5,7 gene=HAC12 histone acetyltransferase of the CBP family 12
hrpL	Ath TAIR10 NC_003076.8_cds_NP_200382.1_45830	3	37854	17 gene=Tic20-V TIC 20-v-like protein
hrpL	Ath TAIR10 NC_003074.8_cds_NP_001189790.1_20428	5	37489	17 locus_tag=AT3G01810 EEIG1/EHBP1 protein amino-terminal domain protein
hrpL	Ath TAIR10 NC_003076.8_cds_NP_001332517.1_44748	5	31145	1,9 gene=FRO6 ferric reduction oxidase 6
hrpL	Ath TAIR10 NC_003076.8_cds_NP_001330525.1_42948	9	26051	1,9 gene=MYB24 myb domain protein 24
hrpL	Ath TAIR10 NC_003076.8_cds_NP_199253.1_43717	4	25564	17 locus_tag=AT5G44400 FAD-binding Berberine family protein
hrpL	Ath TAIR10 NC_003070.9_cds_NP_173089.1_3129	6	24115	1,9 gene=OCT3 organic cation/carnitine transporter 3
hrpL	Ath TAIR10 NC_003076.8_cds_NP_198103.1_41847	7	22749	1,9 locus_tag=AT5G27510 Protein kinase superfamily protein

hrpL	Ath TAIR10	NC_003076.8_cds_NP_201234.1_47447	4	22704	5,7 gene=DIT2.1	dicarboxylate transport 2.1
hrpL	Ath TAIR10	NC_003076.8_cds_NP_197761.2_41187	2	22682	17 gene=PHS1	dual specificity protein phosphatase family protein
hrpL	Ath TAIR10	NC_003070.9_cds_NP_563757.1_1061	2	21619	5,7 locus_tag=AT1G06050	ENHANCED DISEASE RESISTANCE-like protein (DUF1336)
hrpL	Ath TAIR10	NC_003076.8_cds_NP_001331276.1_45111	2	19898	17 gene=AHG1	Protein phosphatase 2C family protein
hrpL	Ath TAIR10	NC_003074.8_cds_NP_191310.1_28593	2	19242	5,7 gene=ADPG1	Pectin lyase-like superfamily protein
hrpL	Ath TAIR10	NC_003071.7_cds_NP_973714.1_20207	5	17874	1,9 locus_tag=AT2G47970	Nuclear pore localization protein NPL4
hrpL	Ath TAIR10	NC_003070.9_cds_NP_564401.1_5865	1	17376	5,7 locus_tag=AT1G32415	pentatricopeptide (PPR) repeat-containing protein
hrpL	Ath TAIR10	NC_003075.7_cds_NP_001031658.1_32777	2	17272	5,7 gene=AGD8	ARF-GAP domain 8
hrpL	Ath TAIR10	NC_003076.8_cds_NP_199575.1_44334	2	16947	5,7 gene=NF-YB2	nuclear factor Y, subunit B2
hrpL	Ath TAIR10	NC_003076.8_cds_NP_196554.1_38789	4	16506	5,7 gene=NRPB4	RNA polymerase II, Rpb4, core protein
hrpL	Ath TAIR10	NC_003070.9_cds_NP_177267.2_10841	4	15931	5,7 locus_tag=AT1G71110	transmembrane protein
hrpL	Ath TAIR10	NC_003075.7_cds_NP_192590.1_30881	4	15878	5,7 gene=MEKK1	MAPK/ERK kinase kinase 1
hrpL	Ath TAIR10	NC_003070.9_cds_NP_565212.1_12310	4	15850	0,64 gene=FTSH12	FTSH protease 12
hrpL	Ath TAIR10	NC_003074.8_cds_NP_001030659.1_21618	7	15616	1,9 gene=COL9	CONSTANS-like 9
hrpL	Ath TAIR10	NC_003076.8_cds_NP_974747.1_38321	4	15454	17 locus_tag=AT5G07225	RING/U-box superfamily protein
hrpL	Ath TAIR10	NC_003070.9_cds_NP_176638.1_9648	2	14842	0,64 gene=DUO3	Homeodomain-like superfamily protein
hrpL	Ath TAIR10	NC_003076.8_cds_NP_200359.1_45771	1	14745	17 gene=SVL1	SHV3-like 1
hrpL	Ath TAIR10	NC_003070.9_cds_NP_001322007.1_1492	6	13961	5,7 gene=MOM	ATP-dependent helicase family protein
hrpL	Ath TAIR10	NC_003076.8_cds_NP_568718.1_44790	1	13773	17 gene=AFB5	auxin F-box protein 5
hrpL	Ath TAIR10	NC_003076.8_cds_NP_001332094.1_43015	4	11897	5,7 gene=SDH2-2	succinate dehydrogenase 2-2
hrpL	Ath TAIR10	NC_003076.8_cds_NP_197483.1_40559	4	11891	17 gene=emb2734	ARM repeat superfamily protein
hrpL	Ath TAIR10	NC_003074.8_cds_NP_191069.2_28130	4	11879	5,7 gene=ABCG16	ABC-2 type transporter family protein
hrpL	Ath TAIR10	NC_003076.8_cds_NP_001331299.1_41301	2	11792	1,9 locus_tag=AT5G24275	En/Spm-like transposon
hrpL	Ath TAIR10	NC_003074.8_cds_NP_191697.1_29312	2	11623	5,7 gene=TRM14	Phosphatidylinositol N-acetylglucosaminyltransferase subunit P-like protein
hrpL	Ath TAIR10	NC_003070.9_cds_NP_001031225.1_9550	3	11566	17 locus_tag=AT1G63980	D111/G-patch domain-containing protein
hrpL	Ath TAIR10	NC_003076.8_cds_NP_568958.1_47160	3	11047	1,9 locus_tag=AT5G62650	Tic22-like family protein
hrpL	Ath TAIR10	NC_003074.8_cds_NP_187170.2_21094	3	10716	1,9 locus_tag=AT3G05190	D-aminoacid aminotransferase-like PLP-dependent enzymes superfamily protein
hrpL	Ath TAIR10	NC_003076.8_cds_NP_568711.1_44721	2	10321	1,9 gene=BL0S2	biogenesis of lysosome organelles complex 1 subunit-like protein
hrpL	Ath TAIR10	NC_003076.8_cds_NP_197318.1_40244	4	10309	17 gene=GDH1	glutamate dehydrogenase 1
hrpL	Ath TAIR10	NC_003071.7_cds_NP_001324747.1_13061	2	9085	5,7 locus_tag=AT2G03140	alpha/beta-Hydrolases superfamily protein
hrpL	Ath TAIR10	NC_003070.9_cds_NP_001322409.1_5083	4	9073	1,9 locus_tag=AT1G27900	RNA helicase family protein
hrpL	Ath TAIR10	NC_003071.7_cds_NP_001031477.1_17537	1	8951	17 locus_tag=AT2G34300	S-adenosyl-L-methionine-dependent methyltransferases superfamily protein
hrpL	Ath TAIR10	NC_003076.8_cds_NP_001154791.1_47159	2	8875	5,7 gene=ELF5	proline-rich family protein
hrpL	Ath TAIR10	NC_003076.8_cds_NP_199956.1_45049	2	8729	5,7 gene=EYE	conserved oligomeric Golgi complex component-related / COG complex component-like protein
hrpL	Ath TAIR10	NC_003075.7_cds_NP_567467.1_32247	2	8652	17 locus_tag=AT4G15475	F-box/RNI-like superfamily protein
hrpL	Ath TAIR10	NC_003070.9_cds_NP_174229.2_5312	2	8503	17 locus_tag=AT1G29350	RNA polymerase II degradation factor-like protein (DUF1296)
hrpL	Ath TAIR10	NC_003070.9_cds_NP_174122.1_5100	2	7703	5,7 gene=ABCB14	P-glycoprotein 14
hrpL	Ath TAIR10	NC_003071.7_cds_NP_179627.2_14840	2	7408	0,64 gene=MUR3	Exostosin family protein
hrpL	Ath TAIR10	NC_003075.7_cds_NP_001031681.1_33300	3	7198	5,7 gene=JMJ14	JUMONJI 14
hrpL	Ath TAIR10	NC_003075.7_cds_NP_194594.1_34924	2	7192	5,7 locus_tag=AT4G28650	Leucine-rich repeat transmembrane protein kinase family protein
hrpL	Ath TAIR10	NC_003076.8_cds_NP_001332397.1_40786	2	7176	17 gene=B73	molybdopterin biosynthesis CNX1 protein / molybdenum cofactor biosynthesis enzyme CNX1 (CNX1)
hrpL	Ath TAIR10	NC_003075.7_cds_NP_191979.2_29874	1	7085	5,7 locus_tag=AT4G00700	C2 calcium/lipid-binding plant phosphoribosyltransferase family protein
hrpL	Ath TAIR10	NC_003071.7_cds_NP_001323677.1_14748	2	7061	5,7 locus_tag=AT2G19890	hypothetical protein
hrpL	Ath TAIR10	NC_003071.7_cds_NP_179497.1_14629	2	6367	5,7 gene=SHT	spermidine hydroxycinnamoyl transferase
hrpL	Ath TAIR10	NC_003075.7_cds_NP_567440.1_32031	2	6041	17 gene=ORC1A	origin recognition complex 1
hrpL	Ath TAIR10	NC_003076.8_cds_NP_199024.1_43277	3	6017	5,7 locus_tag=AT5G42090	Lung seven transmembrane receptor family protein
hrpL	Ath TAIR10	NC_003071.7_cds_NP_565477.1_14887	1	5741	17 gene=RPN1A	26S proteasome regulatory subunit S2 1A
hrpL	Ath TAIR10	NC_003071.7_cds_NP_973615.2_17990	2	4870	5,7 locus_tag=AT2G36885	translation initiation factor
hrpL	Ath TAIR10	NC_003070.9_cds_NP_001322651.1_8127	1	4826	17 locus_tag=AT1G54930	GRF zinc finger / Zinc knuckle protein
hrpL	Ath TAIR10	NC_003075.7_cds_NP_193348.1_32368	2	4616	1,9 gene=ARA1	arabinose kinase
hrpL	Ath TAIR10	NC_003071.7_cds_NP_182268.1_20114	2	4447	17 gene=MYB12	myb domain protein 12
hrpL	Ath TAIR10	NC_003074.8_cds_NP_187914.1_22603	2	4406	0,64 locus_tag=AT3G13070	CBS domain-containing protein / transporter associated domain-containing protein
hrpL	Ath TAIR10	NC_003076.8_cds_NP_001318654.1_41625	2	3844	5,7 locus_tag=AT5G26090	Plant self-incompatibility protein S1 family
hrpL	Ath TAIR10	NC_003075.7_cds_NP_192982.1_31527	1	3665	5,7 locus_tag=AT4G12450	zinc finger (C2H2 type) family protein
hrpL	Ath TAIR10	NC_003075.7_cds_NP_193381.1_32446	1	3515	17 gene=INT4	inositol transporter 4
hrpL	Ath TAIR10	NC_003070.9_cds_NP_174067.2_5006	1	3346	5,7 locus_tag=AT1G27470	transducin family protein / WD-40 repeat family protein
hrpL	Ath TAIR10	NC_003076.8_cds_NP_974906.2_44493	1	3056	17 locus_tag=AT5G48440	FAD-dependent oxidoreductase family protein
hrpL	Ath TAIR10	NC_003076.8_cds_NP_197528.1_40653	1	2937	5,7 gene=SPS1F	sucrose phosphate synthase 1F

hrpL	Ath TAIR10 NC_003075.7_cds_NP_192847.1_31297	1	2825	5,7 gene=TBL23	TRICHOME BIREFRINGENCE-LIKE 23
hrpL	Ath TAIR10 NC_003076.8_cds_NP_001032168.1_48144	1	2783	5,7 locus_tag=AT5G67610	transmembrane protein (DUF2215)
hrpL	Ath TAIR10 NC_003076.8_cds_NP_196842.2_39381	1	2778	5,7 locus_tag=AT5G13380	Auxin-responsive GH3 family protein
hrpL	Ath TAIR10 NC_003071.7_cds_NP_180277.1_16154	1	2264	1,9 locus_tag=AT2G27090	bZIP transcription factor (DUF630 and DUF632)
hrpL	Ath TAIR10 NC_003075.7_cds_NP_195338.1_36462	1	2109	17 locus_tag=AT4G36150	Disease resistance protein (TIR-NBS-LRR class) family
hrpL	Ath TAIR10 NC_003074.8_cds_NP_566552.2_23337	1	2044	1,9 gene=DEG12	protease Do-like protein
hrpL	Ath TAIR10 NC_003074.8_cds_NP_189192.1_25015	1	1987	5,8 locus_tag=AT3G25640	MIZU-KUSSEI-like protein (Protein of unknown function, DUF617)
hrpL	Ath TAIR10 NC_003070.9_cds_NP_001321631.1_3315	1	1943	5,7 gene=UBC34	ubiquitin-conjugating enzyme 34
hrpL	Ath TAIR10 NC_003070.9_cds_NP_001321597.1_12311	1	1918	5,8 gene=FTSH12	FTSH protease 12
hrpL	Ath TAIR10 NC_003071.7_cds_NP_180545.2_16644	1	1884	1,9 locus_tag=AT2G29840	RING/U-box superfamily protein
hrpL	Ath TAIR10 NC_003076.8_cds_NP_568891.1_46423	1	1836	17 gene=MYB119	myb domain protein 119
hrpL	Ath TAIR10 NC_003071.7_cds_NP_001324400.1_13206	1	1833	17 locus_tag=AT2G04020	GDLS-like Lipase/Acylhydrolase superfamily protein
hrpL	Ath TAIR10 NC_003074.8_cds_NP_191355.1_28674	1	1689	5,7 locus_tag=AT3G57960	Emsy N Terminus (ENT) domain-containing protein
hrpL	Ath TAIR10 NC_003076.8_cds_NP_851188.1_45571	1	1593	17 gene=CNGC4	cyclic nucleotide-gated cation channel 4
hrpL	Ath TAIR10 NC_003071.7_cds_NP_180198.1_15999	1	1591	5,7 gene=GP ALPHA 1	G protein alpha subunit 1
hrpL	Ath TAIR10 NC_003070.9_cds_NP_001185169.1_6950	1	1543	17 gene=AGO1	Stabilizer of iron transporter SufD / Polynucleotidyl transferase
hrpL	Ath TAIR10 NC_003070.9_cds_NP_564824.1_9576	1	1499	5,7 gene=DAA1	P-loop containing nucleoside triphosphate hydrolases superfamily protein
hrpL	Ath TAIR10 NC_003071.7_cds_NP_565297.1_13067	1	1396	1,9 gene=SK16	SKP1-like 16
hrpL	Ath TAIR10 NC_003075.7_cds_NP_974490.1_29835	1	1293	17 locus_tag=AT4G00500	alpha/beta-Hydrolases superfamily protein
hrpL	Ath TAIR10 NC_003071.7_cds_NP_001077916.1_14625	1	1292	5,7 gene=RALFL13	RALF-like 13

sRNA_source	Target	Read	Read_abundance (total count)	Blast.e.value	Pairing_energy (Kcal/mol)
hrpL	WT hrpL	CAAGAAACACGCACTGGAGAA	567546	0,000000173	-36,59
hrpL	WT hrpL	CAGCCAGGTCTGCGGTTTGCT	566924	0,000000173	-41,6
hrpL	WT hrpL	ACACGCACTGGAGAATGTCAT	353690	0,000000173	-35,81
hrpL	WT hrpL	AGCAAACCGCAGACCTGGCTG	253785	0,000000173	-40,21
hrpL	WT hrpL	TTCTCCAGTGCGTGTTCCTTG	229178	0,000000173	-36,55
hrpL	WT hrpL	GATGCCACACAGCCAGGTCTG	211021	0,000000173	-41,59
hrpL	WT hrpL	CGGATCAGGTTTACGCGCATG	167512	0,000000173	-41,42
hrpL	WT hrpL	ATGACATTCTCCAGTGCGTGT	143887	0,000000173	-37,02
hrpL	WT hrpL	ACAGCCAGGTCTGCGGTTTGCT	107864	6,36E-08	-42,25
hrpL	WT hrpL	CAGCTCGGAATGCACTTCGCT	106286	0,000000173	-39,24
hrpL	WT hrpL	GACGAAGTGCATTCCGAGCTG	105824	0,000000173	-38,7
hrpL	WT hrpL	CTGACGATACATTTTGC GGAA	97869	0,000000173	-33,83
hrpL	WT hrpL	TTCCGCAAAATGTATCGTCAG	97203	0,000000173	-31,34
hrpL	WT hrpL	CAGACCTGGCTGTGTGGCATC	94841	0,000000173	-42,49
hrpL	WT hrpL	TTTCCTGATACGGCTGACGAT	86176	0,000000173	-35,92
hrpL	WT hrpL	ATCGTCAGCCGTATCAGGAAA	85934	0,000000173	-36,62
hrpL	WT hrpL	CTGGGAAGACGAAGTGCATTC	74216	0,000000173	-37,77
hrpL	WT hrpL	GAATGCATTCTGCTTCCAG	73725	0,000000173	-38,09
hrpL	WT hrpL	ACAGCCAGGTCTGCGGTTTGC	64415	0,000000173	-40,68
hrpL	WT hrpL	AAACACGCACTGGAGAATGTC	63566	0,000000173	-34,49
hrpL	WT hrpL	CTCTACCTGATGACTGACATC	52565	0,000126	-22,9
hrpL	WT hrpL	AGCAAACCGCAGACCTGGCTGT	48156	6,36E-08	-41,72
hrpL	WT hrpL	ACACGCACTGGAGAATGTCATC	44751	6,36E-08	-38,4
hrpL	WT hrpL	GCGCGATGCCACACAGCCAGG	43853	0,000000173	-42,89
hrpL	WT hrpL	TCAGGTTTACGCGCATGCCAC	42436	0,000000173	-40,62
hrpL	WT hrpL	TGACTGACATCACCGTGCCCT	42186	0,000000173	-39,77
hrpL	WT hrpL	AGGGCACGGTGATGTAGTCA	41799	0,000000173	-41,85
hrpL	WT hrpL	CTCGGAATGCACTTCGCTCTTC	39922	0,000000173	-37,38
hrpL	WT hrpL	TTGGATAAACGCCCTGAGCATCTG	39498	0,0000183	-24,63
hrpL	WT hrpL	GAAGACGAAGTGCATTCCGAG	39440	0,000000173	-36,75
hrpL	WT hrpL	AACACGCACTGGAGAATGTCA	39054	0,000000173	-35,11
hrpL	WT hrpL	AACACGCACTGGAGAATGTCTAT	38433	6,36E-08	-36,09
hrpL	WT hrpL	GATGCCACACAGCCAGGTCTGC	36555	6,36E-08	-44,1
hrpL	WT hrpL	AGGTTTACGCGCATGCCACAC	33405	0,000000173	-38,05
hrpL	WT hrpL	GGTTCATCACCCGCTTTTGGA	33225	0,000000173	-37,15
hrpL	WT hrpL	GCGATGCCACACAGCCAGGTC	31502	0,000000173	-43,23
hrpL	WT hrpL	GCGGATCAGGTTTACGCGCAT	31263	0,000000173	-41,32
hrpL	WT hrpL	GCAAACCGCAGACCTGGCTGT	28863	0,000000173	-40,18
hrpL	WT hrpL	CGGATCAGGTTTACGCGCATGC	28526	6,36E-08	-44,14
hrpL	WT hrpL	GACATTCTCCAGTGCGTGTTC	25616	0,000000173	-36,12
hrpL	WT hrpL	AAGAAACACGCACTGGAGAAAT	25143	0,000000173	-34,99
hrpL	WT hrpL	AGCCAGGTCTGCGGTTTGCTG	24514	0,000000173	-41,71
hrpL	WT hrpL	GGGCACGGTGATGTCAGTCAT	23470	0,000000173	-41,31
hrpL	WT hrpL	CAGGTTTACGCGCATGCCACA	23400	0,000000173	-40,28
hrpL	WT hrpL	ATGACTGACATCACCGTGCCC	23337	0,000000173	-38,84
hrpL	WT hrpL	CTGGCATGTTGAACTTTGTGC	23001	0,000000173	-35,69
hrpL	WT hrpL	GGCTTCAAGAAACACGCATG	22934	0,000000173	-37,37
hrpL	WT hrpL	AAACACGCACTGGAGAATGTCTATC	19548	8,35E-09	-39,6
hrpL	WT hrpL	CCTGGCTGTGTGGCATCGCGC	19380	0,000000173	-45,16
hrpL	WT hrpL	TGACGATACATTTTGC GGAA	19321	0,000000173	-33,75
hrpL	WT hrpL	CTTCGCAAAATGTATCGTCA	19136	0,000000173	-33,2
hrpL	WT hrpL	GTGGCATCGCGCTGAACCTGA	19019	0,000000173	-40,56
hrpL	WT hrpL	CAGGTCTGCGGTTTGCTGGCA	18446	0,000000173	-41,98
hrpL	WT hrpL	GACGAAGTGCATTCCGAGCTGG	18259	6,36E-08	-41,93
hrpL	WT hrpL	CCAGCTCGGAATGCACTTCGTC	18237	6,36E-08	-42,45
hrpL	WT hrpL	GATGACATTCTCCAGTGCGTGT	18110	6,36E-08	-39,9
hrpL	WT hrpL	GGTTCATCACCCGCTTTTGATAA	17376	8,35E-09	-37,65
hrpL	WT hrpL	AGCGCGATGCCACACAGCCAG	17318	0,000000173	-40,01
hrpL	WT hrpL	ACGGCTGACGATACATTTTGC GGGA	17281	8,35E-09	-43,3
hrpL	WT hrpL	TCCGCAAAATGTATCGTCAGCCGT	17224	8,35E-09	-40,86
hrpL	WT hrpL	AGGTTTACGCGCATGCCACACA	16921	6,36E-08	-39,64
hrpL	WT hrpL	CTGACGATACATTTTGC GGAA	16916	6,36E-08	-36,32
hrpL	WT hrpL	CTTCGCAAAATGTATCGTCAG	16855	6,36E-08	-33,81
hrpL	WT hrpL	ACGGCTGACGATACATTTTGC	16412	0,000000173	-35,87
hrpL	WT hrpL	GCAGACCTGGCTGTGTGGCATC	16320	6,36E-08	-45,78
hrpL	WT hrpL	GCAAAATGTATCGTCAGCCGT	16293	0,000000173	-35,49
hrpL	WT hrpL	AGGGCTTCAAGAAACACGCACTGG	16256	8,35E-09	-45,01
hrpL	WT hrpL	CAGATGCTCAGGGCGTTTATCAA	15961	0,0000183	-30,71
hrpL	WT hrpL	TGACATTCTCCAGTGCGTGT	15867	0,000000173	-36,72
hrpL	WT hrpL	ACGAAGTGCATTCCGAGCTGG	15705	0,000000173	-39,04
hrpL	WT hrpL	TGGCATGTTGAACTTTGTGCT	15665	0,000000173	-34,54
hrpL	WT hrpL	ATGACATTCTCCAGTGCGTGTT	15642	6,36E-08	-37,8
hrpL	WT hrpL	CCAGCTCGGAATGCACTTCGT	15619	0,000000173	-39,57

hrpL	WT hrpL	TCCGCAAAATGTATCGTCAGC	15009	0,000000173	-33,53
hrpL	WT hrpL	CGATGCCACACAGCCAGGTCTG	14954	6,36E-08	-43,9
hrpL	WT hrpL	CACGCACTGGAGAATGTCATC	14901	0,000000173	-36,83
hrpL	WT hrpL	GTGTGGCATCGCGCTGAACCT	14793	0,000000173	-40,35
hrpL	WT hrpL	TCAAGAAACACGCACTGGAGA	14745	0,000000173	-37,39
hrpL	WT hrpL	AGGGCTTCAAGAAACACGCACT	14744	6,36E-08	-39,21
hrpL	WT hrpL	GCTGACGATACATTTTGCGGA	14681	0,000000173	-36,17
hrpL	WT hrpL	ACGCACTGGAGAATGTCATCC	14550	0,000000173	-36,86
hrpL	WT hrpL	CACAGCCAGGTCTGCGGTTTG	14288	0,000000173	-38,19
hrpL	WT hrpL	GACCTGGCTGTGTGGCATCGC	14183	0,000000173	-44,07
hrpL	WT hrpL	TTCAAGAAACACGCACTGGAGA	14139	6,36E-08	-38,3
hrpL	WT hrpL	CTTGTGCTCGTTACGCAAGGC	14132	0,000000173	-39,85
hrpL	WT hrpL	GATCAGGTTGAGCGCGATGCC	14062	0,000000173	-41,13
hrpL	WT hrpL	ATGCCACACAGCCAGGTCTGC	13884	0,000000173	-41,2
hrpL	WT hrpL	TTACGCAGGGCTTCAAGAAAC	13803	0,000000173	-36,3
hrpL	WT hrpL	TTGCTGGCATGTTGAACTTGTGC	13773	8,35E-09	-41,19
hrpL	WT hrpL	TCAAGAAACACGCACTGGAGAA	13668	6,36E-08	-38,39
hrpL	WT hrpL	CCAGGTCTGCGGTTTGCTGGC	13612	0,000000173	-42,85
hrpL	WT hrpL	GCTGGCATGTTGAACTTGTGC	13562	6,36E-08	-39,04
hrpL	WT hrpL	ATGACTGACATCACCGTGCCCT	13549	6,36E-08	-40,45
hrpL	WT hrpL	AGGGCACGGTGATGTGATCAT	13548	6,36E-08	-42,9
hrpL	WT hrpL	TCCAAAAGCGGGTGATGAACC	13453	0,000000173	-37,34
hrpL	WT hrpL	GATCAGGTTGAGCGCGATGCCA	13150	6,36E-08	-42,44
hrpL	WT hrpL	GCCCTTCCAGCTCGGAATGCA	13069	0,000000173	-39,4
hrpL	WT hrpL	TGCATTCGAGCTGGAAGGGC	12933	0,000000173	-39,23
hrpL	WT hrpL	GCTGACGATACATTTTGCGGAA	12551	6,36E-08	-37,17
hrpL	WT hrpL	TTCCGCAAAATGTATCGTCAGC	12434	6,36E-08	-34,53
hrpL	WT hrpL	AAACACGCACTGGAGAATGTCA	12316	6,36E-08	-35,79
hrpL	WT hrpL	TACGGCTGACGATACATTTTGC	11375	6,36E-08	-37,13
hrpL	WT hrpL	GCAAAATGTATCGTCAGCCGTA	11260	6,36E-08	-36,75
hrpL	WT hrpL	CGCGATGCCACACAGCCAGGTCTG	11025	8,35E-09	-48,97
hrpL	WT hrpL	CAGCAAACCGCAGACCTGGCT	10896	0,000000173	-40,45
hrpL	WT hrpL	GAATGTCATCCACATCATCAG	10694	0,000000173	-32,92
hrpL	WT hrpL	TGTGGCATCGCGCTGAACCTG	10665	0,000000173	-40,25
hrpL	WT hrpL	CGCACTGGAGAATGTCATCCA	10458	0,000000173	-36,01
hrpL	WT hrpL	ATTCTCCAGTGCGTGTCTTCT	10374	0,000000173	-34,96
hrpL	WT hrpL	TCAGGTTCAGCGCATGCCACA	10185	6,36E-08	-42,18
hrpL	WT hrpL	CACGCACTGGAGAATGTCATCC	10098	6,36E-08	-39,34
hrpL	WT hrpL	ATGCACTTCGTCTTCCCAGCT	9914	0,000000173	-38,74
hrpL	WT hrpL	AGCTGGGAAGACGAAGTGCAT	9839	0,000000173	-39,64
hrpL	WT hrpL	CAAGAAACACGCACTGGAGAAT	9800	6,36E-08	-37,58
hrpL	WT hrpL	CGCTTTTGATAAACGCCCTGAGC	9609	7,51E-08	-36,44
hrpL	WT hrpL	GCACAAGTTTCAACATGCCAG	9414	0,000000173	-34,14
hrpL	WT hrpL	CAGTGCGTGTTTCTTGAAGCC	9275	0,000000173	-37,72
hrpL	WT hrpL	GAAGTGCATTCCGAGCTGGAA	9080	0,000000173	-36,71
hrpL	WT hrpL	TTCCAGCTCGGAATGCATTC	8951	0,000000173	-36,61
hrpL	WT hrpL	TCCCAGCTTTCTGATACGGC	8784	0,000000173	-38,98
hrpL	WT hrpL	CATGTTGAACTTGTGCTCGT	8746	0,000000173	-33,28
hrpL	WT hrpL	GATCCGCAATCACTTCCGCAA	8649	0,000000173	-35,7
hrpL	WT hrpL	GCCGTATCAGGAAAGCTGGGA	8634	0,000000173	-40,74
hrpL	WT hrpL	TTGCGGAAGTGATTGCGGATC	8623	0,000000173	-38,19
hrpL	WT hrpL	TATCGTCAGCCGTATCAGGAA	8545	0,000000173	-36,97
hrpL	WT hrpL	TTCTGATACGGCTGACGATA	8402	0,000000173	-36,32
hrpL	WT hrpL	TGATTGCGGATCAGGTTACAGC	8383	0,000000173	-37,55
hrpL	WT hrpL	AATGTCATCCACATCATCAGG	8354	0,000000173	-33,56
hrpL	WT hrpL	TGAAACTTGTGCTCGTTACG	8256	0,000000173	-32,34
hrpL	WT hrpL	TGCCAGCAAACCGCAGACTCG	8247	0,000000173	-38,76
hrpL	WT hrpL	GATGCCACACAGCCAGGTCT	8129	0,000000468	-39,33
hrpL	WT hrpL	CGGCTGACGATACATTTTGCGGAA	8083	8,35E-09	-42,69
hrpL	WT hrpL	TGCTGGCATGTTGAACTTGT	7951	0,000000173	-35,2
hrpL	WT hrpL	TTCCGCAAAATGTATCGTCAGCCG	7926	8,35E-09	-40,16
hrpL	WT hrpL	AGACGAAGTGCATTCCGAGCT	7925	0,000000173	-37,57
hrpL	WT hrpL	GATGACATTCTCCAGTGCGTGT	7839	8,35E-09	-41,58
hrpL	WT hrpL	AGCTCGGAATGCACTTCGTCT	7798	0,000000173	-38,4
hrpL	WT hrpL	CTGGCTGTGTGGCATCGCGCT	7793	0,000000173	-43,5
hrpL	WT hrpL	TGAACCTGATCCGCAATCACT	7778	0,000000173	-36,14
hrpL	WT hrpL	CAGCCAGGTCTGCGGTTTGCTG	7735	6,36E-08	-43,34
hrpL	WT hrpL	AGTGATTGCGGATCAGGTTCA	7705	0,000000173	-37,1
hrpL	WT hrpL	TGTGTGGCATCGCGCTGAACCT	7508	6,36E-08	-41,93
hrpL	WT hrpL	GTCATCCACATCATCAGGGTT	7345	0,000000173	-37,28
hrpL	WT hrpL	GAACCTGATCCGCAATCACTCCG	7275	8,35E-09	-42,84
hrpL	WT hrpL	TCCACATCATCAGGGTTCA	7264	6,36E-08	-39,1
hrpL	WT hrpL	CTTCCGCAAAATGTATCGTCAGCC	7212	8,35E-09	-40,31
hrpL	WT hrpL	CGGAAGTGATTGCGGATCAGGTT	7182	8,35E-09	-44,6

hrpL	WT hrpL	CAAAATGTATCGTCAGCCGTA	7149	0,000000173	-33,46
hrpL	WT hrpL	GGCTGACGATACATTTTGCGGAAG	7129	8,35E-09	-42,83
hrpL	WT hrpL	TCCAGCTCGGAATGCACTTCG	7098	0,000000173	-39
hrpL	WT hrpL	GTGATGTCAGTCATCAGGTAG	7088	0,000014	-26,9
hrpL	WT hrpL	TTATCCAAAAGCGGGTGATGAACC	7085	8,35E-09	-40,39
hrpL	WT hrpL	CAGCCAGGTCTGCGGTTTG	7081	0,000000468	-40,03
hrpL	WT hrpL	TACGGCTGACGATACATTTTG	7057	0,000000173	-33,99
hrpL	WT hrpL	CGAAGTGCATTCCGAGCTGGA	6977	0,000000173	-38,79
hrpL	WT hrpL	CTACCTGATGACTGACATCAC	6968	0,000014	-25,65
hrpL	WT hrpL	CCACATCATCAGGGTTCATCA	6913	0,000000173	-37,74
hrpL	WT hrpL	ACACGCACTGGAGAATGTCA	6894	0,000000468	-35,13
hrpL	WT hrpL	CTTGTGCTCGTTACGCGAGGCT	6867	6,36E-08	-41,48
hrpL	WT hrpL	GGATCAGGTTTCAGCGCGATGC	6816	0,000000173	-41,89
hrpL	WT hrpL	GGCTGACGATACATTTTGCGG	6725	0,000000173	-37,44
hrpL	WT hrpL	CAGACCTGGCTGTGTGGCATCG	6697	6,36E-08	-44,82
hrpL	WT hrpL	CCGCAAAATGTATCGTCAGCC	6636	0,000000173	-35,89
hrpL	WT hrpL	CAAACCGCAGACCTGGCTGTG	6630	0,000000173	-39,45
hrpL	WT hrpL	GCTCGGAATGCACTTCGTCTT	6626	0,000000173	-38,01
hrpL	WT hrpL	CAGGTTTCAGCGCGATGCCAC	6622	0,000000468	-38,73
hrpL	WT hrpL	CCAGTGCGTGTTCCTGAAGCCCT	6609	8,35E-09	-45,45
hrpL	WT hrpL	AAGACGAAGTGCATTCCGAGC	6534	0,000000173	-36,87
hrpL	WT hrpL	GGCATCGCGCTGAACCTGATC	6480	0,000000173	-41
hrpL	WT hrpL	TGTATCGTCAGCCGTATCAGGAAA	6398	8,35E-09	-42
hrpL	WT hrpL	CATCATCAGGGTTTCATACCC	6376	0,000000173	-34,09
hrpL	WT hrpL	ATCAGGTTTCAGCGCGATGCCA	6338	0,000000173	-39,55
hrpL	WT hrpL	AGCACAAGTTTCAACATGCCA	6336	0,000000173	-31,33
hrpL	WT hrpL	TTTCCTGATACGGCTGACGATACA	6287	8,35E-09	-41,4
hrpL	WT hrpL	CGCAAATGTATCGTCAGCCG	6279	0,000000173	-35,78
hrpL	WT hrpL	CGGCTGACGATACATTTTGCG	6240	0,000000173	-36,52
hrpL	WT hrpL	CAGGGCTTCAAGAAACACGCA	6142	0,000000173	-37,82
hrpL	WT hrpL	GCAGACCTGGCTGTGTGGCAT	6060	0,000000173	-43,04
hrpL	WT hrpL	GCCAGCAAACCGCAGACCTGG	6059	0,000000173	-40,9
hrpL	WT hrpL	TTGTGCTCGTTACGCGAGGGCT	6053	0,000000173	-38,99
hrpL	WT hrpL	GATGACATTCTCCAGTGCGTG	6028	0,000000173	-38,32
hrpL	WT hrpL	ACCTGATCCGCAATCACITTC	6019	0,000000173	-37,91
hrpL	WT hrpL	AGTGCGTGTTTCTTGAAGCCCT	5992	6,36E-08	-39,75
hrpL	WT hrpL	GGAAGTGATTGCGGATCAGGT	5960	0,000000173	-38,85
hrpL	WT hrpL	TGGCATCGCGCTGAACCTGATC	5958	6,36E-08	-41,79
hrpL	WT hrpL	ATTGCGGATCAGGTTTCAGCGC	5890	0,000000173	-38,31
hrpL	WT hrpL	GGATGACATTCTCCAGTGCGT	5860	0,000000173	-37,98
hrpL	WT hrpL	TCTCCAGTGCGTGTTTCTTGA	5801	0,000000173	-37,38
hrpL	WT hrpL	GCCCTGCGTAACGAGCACAAG	5766	0,000000173	-36,98
hrpL	WT hrpL	GCACAAGTTTCAACATGCCAGCAA	5741	8,35E-09	-40,07
hrpL	WT hrpL	TCTCCAGTGCGTGTTTCTTGAA	5652	6,36E-08	-38,15
hrpL	WT hrpL	GCACAAGTTTCAACATGCCAGC	5595	6,36E-08	-37,47
hrpL	WT hrpL	ACACGCACTGGAGAATGTCA	5577	0,000000052	-35,4
hrpL	WT hrpL	TGATACGGCTGACGATACATT	5469	0,000000173	-34,92
hrpL	WT hrpL	AATGTATCGTCAGCCGTATCA	5457	0,000000173	-34,34
hrpL	WT hrpL	GTTTCTTGAAGCCCTGCGTAA	5439	0,000000173	-36,51
hrpL	WT hrpL	GCGGATCAGGTTTCAGCGCGATGCC	5402	8,35E-09	-48,67
hrpL	WT hrpL	TTCTCCAGTGCGTGTTTCTTGA	5377	6,36E-08	-38,37
hrpL	WT hrpL	GAGAATGTATCCACATCATC	5304	0,000000173	-32,48
hrpL	WT hrpL	GCGCGATGCCACACAGCCAGGT	5279	6,36E-08	-44,59
hrpL	WT hrpL	TGACATTCTCCAGTGCGTGTTT	5096	6,36E-08	-37,62
hrpL	WT hrpL	CAGACCTGGCTGTGTGGCATCGCG	5045	8,35E-09	-50,32
hrpL	WT hrpL	CATGTTGAAACTTGTGCTCGT	5001	6,36E-08	-34,1
hrpL	WT hrpL	TCGTACAGCCGTATCAGGAAAG	4978	0,000000173	-38,01
hrpL	WT hrpL	CTTTCCTGATACGGCTGACGA	4861	0,000000173	-37,31
hrpL	WT hrpL	CGGATCAGGTTTCAGCGCGAT	4827	0,000000468	-38,96
hrpL	WT hrpL	ACTTGTGCTCGTTACGCGAGGGCTT	4826	8,35E-09	-43,99
hrpL	WT hrpL	GCCAGGTCTGCGGTTTGCTGG	4775	0,000000173	-42,66
hrpL	WT hrpL	TGCTCGTTACGCGAGGGCTTCA	4722	0,000000173	-39,46
hrpL	WT hrpL	AGCGCGATGCCACACAGCCAGG	4691	6,36E-08	-43,31
hrpL	WT hrpL	GGGCTTCAAGAAACACGCACT	4647	0,000000173	-37,91
hrpL	WT hrpL	AACCTGATCCGCAATCACTTC	4620	0,000000173	-35,44
hrpL	WT hrpL	GGATCAGGTTTCAGCGCGATGCCAC	4564	8,35E-09	-47,79
hrpL	WT hrpL	ACGCAGGGCTTCAAGAAACAC	4564	0,000000173	-38,06
hrpL	WT hrpL	TGTGGCATCGCGCTGAACCTGA	4539	6,36E-08	-42,15
hrpL	WT hrpL	GAAGTGATTGCGGATCAGGTT	4476	0,000000173	-36,44
hrpL	WT hrpL	AAGAAACACGCACTGGAGAA	4365	0,000000468	-34
hrpL	WT hrpL	CCCAGCTTTCCTGATACGGCT	4348	0,000000173	-38,77
hrpL	WT hrpL	TTTCCTGATACGGCTGACGATA	4326	6,36E-08	-37,22
hrpL	WT hrpL	AGCCGTATCAGGAAAGCTGGG	4304	0,000000173	-39,93
hrpL	WT hrpL	TGGATGACATTCTCCAGTGCG	4286	0,000000173	-35,99

hrpL	WT hrpL	TATCGTCAGCCGTATCAGGAAA	4266	6,36E-08	-37,85
hrpL	WT hrpL	GCAGGGCTTCAAGAAACACGC	4252	0,000000173	-39,1
hrpL	WT hrpL	GCCACACAGCCAGGTCTGCGG	4225	0,000000173	-41,67
hrpL	WT hrpL	CGAGCTGGAAGGGCACGGTGA	4176	0,000000173	-43,89
hrpL	WT hrpL	CTGATGATGTGGATGACATTC	4148	0,000000173	-34,39
hrpL	WT hrpL	CAAGAAACACGCACTGGAGA	4139	0,000000468	-35,59
hrpL	WT hrpL	AACACGCACTGGAGAATGTCATC	4071	2,31E-08	-38,86
hrpL	WT hrpL	GGATGACATTCTCCAGTGCGTG	4058	6,36E-08	-40,57
hrpL	WT hrpL	CGATGCCACACAGCCAGGTCT	4036	0,000000173	-41,59
hrpL	WT hrpL	TCACCGTGCCCTTCCAGCTCG	3997	0,000000173	-41,32
hrpL	WT hrpL	TCCACATCATCAGGGTTCATC	3991	0,000000173	-37,51
hrpL	WT hrpL	GAAACACGCACTGGAGAATGT	3973	0,000000173	-35,64
hrpL	WT hrpL	GCTCAGGGCGTTTATCCAAAAGCG	3907	7,51E-08	-40,54
hrpL	WT hrpL	ATTCTCCAGTGCGTGTTCCTTG	3894	6,36E-08	-37,55
hrpL	WT hrpL	CGCAAATGTATCGTCAGCCG	3861	0,000000052	-34,95
hrpL	WT hrpL	CGGCTGACGATACATTTTGCG	3775	0,000000052	-35,55
hrpL	WT hrpL	TGACGATACATTTTGCGGAAGT	3722	6,36E-08	-35,43
hrpL	WT hrpL	ACAAGTTTCAACATGCCAGCA	3716	0,000000173	-34,52
hrpL	WT hrpL	TGCGGATCAGGTTACGCGCGA	3712	0,000000173	-40,74
hrpL	WT hrpL	TCCGCAAAATGTATCGTCAGCC	3706	6,36E-08	-36,83
hrpL	WT hrpL	TTTGCTGGCATGTTGAAACTT	3675	0,000000173	-32,87
hrpL	WT hrpL	AGTGATTGCGGATCAGGTTACGCG	3665	8,35E-09	-44,39
hrpL	WT hrpL	ACGAGCACAAGTTTCAACATG	3646	0,000000173	-33,83
hrpL	WT hrpL	AGACCTGGCTGTGTGGCATC	3607	0,000000468	-39,99
hrpL	WT hrpL	GGCTGACGATACATTTTGCGGA	3556	6,36E-08	-39,34
hrpL	WT hrpL	ACTTCGCAAAATGTATCGTCA	3545	6,36E-08	-34,84
hrpL	WT hrpL	TTCCAGCTCGGAATGCATTCGTC	3515	8,35E-09	-43,56
hrpL	WT hrpL	CAGCAAACCGCAGACCTGGCTG	3513	6,36E-08	-41,88
hrpL	WT hrpL	CGGATCAGGTTACGCGCATGCC	3470	2,31E-08	-45,3
hrpL	WT hrpL	CCTGATGATGTGGATGACATT	3438	0,000000173	-35,41
hrpL	WT hrpL	GATCAGGTTACGCGCATGCCACA	3430	8,35E-09	-46,16
hrpL	WT hrpL	GACGAAGTGCAATCCGAGCTGGAA	3403	8,35E-09	-43,62
hrpL	WT hrpL	TGCATTCCGAGCTGGAAGGGCACG	3401	8,35E-09	-45,55
hrpL	WT hrpL	TTCAAGAAACACGCACTGGAGAAT	3387	8,35E-09	-40,26
hrpL	WT hrpL	GCTTTCTGATACGGCTGACGATA	3387	8,35E-09	-42,87
hrpL	WT hrpL	CGTAACGAGCACAAGTTTCAA	3367	0,000000173	-32,98
hrpL	WT hrpL	CGTGCCCTTCCAGCTCGGAATGCA	3346	8,35E-09	-44,05
hrpL	WT hrpL	TATCGTCAGCCGTATCAGGAAAGC	3324	8,35E-09	-43,44
hrpL	WT hrpL	AGAAACACGCACTGGAGAATGTCA	3250	8,35E-09	-40,28
hrpL	WT hrpL	GCGGTTTGCTGGCATGTTGAAA	3169	6,36E-08	-39,44
hrpL	WT hrpL	CATCATCAGGGTTCA	3153	0,0000633	-24,96
hrpL	WT hrpL	CTGAACCTGATCCGCAATCACT	3141	6,36E-08	-38,69
hrpL	WT hrpL	AGTGATTGCGGATCAGGTTTCAG	3135	6,36E-08	-39,68
hrpL	WT hrpL	CAGCCAGGTCTGCGGTTTGCT	3134	0,000000052	-41,26
hrpL	WT hrpL	GCAAACCGCAGACCTGGCTG	3076	0,000000468	-38,68
hrpL	WT hrpL	TCGTACGCCGTATCAGGAAAGC	3056	6,36E-08	-41,09
hrpL	WT hrpL	GCTTTCTGATACGGCTGACGA	3048	6,36E-08	-40,47
hrpL	WT hrpL	GCAAAATGTATCGTCAGCCGTATC	3035	8,35E-09	-40,59
hrpL	WT hrpL	TTGCTGGCATGTTGAAACTTG	3035	0,000000173	-34,55
hrpL	WT hrpL	GATACGGCTGACGATACATTTTGC	3006	8,35E-09	-41,02
hrpL	WT hrpL	TGATGAACCTGATGATGTGGA	3000	6,36E-08	-39,3
hrpL	WT hrpL	TGGCATCGCGCTGAACTGAT	2990	0,000000173	-38,94
hrpL	WT hrpL	CGGCTGACGATACATTTTGCGG	2987	0,000000191	-38,84
hrpL	WT hrpL	TCTACCTGATGACTGACATC	2978	0,000114	-22,49
hrpL	WT hrpL	AACCTGATGATGTGGATGAC	2976	0,000000173	-36,7
hrpL	WT hrpL	GTGGCATCGCGCTGAACCTG	2962	0,000000468	-38,67
hrpL	WT hrpL	CGCACTGGAGAATGTCATCCAC	2937	6,36E-08	-38,65
hrpL	WT hrpL	CCGCAAAATGTATCGTCAGCCG	2925	0,000000191	-37,29
hrpL	WT hrpL	GGTTCAGCGCATGCCACACA	2921	0,000000173	-38,04
hrpL	WT hrpL	TGACATTCTCCAGTGCGTGT	2920	0,000000468	-35,95
hrpL	WT hrpL	GATGTCAGTCATCAGGTAGA	2908	0,000114	-24,92
hrpL	WT hrpL	CCTTCCAGCTCGGAATGCACT	2893	0,000000173	-38,1
hrpL	WT hrpL	GCAGGGCTTCAAGAAACACGCACT	2885	8,35E-09	-44,65
hrpL	WT hrpL	GGGCTTCAAGAAACACGCACTG	2867	0,000000191	-38,49
hrpL	WT hrpL	TTTTGGATAAACGCCCTGAGC	2856	0,00000156	-29,03
hrpL	WT hrpL	CCAGGTCTGCGGTTTGCTGGCA	2853	6,36E-08	-44,4
hrpL	WT hrpL	AGTGCAATCCGAGCTGGAAGG	2838	0,000000173	-38,45
hrpL	WT hrpL	TCTGCGGTTTGCTGGCATGTT	2825	0,000000173	-38,07
hrpL	WT hrpL	TGATGAACCTGATGATGTGG	2789	0,000000173	-37,67
hrpL	WT hrpL	AGCCCTGCGTAACGACACAAG	2788	6,36E-08	-38,26
hrpL	WT hrpL	GGGTTTCATCACCCGCTTTTGG	2783	0,000000173	-35,58
hrpL	WT hrpL	CGTTACGCAGGGCTTCAAGAA	2778	0,000000173	-37,96
hrpL	WT hrpL	CCACACAGCCAGGTCTGCGGT	2773	0,000000173	-39,84
hrpL	WT hrpL	CACAGCCAGGTCTGCGGTTTGCTG	2731	8,35E-09	-45,72

hrpL	WT hrpL	ACTTCGCAAAATGTATCGTC	2714	0,000000173	-33,39
hrpL	WT hrpL	CGCACTGGAGAATGTCATCCA	2696	0,000000052	-36,07
hrpL	WT hrpL	CACAGCCAGGTCTGCGGTTTGC	2684	6,36E-08	-41,57
hrpL	WT hrpL	GACGATACATTTTGCGGAAGT	2672	0,000000173	-33,86
hrpL	WT hrpL	GAAACACGCACTGGAGAATGTCAT	2662	8,35E-09	-39,7
hrpL	WT hrpL	GCGGTTTGCTGGCATGTTGAA	2657	0,000000173	-38,54
hrpL	WT hrpL	CGTGCCCTTCCAGCTCGGAAT	2646	0,000000173	-39,41
hrpL	WT hrpL	ATTCCGAGCTGGAAGGGCACG	2637	0,000000173	-39,92
hrpL	WT hrpL	TGCGTGTTCCTTGAAGCCCTG	2587	0,000000173	-38,35
hrpL	WT hrpL	AGCGGATGCCACACAGCCAGG	2577	0,000000191	-42,18
hrpL	WT hrpL	CAGCTCGGAATGCACTTCGTCT	2547	6,36E-08	-40,82
hrpL	WT hrpL	CTTCCGCAAAATGTATCGTCAGC	2545	2,31E-08	-37,01
hrpL	WT hrpL	GCTGACGATACATTTTGCGGAAG	2526	2,31E-08	-39,66
hrpL	WT hrpL	GGGTGATGAACCCTGATGATG	2499	0,000000173	-36,42
hrpL	WT hrpL	GTATCGTCAGCCGTATCAGGA	2494	0,000000173	-38,66
hrpL	WT hrpL	TGCACTTCGTCTTCCCAGCTT	2482	0,000000173	-38,54
hrpL	WT hrpL	TGATGTCAGTCAATCAGGTAGA	2459	0,0000421	-24,65
hrpL	WT hrpL	TCCTGATACGGCTGACGATAC	2458	0,000000173	-37,98
hrpL	WT hrpL	ATGTTGAAACTTGTGCTCGTT	2452	0,000000173	-31,51
hrpL	WT hrpL	AGCCCTGCGTAACGAGCACAA	2450	0,000000173	-36,21
hrpL	WT hrpL	AGACGAAGTGCAATTCGAGCTG	2446	6,36E-08	-40,12
hrpL	WT hrpL	AAGCTGGGAAGACGAAGTGCA	2442	0,000000173	-39,45
hrpL	WT hrpL	TCTACCTGATGACTGACATCA	2425	0,0000421	-23,33
hrpL	WT hrpL	ACCTGGCTGTGTGGCATCGCGC	2420	6,36E-08	-46,65
hrpL	WT hrpL	AAGAAACACGCACTGGAGAATG	2385	6,36E-08	-37,24
hrpL	WT hrpL	CGTCAGCCGTATCAGGAAAGC	2378	0,000000173	-39,31
hrpL	WT hrpL	AGCCAGGTCTGCGGTTTGCTGG	2373	6,36E-08	-44,23
hrpL	WT hrpL	CGCACTGGAGAATGTCATCCA	2371	0,000000052	-36,87
hrpL	WT hrpL	GCTTTCCTGATACGGCTGACG	2357	0,000000173	-38,58
hrpL	WT hrpL	GCCAGGTCTGCGGTTTGC	2319	0,00000328	-36,06
hrpL	WT hrpL	ACATTTTGCGGAAGTGATTGCGGA	2315	8,35E-09	-41,41
hrpL	WT hrpL	TCCGCAATCACTTCGCAAAATGT	2301	8,35E-09	-38,32
hrpL	WT hrpL	CACACAGCCAGGTCTGCGGTT	2299	0,000000173	-38,18
hrpL	WT hrpL	AACACGCACTGGAGAATGTCATCC	2275	8,35E-09	-41,67
hrpL	WT hrpL	ATCCACATCATCAGGGTTTCAT	2264	0,000000173	-35,71
hrpL	WT hrpL	GGCTTCAAGAAACACGCACTGGAG	2245	8,35E-09	-45,07
hrpL	WT hrpL	CAGGGCTTCAAGAAACACGCAC	2245	6,36E-08	-40,35
hrpL	WT hrpL	AGCCAGGTCTGCGGTTTGCT	2228	0,000000468	-39,19
hrpL	WT hrpL	AGGAAAGCTGGGAAGACGAAG	2207	0,000000173	-39,03
hrpL	WT hrpL	CTTCGTCTTCCCAGCTTTCCT	2199	0,000000173	-38,58
hrpL	WT hrpL	ATGACATTCTCCAGTGCGTGT	2126	0,000000052	-37,22
hrpL	WT hrpL	CCAGCAAACCGCAGACCTGGC	2123	0,000000173	-41,21
hrpL	WT hrpL	AGCGCGATGCCACACAGCCAGTC	2121	8,35E-09	-47,9
hrpL	WT hrpL	ACCCGCTTTTGATAAACGCCCTGA	2109	0,000000025	-43,26
hrpL	WT hrpL	GATGATGTGGATGACATTCTC	2104	0,000000173	-34,31
hrpL	WT hrpL	AGGTTTCAGCGCGATGCCACACAGC	2090	8,35E-09	-45,32
hrpL	WT hrpL	CCTGGCTGTGTGGCATCGCGCT	2074	6,36E-08	-46,77
hrpL	WT hrpL	AACGAGCACAGTTTCAACATG	2060	6,36E-08	-34,66
hrpL	WT hrpL	CAGGTCTGCGGTTTGCTGGC	2045	0,000000468	-40,59
hrpL	WT hrpL	TCACCCGCTTTTGATAAACG	2044	0,000000173	-31,31
hrpL	WT hrpL	AGCTTTCCTGATACGGCTGAC	2027	0,000000173	-37,32
hrpL	WT hrpL	ACACAGCCAGGTCTGCGGTTTG	2023	6,36E-08	-39,22
hrpL	WT hrpL	CACGCACTGGAGAATGTCAT	2009	0,000000468	-34,33
hrpL	WT hrpL	ACTTCGCAAAATGTATCGTCAGC	2007	8,35E-09	-38,32
hrpL	WT hrpL	CCGCAGACCTGGCTGTGTGGC	2007	0,000000173	-45,37
hrpL	WT hrpL	CATCATCAGGGTTTCATC	1987	0,00000328	-30,49
hrpL	WT hrpL	GCTCGGAATGCACATTCGTCTTC	1983	6,36E-08	-40,74
hrpL	WT hrpL	CGGAAGTGATTGCGGATCAGG	1979	0,000000173	-39,54
hrpL	WT hrpL	CCTGATCCGCAATCACTCCG	1973	0,000000173	-38,19
hrpL	WT hrpL	GTCAGCCGTATCAGGAAAGCT	1970	0,000000173	-37,81
hrpL	WT hrpL	CCCTTCCAGCTCGGAATGCACT	1968	6,36E-08	-41,07
hrpL	WT hrpL	CACTTCCGCAAAATGTATCGT	1960	0,000000173	-32,47
hrpL	WT hrpL	GCTGACGATACATTTGCGGAAGT	1956	8,35E-09	-41,32
hrpL	WT hrpL	GCTTCAAGAAACACGCACTGGA	1956	6,36E-08	-39,57
hrpL	WT hrpL	AAGCCCTGCGTAACGAGCACAAAT	1943	8,35E-09	-40,69
hrpL	WT hrpL	ACACGCACTGGAGAATGTCATC	1943	0,000000191	-36,28
hrpL	WT hrpL	TCACCGTGCCCTTCCAGCTCG	1926	0,000000052	-40,91
hrpL	WT hrpL	AGTGCAATCCGAGCTGGAAGGG	1922	6,36E-08	-41,71
hrpL	WT hrpL	CGCACTGGAGAATGTC	1922	0,0000253	-28,28
hrpL	WT hrpL	GGCTTCAAGAAACACGCACTGG	1920	6,36E-08	-40,64
hrpL	WT hrpL	CATCATCAGGGTTTCATC	1918	0,00000985	-28,92
hrpL	WT hrpL	AACTTGTGCTCGTTACGCAGGGCT	1911	8,35E-09	-43,85
hrpL	WT hrpL	ATCACTTCCGCAAAATGTATC	1910	0,000000173	-30,82
hrpL	WT hrpL	AGTGCGTGTTTCTTGAAGCCC	1904	0,000000173	-38,25

hrpL	WT hrpL	CTGGCATGTTGAAACTTGTGCT	1884	6,36E-08	-37,14
hrpL	WT hrpL	ACGATACATTTTGC GGAAGTG	1884	0,000000173	-33,56
hrpL	WT hrpL	TGCCACACAGCCAGGTCTGCG	1862	0,000000173	-41,05
hrpL	WT hrpL	GAAGACGAAGTGCATTCCGAGC	1861	6,36E-08	-39,74
hrpL	WT hrpL	TGAAGCCCTGCGTAACGAGCA	1854	0,000000173	-36,35
hrpL	WT hrpL	TGCGGATCAGGTTCAGCGCGAT	1844	6,36E-08	-41,84
hrpL	WT hrpL	AAGAAACACGCACTGGAGAATGTC	1836	8,35E-09	-39,88
hrpL	WT hrpL	CATCATCAGGGTTCAT	1836	0,0000253	-26,05
hrpL	WT hrpL	AGAAACACGCACTGGAGAATGT	1833	6,36E-08	-37,24
hrpL	WT hrpL	GCCGTATCAGGAAAGCTGGGAA	1823	6,36E-08	-41,74
hrpL	WT hrpL	AGACCTGGCTGTGTGGCATCG	1823	0,000000173	-42,32
hrpL	WT hrpL	AGGGCTTCAAGAAACACGCAC	1816	0,000000173	-37,76
hrpL	WT hrpL	CCACACAGCCAGGTCTGCGGTTTG	1813	8,35E-09	-44,22
hrpL	WT hrpL	GTGTTTCTTGAAGCCCTGCGT	1803	0,000000173	-38,5
hrpL	WT hrpL	ATGCCACACAGCCAGGTCTGCGGT	1793	8,35E-09	-45,58
hrpL	WT hrpL	TGTGCTCGTTACGCAGGGCTT	1790	0,000000173	-38,91
hrpL	WT hrpL	GCGTGTTTCTTGAAGCCCTGC	1783	0,000000173	-40,13
hrpL	WT hrpL	CGAGCTGGAAGGGCACGGTGA	1780	0,00000052	-43,49
hrpL	WT hrpL	TTCCAGCTTTCTGATACGGC	1779	6,36E-08	-39,96
hrpL	WT hrpL	GATACATTTTGC GGAAGTGAT	1774	0,000000173	-32,49
hrpL	WT hrpL	TTCTCCAGTGCGTGTTTCTT	1734	0,000000468	-33,96
hrpL	WT hrpL	GGATAAACGCCCTGAGCATCTG	1715	0,000139	-22,42
hrpL	WT hrpL	CAGCCAGGTCTGCGGTTTGCT	1714	0,00000156	-35,44
hrpL	WT hrpL	TGCGGTTTGCTGGCATGTGTA	1691	0,000000173	-38,31
hrpL	WT hrpL	TTGCGGATCAGGTTCAGCGCG	1689	0,000000173	-39,84
hrpL	WT hrpL	TTTGATAAACGCCCTGAGCATCT	1686	0,00000608	-26,22
hrpL	WT hrpL	ACATTCTCCAGTGCGTGTTTC	1667	0,000000173	-36,31
hrpL	WT hrpL	TGTTGAAACTTGTGCTCGTTA	1662	0,000000173	-31,66
hrpL	WT hrpL	AAAGCTGGGAAGACGAAGTGCATT	1661	8,35E-09	-42,31
hrpL	WT hrpL	CTTTTGGATAAACGCCCTGAGC	1658	0,000000572	-31,53
hrpL	WT hrpL	AATGCACTTCGTCTTCCAGCTTT	1657	8,35E-09	-41,42
hrpL	WT hrpL	TCTCCAGTGCGTGTTTCTTG	1653	0,000000468	-35,55
hrpL	WT hrpL	CGCAGGGCTTCAAGAAACACGC	1647	6,36E-08	-40,12
hrpL	WT hrpL	GATGACATTCTCCAGTGCGTGTT	1640	2,31E-08	-40,68
hrpL	WT hrpL	TCAGCGCATGCCACACAGCCAGG	1628	8,35E-09	-47,33
hrpL	WT hrpL	ATCAGGTTACGCGCATGCCAC	1628	6,36E-08	-41,72
hrpL	WT hrpL	CTGAACCTGATCCGCAATCAC	1626	0,000000173	-37,09
hrpL	WT hrpL	ACCCGCTTTTGGATAAACGCCCTG	1617	8,35E-09	-42,39
hrpL	WT hrpL	CAGCGCGATGCCACACAGCCA	1613	0,000000173	-39,8
hrpL	WT hrpL	CGCGATGCCACACAGCCAGGT	1613	0,000000173	-42,31
hrpL	WT hrpL	AAATGTATCGTCAGCCGTATCAGG	1602	8,35E-09	-41
hrpL	WT hrpL	GGTTTGCTGGCATGTTGAAAC	1599	0,000000173	-36,18
hrpL	WT hrpL	GATGAACCCTGATGATGGGA	1595	0,000000173	-37,72
hrpL	WT hrpL	TGTGGCATCGCGCTGAACCTGATC	1593	8,35E-09	-46,07
hrpL	WT hrpL	GAAACTTGTGCTCGTTACGCA	1591	0,000000173	-33,84
hrpL	WT hrpL	GTGATTGCGGATCAGGTTTCAAG	1589	0,000000173	-38,09
hrpL	WT hrpL	CCTGATACGGCTGACGATACATTT	1569	8,35E-09	-41,23
hrpL	WT hrpL	AGCCGTATCAGGAAAGCTGGGA	1555	6,36E-08	-41,82
hrpL	WT hrpL	GTTTGCTGGCATGTTGAAACT	1552	0,000000173	-34,54
hrpL	WT hrpL	GATGCCACACAGCCAGGTCTGCGG	1543	8,35E-09	-47,26
hrpL	WT hrpL	TTCAAGAAACACGCACTGGAG	1542	0,000000173	-36,42
hrpL	WT hrpL	TCCAGCTTTCTGATACGGCT	1540	6,36E-08	-40,57
hrpL	WT hrpL	TACGGCTGACGATACATTTTGC	1535	0,000000191	-34,98
hrpL	WT hrpL	GTGCCCTTCCAGCTCGGAATG	1531	0,000000173	-39,48
hrpL	WT hrpL	AAGTTTCAACATGCCAGCAAA	1527	0,000000173	-32,73
hrpL	WT hrpL	GCGCGATGCCACACAGCCAGGTCT	1525	8,35E-09	-49,04
hrpL	WT hrpL	CATTCCGAGCTGGAAGGCAC	1511	0,000000173	-40,19
hrpL	WT hrpL	TACGCAGGGCTTCAAGAAACACGC	1499	8,35E-09	-42,63
hrpL	WT hrpL	ACGCAGGGCTTCAAGAAACACG	1485	6,36E-08	-39,99
hrpL	WT hrpL	ATTCTCCAGTGCGTGTTCCTTGAA	1483	8,35E-09	-40,13
hrpL	WT hrpL	ATGCCACACAGCCAGGTCTG	1475	0,000000468	-38,7
hrpL	WT hrpL	TGTGTGGCATCGCGCTGAACC	1473	0,000000173	-40,15
hrpL	WT hrpL	GCCAGGTCTGCGGTTTGCT	1472	0,00000125	-37,73
hrpL	WT hrpL	TGCTGGCATGTTGAAACTTGTGC	1454	2,31E-08	-40,19
hrpL	WT hrpL	CTTTTGGATAAACGCCCTGAGCAT	1416	0,000000676	-30,17
hrpL	WT hrpL	CAAGAAACACGCACTGGAGAATGT	1396	8,35E-09	-40,74
hrpL	WT hrpL	GCAAAATGTATCGTCAGCCGTA	1394	0,000000191	-35,02
hrpL	WT hrpL	AGAAACACGCACTGGAGAATG	1375	0,000000173	-36,34
hrpL	WT hrpL	TTTCAACATGCCAGCAAAACCGC	1372	6,36E-08	-37,69
hrpL	WT hrpL	TTCAAGAAACACGCACTGGAG	1371	0,00000052	-36,05
hrpL	WT hrpL	CACATCATCAGGGTTCATCAC	1345	0,000000173	-37,17
hrpL	WT hrpL	GCCACACAGCCAGGTCTGCGGT	1343	6,36E-08	-42,88
hrpL	WT hrpL	CTTGTGCTCGTTACGCAGGGCTT	1334	2,31E-08	-42,38
hrpL	WT hrpL	TCAGGTTCAGCGCATGCCAC	1329	0,00000052	-40,22

hrpL	WT hrpL	TGACATTCTCCAGTGC GTGTTTCT	1325	8,35E-09	-42,07
hrpL	WT hrpL	AGCAAACCGCAGACCTGGCTG	1313	0,00000052	-39,66
hrpL	WT hrpL	GGGCTTCAAGAAACACGCACTG	1305	6,36E-08	-40,49
hrpL	WT hrpL	CAAGTTTCAACATGCCAGCAA	1305	0,000000173	-34,51
hrpL	WT hrpL	ATTGCGGATCAGGTT CAGCGCG	1302	0,000000191	-40,06
hrpL	WT hrpL	CACGCACTGGAGAATGTCATCCAC	1299	8,35E-09	-42,91
hrpL	WT hrpL	ACGCACTGGAGAATGTCATC	1293	0,000000468	-34,83
hrpL	WT hrpL	TGATCCGCAATCACTTCGCGCA	1292	0,000000173	-36,63
hrpL	WT hrpL	TGAACCCTGATGATG	1289	0,0000633	-24,88
hrpL	mut hrpL	TTGGGAGGATGAGGTTCACTC	74216	0,031	-23,24
hrpL	mut hrpL	AGTTGGGAGGATGAGGTT CAC	9839	0,031	-27,67
hrpL	mut hrpL	TCCCAACTCTCTTG GTAAGGT	8784	0,031	-18,25
hrpL	mut hrpL	ACCTTACCAAGAGAGTTGGGA	8634	0,031	-19,2
hrpL	mut hrpL	TGACGGCCGATCAGATT CAGA	8383	0,003	-17,72
hrpL	mut hrpL	TGAATCTGATCGGCCGTCATT	7778	0,01	-23,16
hrpL	mut hrpL	AATGACGGCCGATCAGATTCA	7705	0,01	-15,67
hrpL	mut hrpL	TCAACGTCGTCCGGATT CATAA	7264	0,034	-17,09
hrpL	mut hrpL	CAACGTCGTCCGGATT CATAA	6913	0,092	-17,42
hrpL	mut hrpL	CGTCGTCCGGATT CATACTC	6376	0,092	-12,82
hrpL	mut hrpL	AATCTGATCGGCCGTCATTTG	4620	0,031	-20,67
hrpL	mut hrpL	CCCAACTCTCTTG GTAAGGT	4348	0,092	-17,15
hrpL	mut hrpL	AACCTTACCAAGAGAGTTGGG	4304	0,092	-16,79
hrpL	mut hrpL	TCAACGTCGTCCGGATT CATA	3991	0,031	-17,1
hrpL	mut hrpL	CGTCGTCCGGATTCA	3153	0,42	-12,84
hrpL	mut hrpL	CTGAATCTGATCGGCCGTCATT	3141	0,004	-25,72
hrpL	mut hrpL	AATGACGGCCGATCAGATT CAG	3135	0,004	-18,26
hrpL	mut hrpL	GAGTTGGGAGGATGAGGTTCA	2442	0,031	-28,38
hrpL	mut hrpL	GTCAACGTCGTCCGGATT CAT	2264	0,031	-17,4
hrpL	mut hrpL	AAGAGAGTTGGGAGGATGAGG	2207	0,092	-30,43
hrpL	mut hrpL	CGTCGTCCGGATT CATAA	1987	0,19	-14,97
hrpL	mut hrpL	CGTCGTCCGGATT CATA	1918	0,19	-15
hrpL	mut hrpL	CGTCGTCCGGATT CAT	1836	0,17	-13,93
hrpL	mut hrpL	AGAGTTGGGAGGATGAGGTTCACT	1661	0,04	-29,64
hrpL	mut hrpL	CTGAATCTGATCGGCCGTCAT	1626	0,003	-25,01
hrpL	mut hrpL	ATGACGGCCGATCAGATT CAG	1589	0,003	-18,26
hrpL	mut hrpL	AACCTTACCAAGAGAGTTGGGA	1555	0,034	-18,68
hrpL	mut hrpL	TCCCAACTCTCTTG GTAAGGT	1540	0,034	-18,94
hrpL	mut hrpL	AACGTCGTCCGGATT CATAAC	1345	0,031	-17,46
hrpL	mut hrpL	TGAATCCGACGACG	1289	0,42	-10,13

Oligo name	Oligo Sequence (5'-3')	Usage
GyrA-F	AACTGCTGGGTGAGTACCA	qPCR of bacterial transcript <i>gyrA</i>
GyrA-R	GAGCTCTTCGCGGATCACT	
ProC-F	CGCAGATGATGAAAAGCGTC	qPCR of bacterial transcript <i>proC</i>
ProC-R	AGTCAGGCTGGCACAGGTG	
RpoB-F	GTAGGTCTGGTCCGTGTTGA	qPCR of bacterial transcript <i>rpoB</i>
RpoB-R	GCAAGTAATCTCGGACAGCG	
Cfa6-F	GTCTTCATCTTTCCCGTCA	qPCR of bacterial transcript <i>cfa6</i>
Cfa6-R	GTCTCGATCTGGTCGATGGT	
HrpL-F	CGAGTCATTAGGCCATTGATT	qPCR of bacterial transcript <i>hrpL</i>
HrpL-R	GTTTCCTGATAATTGCCGTCCA	
luxA-F	CGGAGTTTGTTTGCTTGGT	qPCR of bacterial transcript <i>luxA</i>
luxA-R	CAAGTTGGCGTACTGGATGG	
luxB-F	GCGGAGGAAGCTTGCTTATT	qPCR of bacterial transcript <i>luxB</i>
luxB-R	TGATATTCAACCGGGCGATT	
Nb-IUXA/LUXB-F	TGCTTACATACCAACCTCCCAATT	Template DNA amplification for siRNA probe preparation for northern blot analysis
Nb-IUXA/LUXB-R	GGCGCGCCCAGGATGATGAGTTGTA	
Nb-CFA6-F	CAACAACCTGGCGGGATCCTG	Template DNA amplification for siRNA probe preparation for northern blot analysis
Nb-CFA6-R	GGCTTGCATCCGACAGAATCAG	
Nb-HRPL-F	CACCTCGCTTCCCAGCTTTC	Template DNA amplification for siRNA probe preparation for northern blot analysis
Nb-HRPL-R	TTTATCCAAAAGCGGGTGATGAAC	
Nb-CYP51-F	ATCCGAGCATGACATGATGA	Template DNA amplification for siRNA probe preparation for northern blot analysis
Nb-CYP51-R	GTACGGGTCGATGCCATATT	
U6	AGGGGCCATGCTAATCTTCTC	probe for northern blot analysis
tomato-Ubi-F	GGACGGACGTACTCTAGCTGAT	qPCR on genomic DNA from Tomato infected with <i>Pto</i> DC3000
tomato-Ubi-F	AGCTTTCGACCTCAAGGGTA	
Pto-GFP-F	TGGAAGCGTTCAACTAGCAG	qPCR on genomic DNA from Tomato infected with <i>Pto</i> DC3000
Pto-GFP-R	AAAGGGCAGATTGTGTGGAC	
IR-CFA6/HRPL-F	GTTTCATCACCGCTTTTGGTA	qPCR of the IR-CFA6/HRPL precursor
IR-CFA6/HRPL-R	CCCCTCTTTCTACCTTCCCA	
Ath-Ubi-F	TGAAGTCGTGAGACAGCGTTG	qPCR of the IR-CFA6/HRPL precursor
Ath-Ubi-F	GGGCTTTCTCATTGTTGGTC	
HRPL-pDON207-F	AAAAAGCAGGCTTCATGTTTCAGAAGATTGTGATC	Cloning of WT <i>HRPL</i> and mut <i>HRPL</i> in pDON207-attB1/B2
HRPL-pDON207-R	AGAAAGCTGGGTGCGCGAACGGGTCGATTGCTG	
IR-HRPL-F	aGGTCTCaCTGCCTGATGACTGACATCACCGTG	Cloning of IR- <i>HRPL</i> region in a "E" Green Gate module
IR-HRPL-R	aGGTCTCaTAGTCAGGGCGTTTATCCAAAAGCG	
dcl2-1-WT-FP	TGAATCATCTGGAAGAGGTGG	genotyping of <i>dcl2-1</i> allele
dcl2-1-mut-FP	ATTTTGCCGATTTTCGGAAC	
dcl2-1-WT-RP	TGAATCATCTGGAAGAGGTGG	genotyping of <i>dcl3-1</i> allele
dcl3-1-LP	ACAGGTAACCTTGCCATGTTG	
dcl3-1-RP	TGGAAAAGTTTGCTACAACGG	genotyping of <i>dcl4-2</i> allele
LBa1	TGGTTCACGTAGTGGGCCATCG	
dcl4-2-G8605-FP	GGCTGCACAGCTGATGATTACAA	genotyping of <i>dcl4-2</i> allele
dcl4-2-G9512-RP	GCCGCTCGAGATCATCAGCAAAGGAAT	
GABI-8474-LP	ATAATAACGCTGCGGACATCTACATTTT	