4-1-2014

IDENTIFICATION OF ARABIDOPSIS MUTANTS WITH ENHANCED STOMATAL IMMUNITY

Samantha Trinh

IDENTIFICATION OF ARABIDOPSIS MUTANTS WITH ENHANCED STOMATAL IMMUNITY

by

SAMANTHA THANH TRINH

Presented to the Faculty of the Honors College of

The University of Texas at Arlington in Partial Fulfillment

of the Requirements

for the Degree of

HONORS BACHELOR OF SCIENCE IN MEDICAL TECHNOLOGY

THE UNIVERSITY OF TEXAS AT ARLINGTON

April 2014

ACKNOWLEDGMENTS

First and foremost, I would like to thank my mentor, Dr. Maeli Melotto, for her guidance and encouragement. Her motivation and assistance made the completion of this project possible. Also, I thank all the members of the lab, including Lenore Price, Giselle de Carvalho, Cleverson Matiolli, Paula Oblessuc, Nisita Obulareddy, Shweta Panchal, Debanjana Roy, Blaine Thompson, Bich Ta, and Hannah Webber. I give special thanks to my lab partner, Anh Le, for his hard work and dedication. I am grateful for their contributions, and they have truly made being part of the lab a wonderful experience. Last but not least, I wish to express my gratitude to my wonderful family for their love and support.

April 6, 2012

ABSTRACT

IDENTIFICATION OF ARABIDOPSIS
MUTANTS WITH ENHANCED
STOMATAL IMMUNITY

Samantha Thanh Trinh, B.S.

The University of Texas at Arlington, 2012

Faculty Mentor: Dr. Maeli Melotto

Bacteria cannot directly penetrate the epidermis of a plant, so they rely on stomatal pores to enter the leaves. These surface openings play an important role in limiting bacterial invasion. The guard cells that form the stomatal pore can sense bacterial molecules, such as pathogen-associated molecular patterns (PAMPs) and close the stomata by changing the turgor pressure of the guard cells. In this study, we are using a genetic approach to identify genes that are involved in stomatal immunity. The bacterium pathogen *Pseudomonas syringae* pv. *tomato* strain DC3118 has been shown to induce strong stomatal closure in the wild type Arabidopsis plant Col-0. Using a collection of homozygous T-DNA insertion lines that represent mutations in 10,848 different genes of Arabidopsis, we are screening plants for high leaf surface temperature after inoculation

(*i.e.* indicative of closed stomata) using an infrared camera (Flir System T300). Plants with mutations in genes involved in bacterium-triggered stomatal closure should have leaves hotter than the Col-0 wild type plant. To date, we have screened 1,117 mutant plants and selected 77 lines for further investigation. This screen will further our current understanding of the genetic regulation of stomatal immunity and provide insights for additional and/or complementary control measures to alleviate plant diseases in the field. Furthermore, stomatal immunity may be an important mechanism to prevent fresh produce contamination with human pathogens, thus benefiting human health.

TABLE OF CONTENTS

LIST OF ILLUSTRATIONS

LIST OF TABLES

CHAPTER 1

INTRODUCTION

The interior of a leaf is mostly made up of mesophyll cells, vascular tissues, and intercellular space between mesophyll cells. The mesophyll cells are where most of the photosynthate is made. The intercellular space, also known as the apoplast, contains some nutrients. Pathogenic bacteria can multiply in the apoplast, leading to disease under the right conditions. Pathogens must cross the surface cuticle and epidermis in order to gain access to the intercellular spaces and internal leaf tissues. Fungal plant pathogens have the ability to directly penetrate the epidermis using cuticle-degrading enzymes, cell wall-degrading enzymes, or mechanical force. However, bacteria cannot directly penetrate the leaf epidermis and must enter the leaf tissues through natural surface openings, such as hydathodes, stomata, nectarthodes, and lenticels. Stomata are one of the most important routes for the entry of bacterial pathogens (Underwood et al, 2007).

Stomata are distributed across the leaf epidermis with characteristic spacing, and they are formed by asymmetric divisions of epidermal cells. Intercellular signaling provides spatial signs that regulate division orientation and may block asymmetric division in cells adjacent to two stomata (Nadeau and Sack, 2002). The pores allow for the conduction of water transpiration and gas exchange for photosynthesis and allow the entry of many microbes into the plant. The surface openings were previously thought as passive portals of entry for plant pathogenic bacteria, but the stomata actually plays an important role in limiting bacterial invasion (Melotto et al., 2006).

Plants have the ability to close stomata by changing the turgor of the guard cells that form the stomata. The guard cells can sense bacterial molecules, such as pathogen-associated molecular patterns (PAMPs). The plant hormone abscisic acid (ABA) is important in stress-related signaling pathways in plants and guard cell signaling that leads to stomatal closure. The ABA signal in guard cells is transduced through the production of compounds such as nitric oxide (NO) and H_2O_2 signalling intermediaries such as the guard cell-specific OST1 kinase, changes in cytosolic Ca^{2+} levels, and Ca^{2+} oscillations. The defense-signaling molecule salicylic acid (SA) also controls the stomatal closing. It has been shown that incubating Arabidopsis leaves with *Escherichia coli* and *Pseudomonas syringae* pv. *tomato* (*Pst*) DC3000 results in a reduced number of open stomata from 70-80% to 30% within 1 to 2h that is correlated with a decrease in the width of the stomatal aperture (Melotto et al., 2006).

In turn, plant bacteria have evolved to be able to cause stomata to reopen. It is known that PAMP-induced basal defense happens during the early stages of plant bacterial infection. For example, *Pst* DC3000 has evolved a natural virulence mechanism to counter stomatal closure. The stomata re-opened 3h after incubation with plant leaves. Coronatine (COR), a non-host-specific polyketide toxin consisting of coronafacic acid and coronamic acid, is produced by at least five different pathovars (pvs) of *P. syringae*, including pvs. *tomato*, *glycinea*, *atropurpurea*, *maculicola*, and *morsprunorum*. COR is a virulence factor that suppresses PAMP-induced stomatal defense, and COR-mediated suppression of stomatal defense is necessary for *Pst* DC3000 to infect the plants. COR also suppresses local and systemic host defenses and promotes disease symptoms. It is

likely that other phytopathogenic bacteria use specific virulence factors to circumvent stomatal closure, and COR is probably only one of several virulence factors used by bacteria to overcome stomatal defences. Bacteria that lack the ability to overcome stomatal defense have to wait for favorable environmental conditions or rely on other entry sites, such as wounds. Heavy rains, high humidity, and frost damage all promote stomatal opening. On the other hand, abiotic stresses, like drought, cause stomatal closure. Environmental factors such as light, humidity, and CO_2 concentration also affect the opening and closing of stomata (Underwood et al., 2007).

CHAPTER 2

MATERIALS AND METHODS

Plant material

Vermiculite, perlite, and growing mix are combined in one part each to prepare the soil. Twenty-one 3x3 inch-square pots are filled with the soil and placed into a porous black tray on top of a green tray. The pots are then soaked with approximately 1 gallon of water overnight. The following day, about ½ inch of vermiculite dampened with water is added on top of the filled pots. The pots are covered with a 5x5 inch square of mesh to prevent the soil from falling out during inoculation, and then secured with a rubber band. Sowing seeds requires a well plate, 0.1% agarose gel, seeds, a micropipette, and pipette tips. The micropipette is adjusted to 100 µL using the measuring dial and used to distribute agarose gel into each well. After three to four seeds of each type are put into the respective well, the liquid and seeds are absorbed into the pipette and dispensed into the pots. Lastly, the pots are labeled by plant type and date.

Plant growth conditions

Arabidopsis plants are left in the cold room at 4°C for two days, and then moved to the 22°C growth chamber. The initial watering is with 300 mL of gnatrol (1g gnatrol/4 L water) and then the plants are watered periodically as necessary. After about one to two weeks, the plants are ready to be thinned. The plants are grown at a 12h photoperiod under light intensity of 100 $\mu mol.m^{-2}.s^{-1}$. After five weeks, the plants are used for pathogenicity assays.

Pathogenicity assay

To prepare the pre-inoculum, a small colony of *Pst* DC3118 (a coronatine-defective mutant) is placed into a test tube containing 10 mL of low-salt Luria-Bertani (LB) broth with 10 µL of 50 µg/mL kanamycin and 50 µL of 100 µg/mL rifampicin. The test tube is then placed in the 30ºC incubator at 225 rpm for the bacteria to grow. The inoculum can be prepared by diluting the pre-inoculum to 0.003 OD with 300 mL of LB broth and putting back into the 30ºC incubator for overnight culturing. The evening before the experiment, the plants are moved to the 25ºC walk-in growth chamber and loosely covered with a plastic cover with a 2 inch opening. After making sure the OD of the DC3118 is between 0.6 and 1.0 in the following day, the suspension is centrifuged for 20 minutes at 2600 rpm speed. Then, water and 0.03% silwet are mixed with the cell pellets to a concentration of 0.4 for dip inoculation. Arabidopsis plants are dipped into the bacterial suspension and incubated in the walk-in growth chamber under the following conditions: 25ºC under light intensity of 100 $\mu mol.m^{-2}.s^{-1}$.

Analysis of stomatal response to *Pst* DC3118

After two hours of post inoculation, pictures are taken in the walk-in growth chamber using an infrared camera. The picture number and plant identification are recorded on a spreadsheet. Using the Flir program, all pictures are set at constant range of temperature to identify possible mutant plants that look hotter than Col-0 plant. The inoculated plants are left in the walk-in growth chamber for one week to check for symptoms.

Plating technique

After leaving the bacteria to incubate over a 48h period, plating is done to determine the number of bacteria able to penetrate the leaf surface and enter the plant using dilutions. Three leaves are picked from the plant and surface sterilized by washing the leaves in 70% ethanol for 3 minutes and rinsing in water for 1 minute. The leaves are laid out on a paper towel, and a #2 cork borer is used to punch four holes per leaf for a total of twelve discs. Four discs are placed into each of the three USA Scientific centrifuge tubes containing 100 µL of sterile water, and a drill is used to grind the leaf tissue. Three samples of six dilutions each are placed on an LB plate. Colonies begin to form on the plate after twenty-four hours of incubation at 30°C and 225 rpm. The colonies are viewed under a microscope and counted. The number of colonies for mutants can be compared to the number of colonies for Col-0 to verify stomatal immunity.

CHAPTER 3

RESULTS

Using mutations in 10,848 different genes of Arabidopsis, we screened plants for high leaf surface temperature after inoculation using an infrared camera and Flir System T300. Mutant plants (1,117) have been screened, and 77 lines were selected for further investigation. All of the mutant plants are compared to Col-0. Plants that have higher leaf surface temperature than Col-0 are recorded on a spreadsheet. Higher leaf surface temperature than Col-0 relates to closed stomata, while lower leaf surface temperature than Col-0 is equivalent to open stomata. The exact temperature varies across experiments due to different experimental conditions. The walk-in growth chamber has a slight fluctuation from 25°C. Breathing on the plants and moving them around also affects the leaf surface temperature.

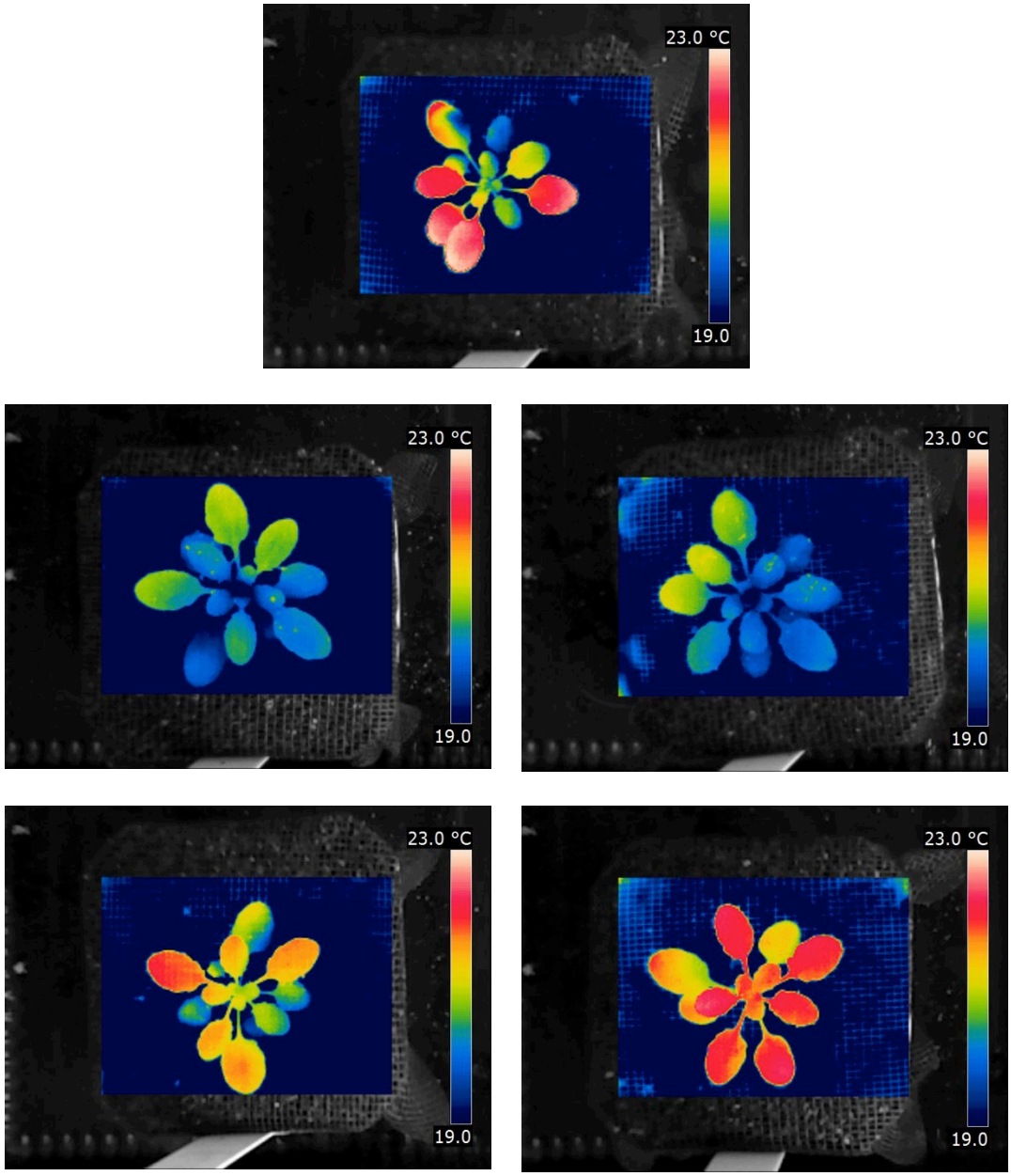

Figure 1. Infrared pictures taken of Col-0 (row 1), Arabidopsis plants with low leaf surface temperature (row 2), and Arabidopsis plants with high leaf surface temperature (row 3) 2h after inoculated with *Pst* DC3118.

In Figure 1, all the pictures have the same temperature range of 19.0ºC-23.0ºC in order to analyze and compare to Col-0. At1g15640 and At3g62340 have lower leaf surface temperature than Col-0 two hours after being inoculated with *Pst* DC3118. Therefore, they have open stomata. At3g22845 and At1g73210 have higher leaf surface temperature than Col-0, meaning they have closed stomata.

The experiment was repeated with the selected mutant plants to confirm results in a second biological replicate. Of the 77 lines selected for further investigation, 10 of them had higher leaf surface temperature than Col-0 and were in agreement with the previous experiments. Infrared pictures were taken before and after dip inoculation to ensure that stomatal closure is from *Pst* DC3118 and not from some other factor, such as closed stomata before the dip and different metabolic rates. It can be seen in Figures 2-6 and Figures 8-10 that before inoculation, Col-0 and the mutant plants had open stomata. Regarding the mutants with stomatal immunity, the stomata of the plants closed 2h after inoculation. At3g22845, At1g73210, At3g01910, At2g06650, At3g48480, At5g25320, At1g60120, At1g75930, At4g29000, and At4g17980 were found to have high leaf surface temperature 2h post inoculation.

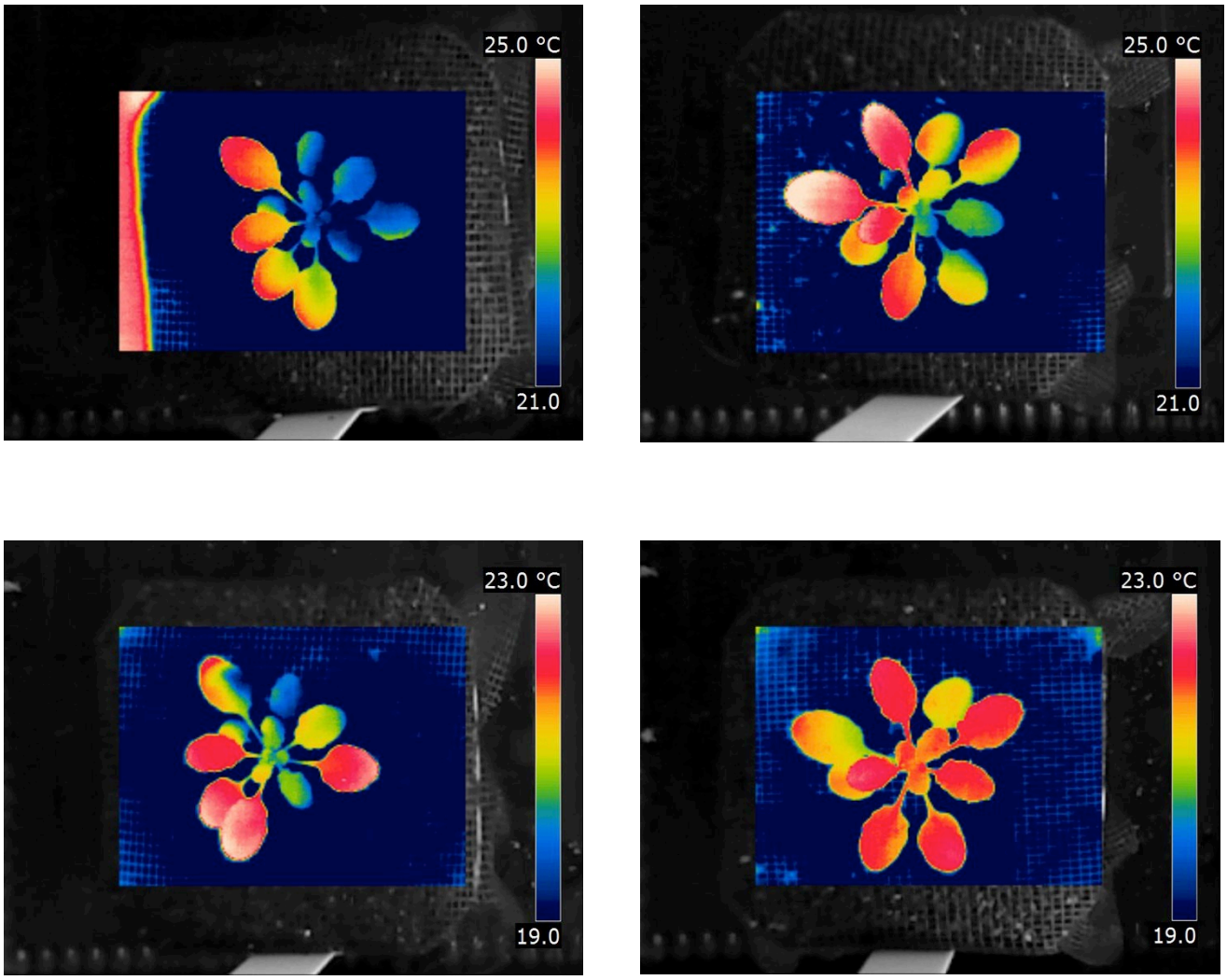

Figure 2. Col-0 (row 1, left) and At3g22845 (row 1, right) before inoculation and Col-0 (row 2, left) and At3g22845 (row 2, right) 2h after inoculation with *Pst* DC3118. At3g22845 has higher leaf surface temperature than Col-0 indicating closed stomata.

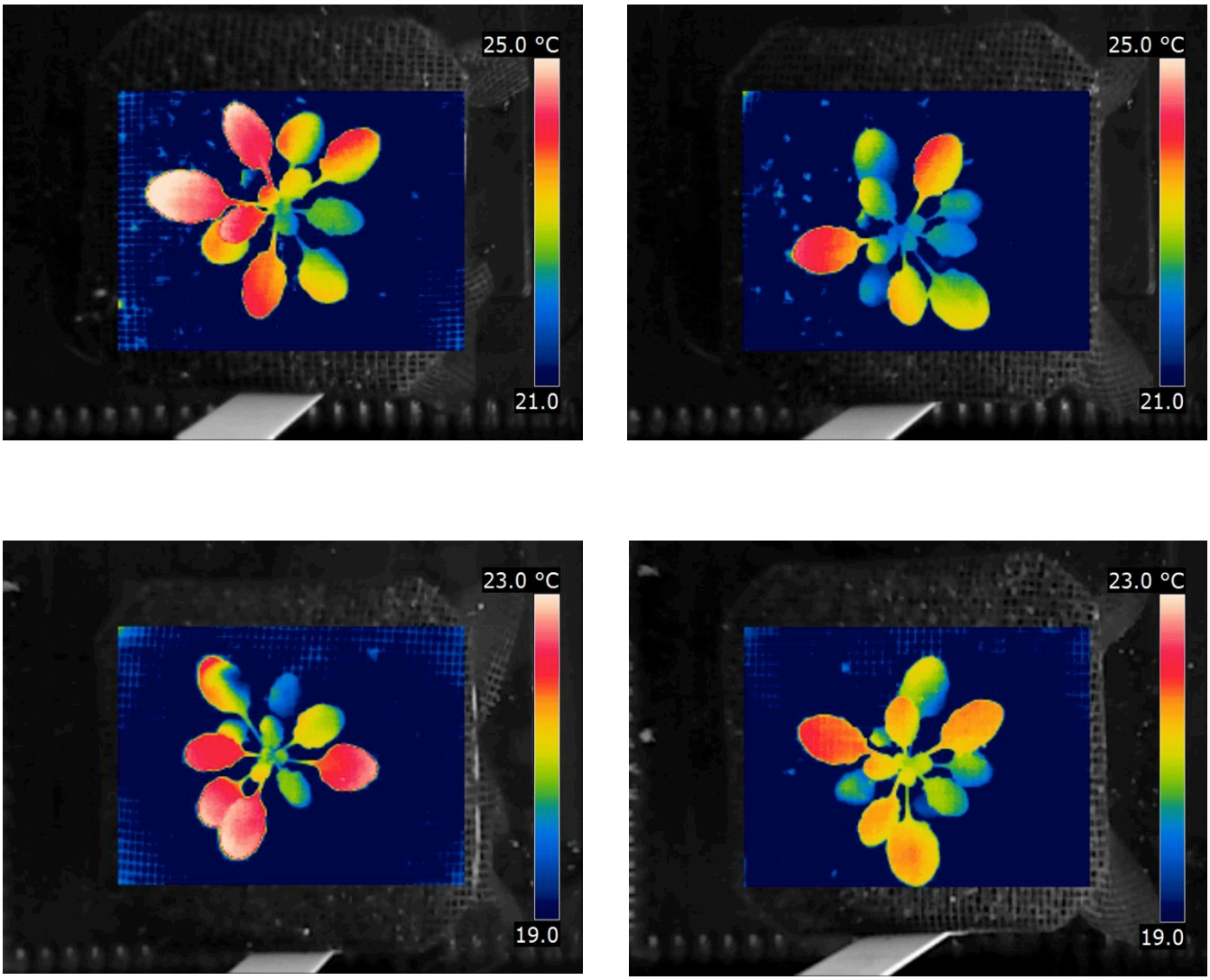

Figure 3. Col-0 (row 1, left) and At1g73210 (row 1, right) before inoculation and Col-0 (row 2, left) and At1g73210 (row 2, right) 2h after inoculation with *Pst* DC3118. At1g73210 has higher leaf surface temperature than Col-0 indicating closed stomata.

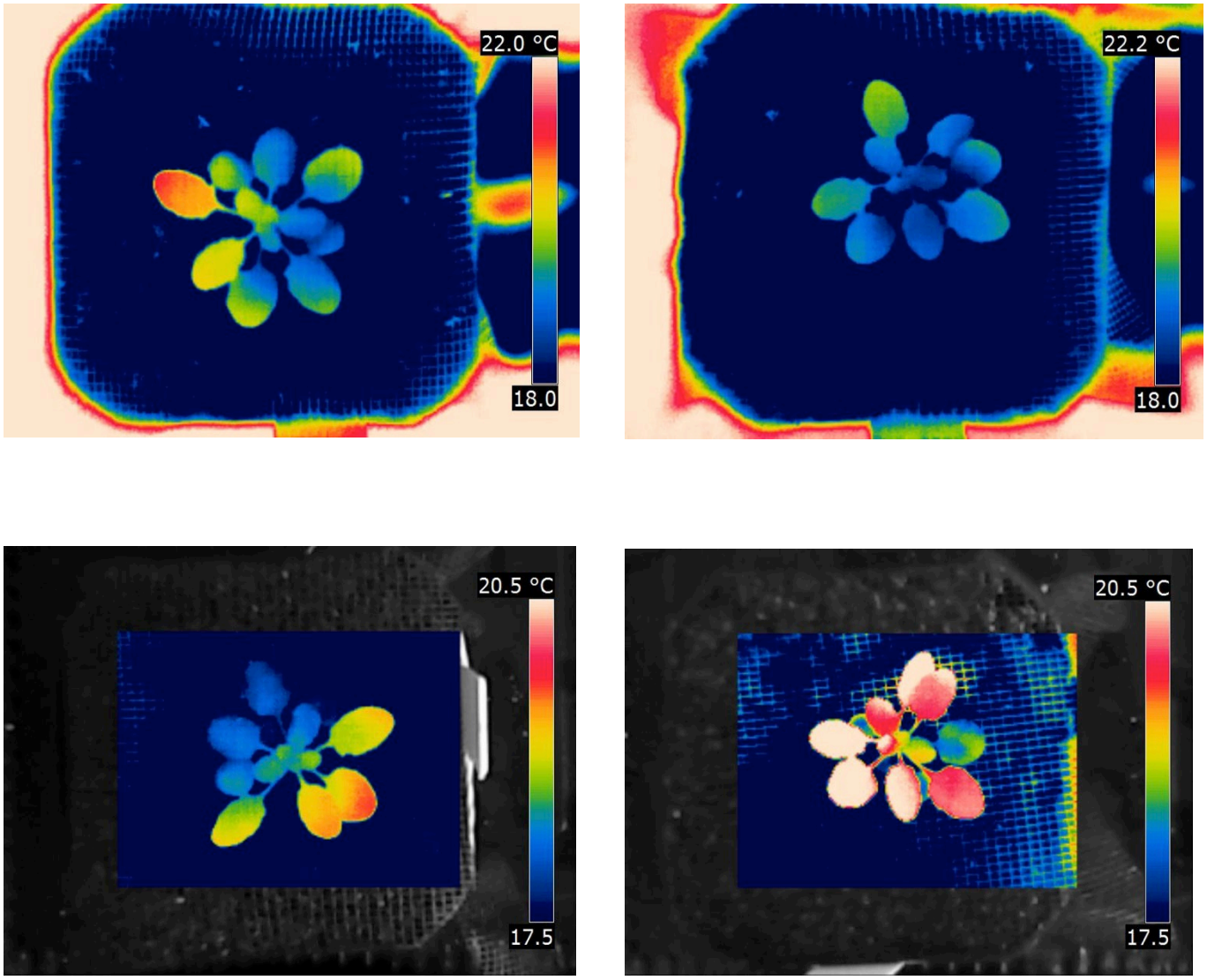

Figure 4. Col-0 (row 1, left) and At3g01910 (row 1, right) before inoculation and Col-0 (row 2, left) and At3g01910 (row 2, right) 2h after inoculation with *Pst* DC3118. At3g01910 has higher leaf surface temperature than Col-0 indicating closed stomata.

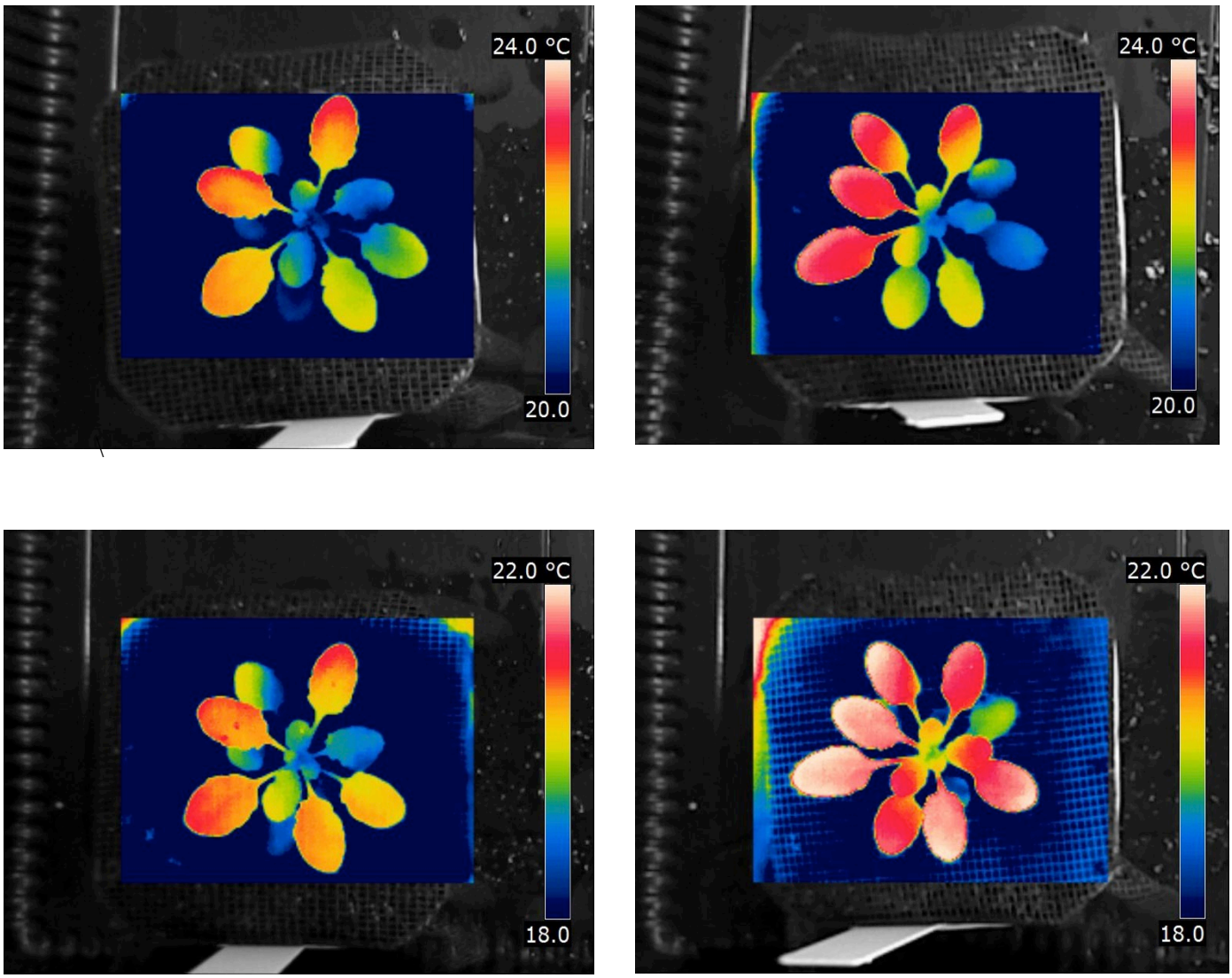

Figure 5. Col-0 (row 1, left) and At2g06650 (row 1, right) before inoculation and Col-0 (row 2, left) and At2g06650 (row 2, right) 2h after inoculation with *Pst* DC3118. At2g06650 has higher leaf surface temperature than Col-0 indicating closed stomata.

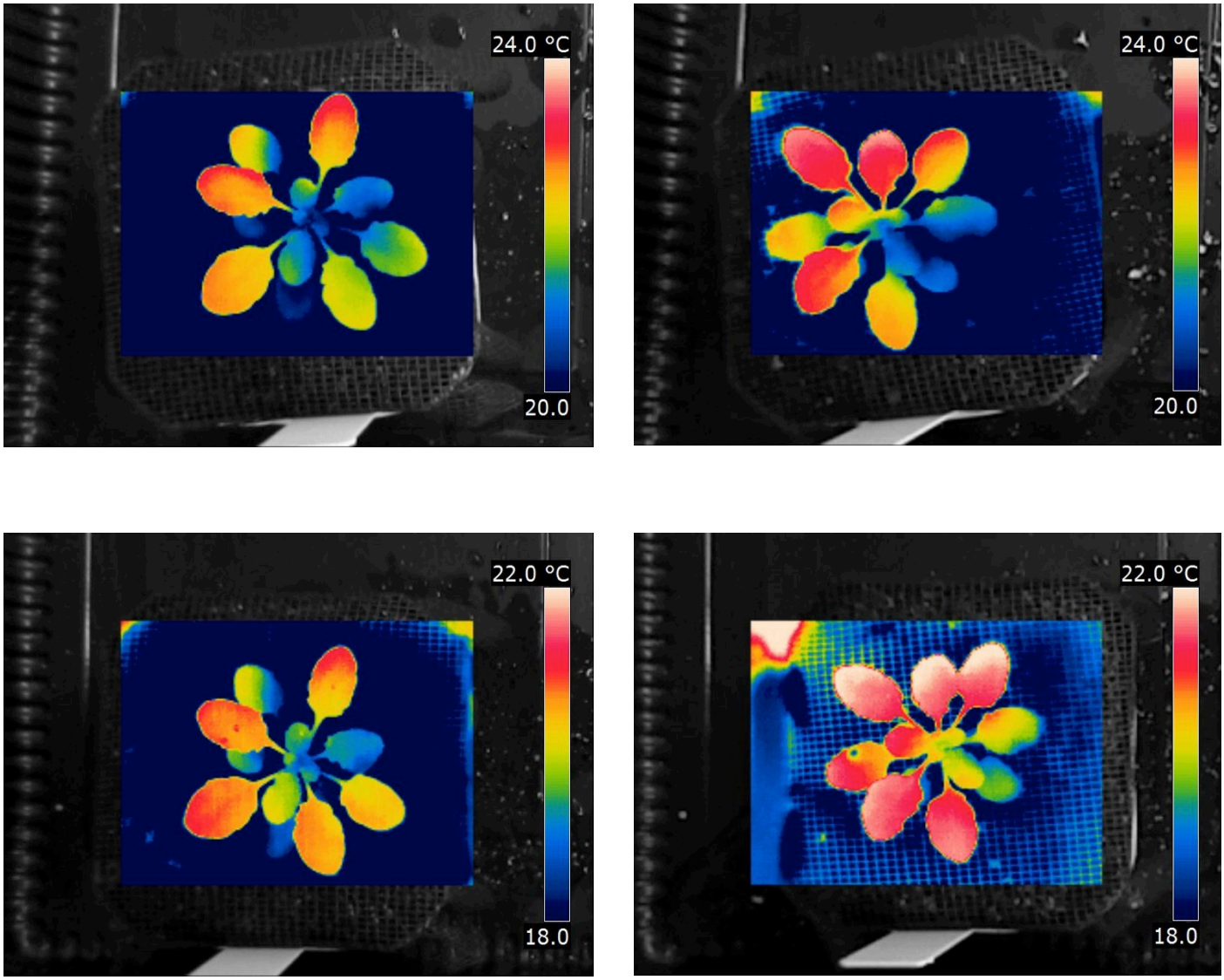

Figure 6. Col-0 (row 1, left) and At3g48480 (row 1, right) before inoculation and Col-0 (row 2, left) and At3g48480 (row 2, right) 2h after inoculation with *Pst* DC3118. At3g48480 has higher leaf surface temperature than Col-0 indicating closed stomata.

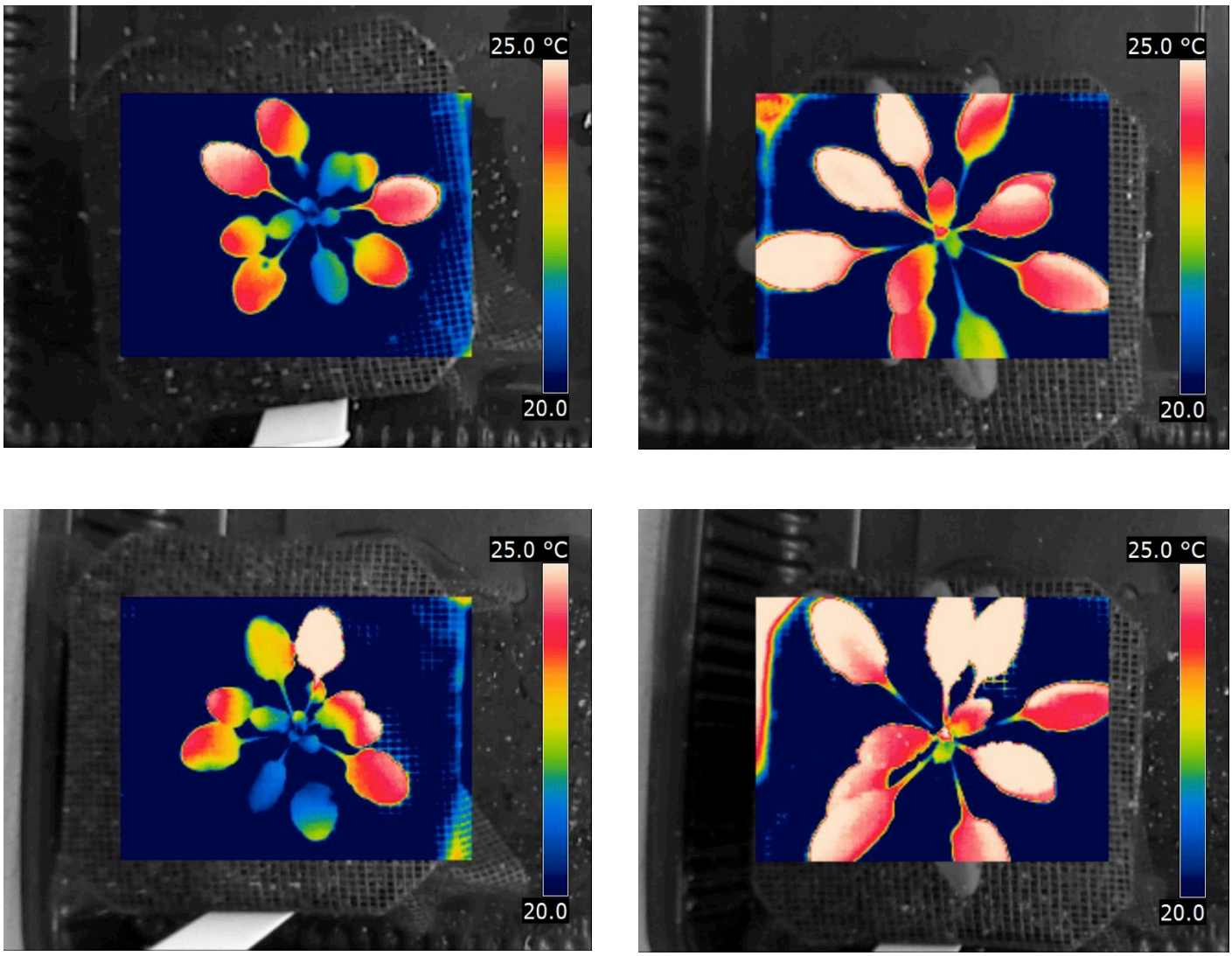

Figure 7. Col-0 (row 1, left) and At5g25320 (row 1, right) before inoculation and Col-0 (row 2, left) and At5g25320 (row 2, right) 2h after inoculation with *Pst* DC3118. At5g25320 has higher leaf surface temperature than Col-0 indicating closed stomata.

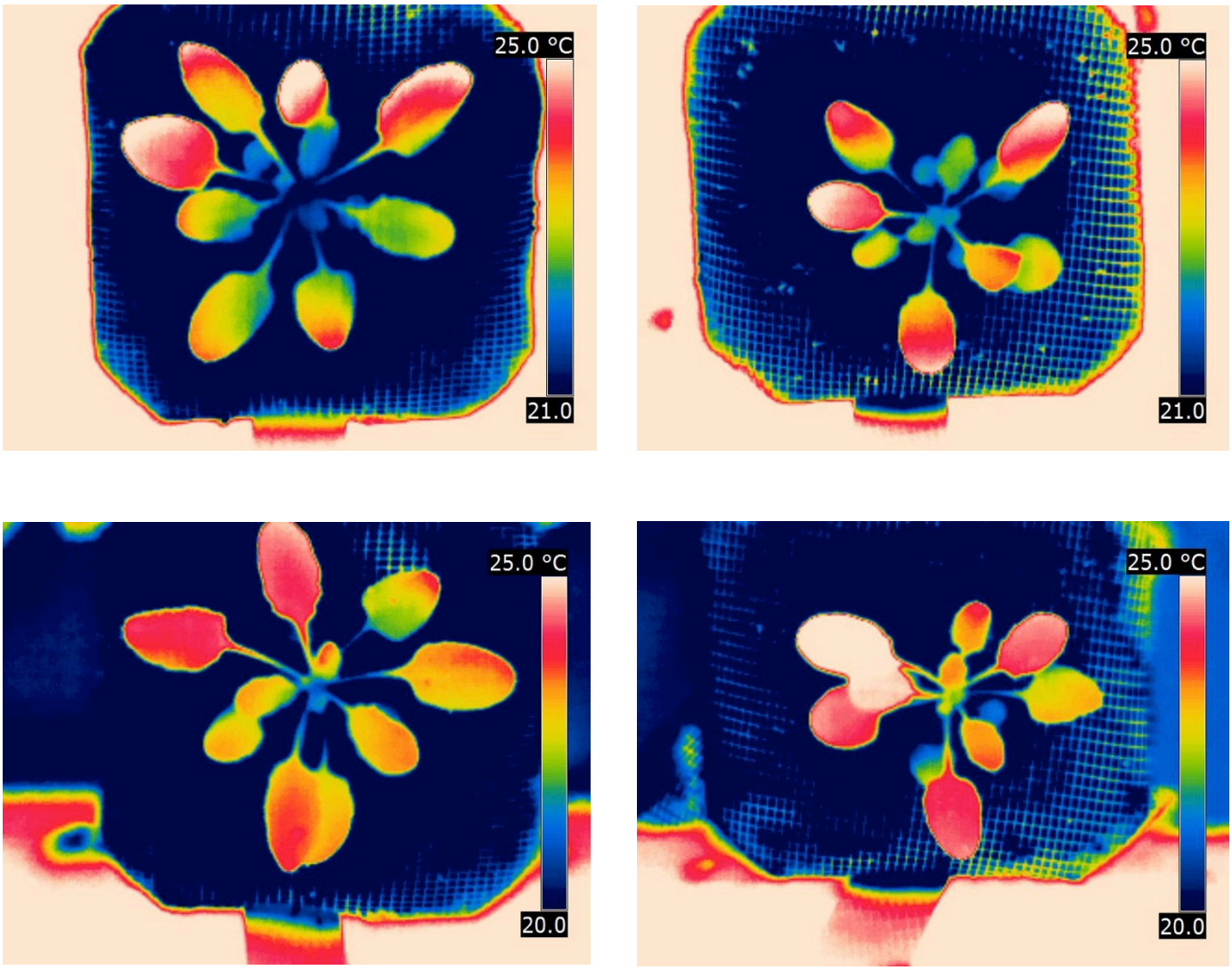

Figure 8. Col-0 (row 1, left) and At1g60120 (row 1, right) before inoculation and Col-0 (row 2, left) and At1g60120 (row 2, right) 2h after inoculation with *Pst* DC3118. At1g60120 has higher leaf surface temperature than Col-0 indicating closed stomata.

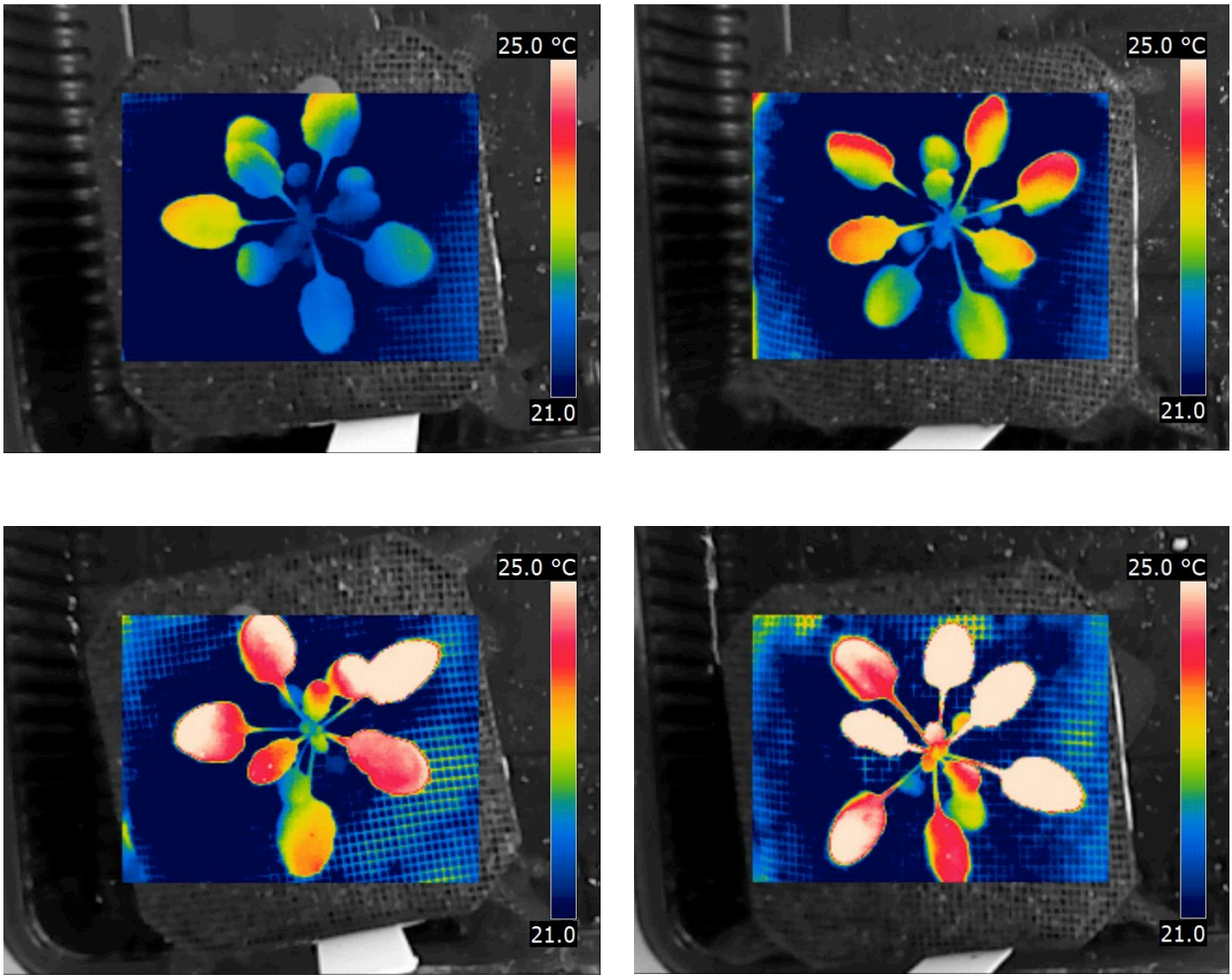

Figure 9. Col-0 (row 1, left) and At1g75930 (row 1, right) before inoculation and Col-0 (row 2, left) and At1g75930 (row 2, right) 2h after inoculation with *Pst* DC3118. At1g75930 has higher leaf surface temperature than Col-0 indicating closed stomata.

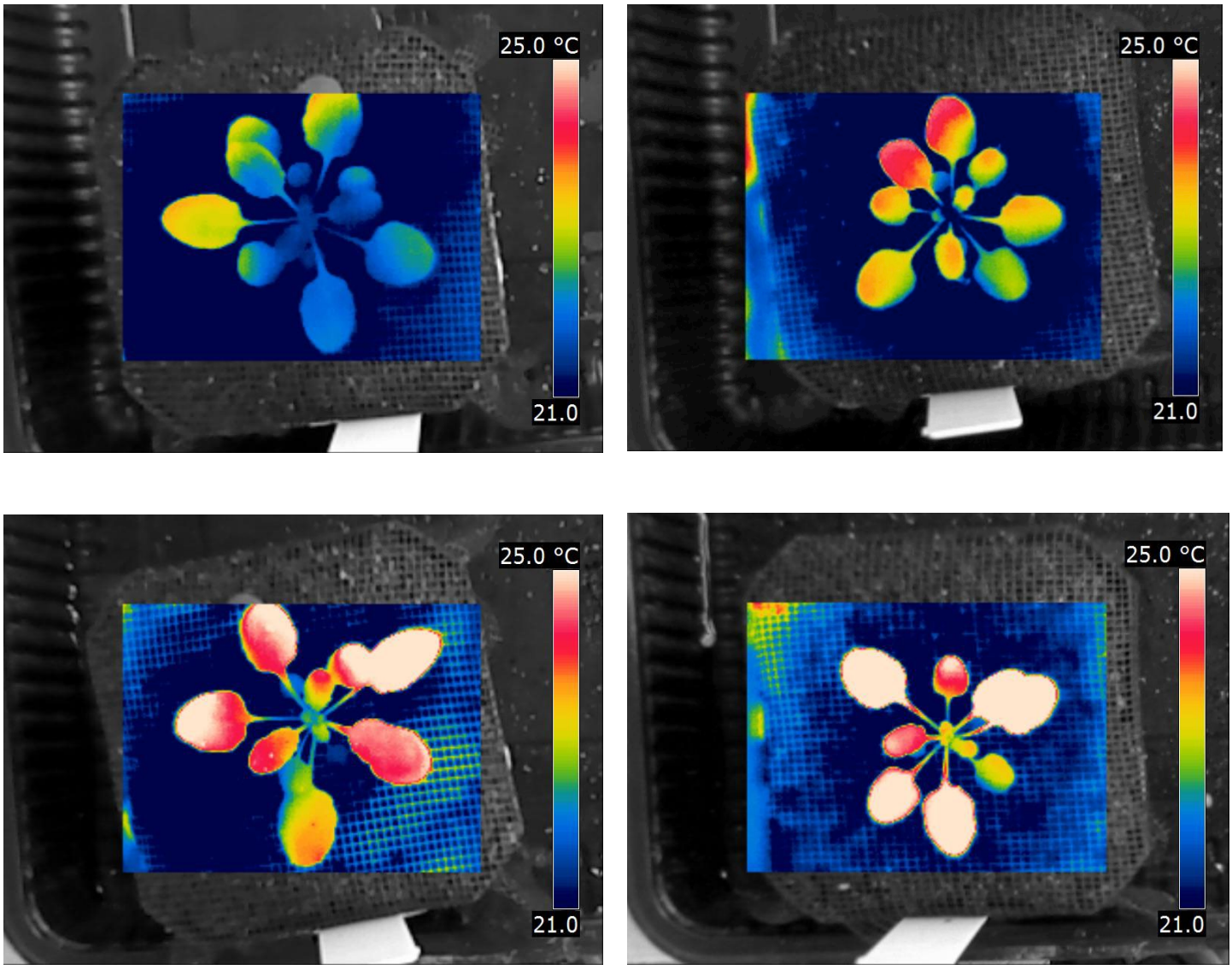

Figure 10. Col-0 (row 1, left) and At4g29000 (row 1, right) before inoculation and Col-0 (row 2, left) and At4g29000 (row 2, right) 2h after inoculation with *Pst* DC3118. At4g29000 has higher leaf surface temperature than Col-0 indicating closed stomata.

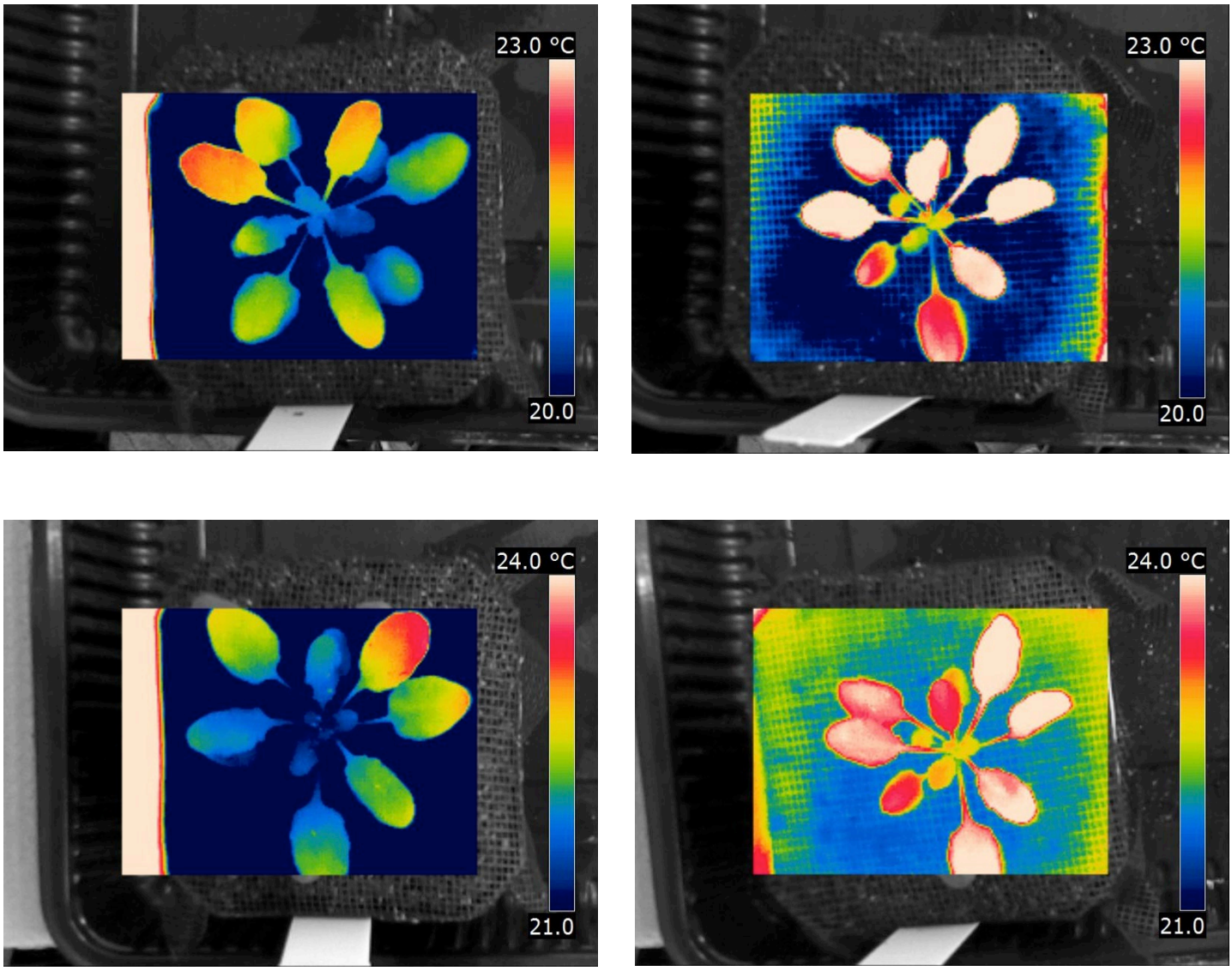

Figure 11. Col-0 (row 1, left) and At4g17980 (row 1, right) before inoculation and Col-0 (row 2, left) and At4g17980 (row 2, right) 2h after inoculation with *Pst* DC3118. At4g17980 has higher leaf surface temperature than Col-0 indicating closed stomata.

Serial dilutions and plating are used to determine the number of bacteria present in the apoplast of leaves of the Arabidopsis plant. This method only identifies living organisms in a population. A colony-forming unit (CFU) is a measure of viable bacterial numbers. The total number of bacteria was determined by counting the number of CFUs and comparing them to the dilution factor. In other words, the total amount of bacteria equals the number of colonies multiplied by the dilution factor multiplied by 2. As can be seen in Table 1, the number of bacteria was determined for each sample of the mutant and compared to the number of bacteria for Col-0. The average, standard deviation, and standard error of the six samples from Col-0 and each mutant were calculated.

Table 1. Amount of *Pst* DC3118 present in leaf samples 48 hours post-treatment. Standard deviation and standard error are listed.

Gene	Sample 1	Sample 2	Sample 3	Sample 4	Sample 5	Sample 6	Average	Standard Deviation	Standard Error
Col-0	$1.40x10^3$	$2.40x10^3$	$8.00x10^3$	$7.80x10^3$	$2.80x10^3$	$5.20x10^3$	$4.60x10^3$	$2.85x10^3$	$1.20x10^3$
At3g10575	$7.40x10^4$	$5.60x10^4$	$3.80x10^3$	$3.20x10^3$	$5.60x10^2$	$1.60x10^3$	$2.30x10^4$	$3.29x10^4$	$1.30x10^4$
At3g26510	$2.00x10^1$	$2.00x10^1$	$2.20x10^2$	$2.40x10^2$	$4.00x10^1$	$6.00x10^1$	$1.00x10^2$	$1.02x10^2$	$4.20x10^1$
At4g17980	0	0	$2.00x10^1$	0	0	$2.00x10^1$	6.70	$1.03x10^1$	4.20
At1g08510	$7.40x10^3$	$7.20x10^3$	$4.00x10^3$	$4.20x10^3$	$3.60x10^2$	$3.40x10^2$	$3.90x10^3$	$3.11x10^3$	$1.30x10^3$

According to Figure 12, At3g26510 and At4g17980 have a significantly lower number of bacteria than Col-0 forty-eight hours after inoculation. The reduced number of bacteria that entered the plant compared to Col-0 along with high leaf surface temperature in the infrared pictures taken 2h after inoculation with *Pst* DC3118 supports strong stomatal closure. It is probable that these two mutants are resistant to the bacteria.

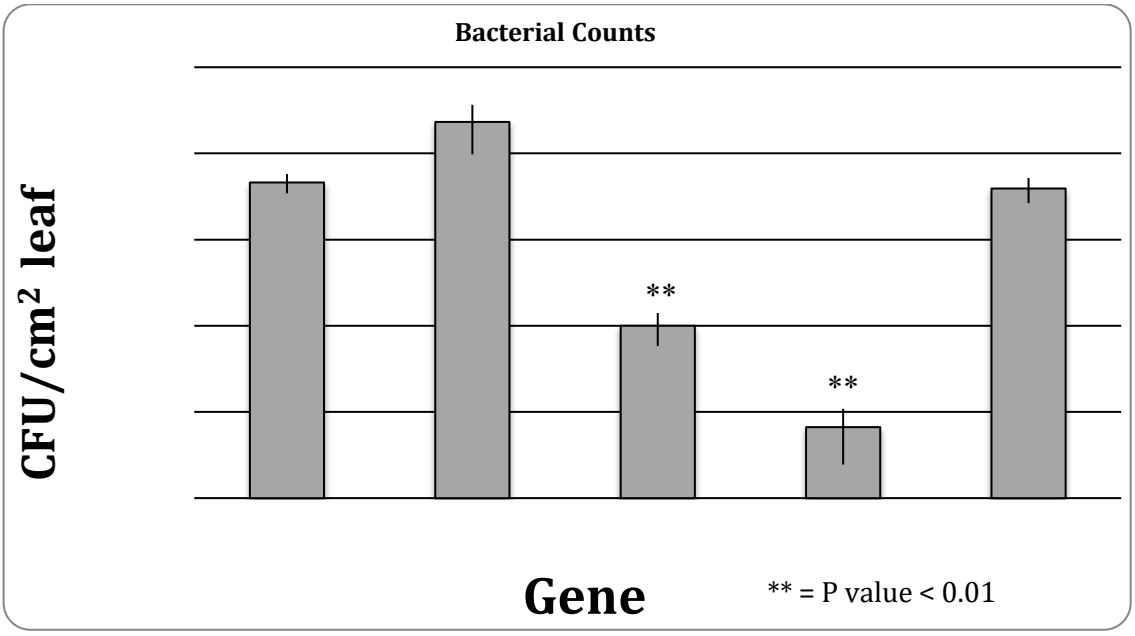

Figure 12. Bacterial counts of Col-0 and mutants 48 hours post-inoculation.

The Student's t-test was used to assess whether two groups are statistically different from each other. To test the significance, the alpha level is at 0.05. The data is unpaired because the two samples are independent. Table 2 lists the p-values of selected mutants. At3g26510 and At4g17980 are statistically different from the wild type and are possibly more resistant to *Pst* DC3118.

Table 2. P-values of amount of *Pst* DC3118 in samples of mutants compared to samples of Col-0.

Gene	P-Value
At3g10575	0.197900145
At3g26510	0.003101804
At4g17980	0.002711508
At1g08510	0.699777211

Table 3 lists possible genes contributing to stomatal immunity and their functions. At3g22845 is located in the vacuole and involved in transport. At1g73210 is located in the cellular component, has a molecular function, and is involved in biological processes. At3g01910 is located in mitochondrion and peroxisome. The gene is involved in chlorophyll metabolic process, oxidation-reduction process, response to sulfur dioxide, sulfur compound metabolic process, and also has sulfite oxidase activity. At2g06650 is a transposable element gene. At3g48480 is located in the cellular component, has cysteine-type peptidase activity, and is involved in proteolysis. At5g25320 is located in the cellular component. The gene functions in amino acid binding and is involved in metabolic process. At1g60120 is a transposable element gene. At1g75930 is located in the

extracellular region. The molecular function is carboxylesterase activity, lipase activity, transferase activity, and transferring acyl groups. The biological process the gene is involved in is metabolic process and sexual reproduction. At4g29000 has sequence-specific DNA binding transcription factor activity and is involved in regulation of transcription. Lastly, At4g17980 is located in the cellular component, has sequence-specific DNA binding transcription factor activity, and is involved in multicellular organismal development and regulation of transcription.

Table 3. Genes with possible role in stomatal immunity.

Locus	Function
At3g22845	Transport
At1g73210	Biological processes, molecular function
At3g01910	Chlorophyll metabolic process, oxidation-reduction process, response to sulfur dioxide, sulfur compound metabolic process
At2g06650	Transposable element gene, non-LTR retrotransposon family (LINE)
At3g48480	Cysteine-type peptidase activity, proteolysis
At5g25320	Amino acid binding, metabolic process
At1g60120	Transposable element gene

At1g75930	Metabolic process, sexual reproduction, carboxylesterase activity, lipase activity, transferase activity, transferring acyl groups
At4g29000	Regulation of transcription, DNA-dependent, sequence-specific DNA binding transcription factor activity
At4g17980	Sequence-specific DNA binding transcription factor activity, multicellular organismal development, regulation of transcription, DNA-dependent

CHAPTER 4

DISCUSSION

Many bacteria can survive and multiply on a plant surface without causing disease. Foliar pathogens enter the plant and multiply in the apoplast as pathogenic endophytes. Bacteria cannot directly penetrate the epidermis of the plant and must rely on natural openings to enter the plant. Stomata were initially regarded as passive ports of bacterial entry into plant leaves. However, the stomata play an active role in limiting bacterial infection. Stomatal guard cells can close the stomatal pore within 1h of exposure to bacterial suspension. Bacterium-induced stomatal closure can be activated by bacterial PAMPs and requires PAMP signaling, SA, and ABA. However, some plant pathogens have evolved specific virulence factors to overcome stomata-based defence. When treated with COR, the leaves of many plants develop chlorosis. COR promotes entry of bacteria through stomata at the initial stages of infection and suppresses defences later in the infection process (Underwood et al., 2007).

Previous studies have shown that the coronatine-defective *Pst* DC3118 bacterium is a strong inducer of stomatal immunity. We show that is it also an excellent organism for genetic screening for mutant plants with stomatal immunity. Col-0 has mostly open stomata in the mid-morning, and dipping Col-0 in *Pst* DC3118 causes the stomata to close. Mutant plants with higher leaf surface temperature than the wild type Col-0 have closed stomata after inoculation, whereas mutant plants with lower leaf surface

temperature than Col-0 have mostly open stomata. Open stomata causes the plants to be colder because of transpiration, loss of water vapor through the open stomata (Merlot et al., 2002). Thus far, 10 mutant plants with high leaf surface temperature or enhanced stomatal immunity have been identified. The mutant plants should be screened again to ensure reproducible results.

Investigating stomatal defenses of plants could greatly contribute to plant disease resistance and the field of agriculture. Severe outbreaks of bacterial disease in crops are usually associated with periods of heavy rain or high humidity that create wounds and leaf surface wetness favorable for bacterial movement. High humidity also promotes stomatal opening, allowing more bacteria to enter the leaf tissue. Mutant plants with compromised stomatal closure in response to bacteria suggest that their defective gene is involved in stomatal immunity. The defective gene of the mutant plant could then be changed to prevent the stomata from opening. As a result, plants would not be affected by common bacteria, and that may increase production of crops.

Based on the gain in knowledge of stomatal defense, disease epidemiology, pathogen lifestyles, phyllosphere ecology, and virulence factors, research could be done to determine whether environmental conditions that modulate stomatal closure and opening influence the effectiveness of stomatal defense, how failure of stomatal defense can contribute to disease outbreaks, and whether different stomatal sizes impact the effectiveness of stomatal defense. It would be interesting to know how sizes and locations of bacterial communities affect stomatal defense, whether all plants have stomatal defense mechanisms, the number of virulence factors that target stomatal defense, and

the mechanisms by which other virulence factors inactivate stomatal defense (Melotto et al., 2008).

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BIOGRAPHICAL INFORMATION

Samantha Trinh was born in Florida but now lives in Texas. She attended the University of Texas at Arlington where she earned a Bachelor of Science in Medical Technology and a minor in Chemistry. She was a member of the Honors College Council, Global Medical Training, and the National Society of Leadership and Success. She joined Dr. Melotto's lab in spring 2011 that focuses on the mechanisms by which plants defend themselves against bacterial infection and virulence strategies evolved by bacterial pathogen to overcome plant defenses. Samantha has done a poster presentation for the Texas Branch Fall Meeting of the American Society for Microbiology, an oral presentation, and the honors thesis. She plans on becoming a physician assistant in the future.