Research Article

Co-5² resistance allele contributes to induce basal defense against *Colletotrichum lindemuthianum* race 7 [1]

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ABSTRACT

The role of the *Co-5²* and *Co-4²* alleles in the early activation of bean immunity against *Colletotrichum lindemuthianum* is poorly known. Thus, by using the anthracnose-susceptible Sutagao cultivar and the anthracnose-resistant bean G2333, this study aimed to evaluate the presence of the *Co-4²* and *Co-5²* alleles by SCAR markers, as well as their role in the respective defense responses, using histochemical tests and differential gene expression. The results showed that the *Co-5²* allele contributes to defense responses against anthracnose, since susceptible beans with the *Co-4²* allele and without the *Co-5²* allele decreased both the number of callose deposits and the accumulation of reactive oxygen species at the fungus attack sites. Additionally, for the Sutagao cultivar, there was a late response of defense-related genes such as *PR-1*, *PR-3* and *Pv-POD*. The *Co-5²* allele contributes to induce defense mechanisms in the *Phaseolus vulgaris* and *C. lindemuthianum* interaction.

KEYWORDS: *Phaseolus vulgaris* L., anthracnose, callose deposits, plant defense.

RESUMO

Alelo de resistência *Co-5²* contribui para induzir a defesa basal contra *Colletotrichum lindemuthianum* raça 7

Em feijoeiro, pouco se conhece sobre o papel dos alelos *Co-5²* e *Co-4²* na ativação precoce da imunidade da planta ao fungo *Colletotrichum lindemuthianum*. Utilizando-se a cultivar Sutagao suscetível à antracnose e o feijoeiro G2333 resistente à antracnose, objetivou-se avaliar a presença dos alelos *Co-4²* e *Co-5²* por meio de marcadores SCAR, bem como sua função nas respectivas respostas de defesa, por meio de testes histoquímicos e expressão gênica diferencial. Os resultados mostraram que o alelo *Co-5²* contribui para as respostas de defesa contra a antracnose, uma vez que os feijoeiros suscetíveis com o alelo *Co-4²* e sem o alelo *Co-5²* diminuíram tanto a quantidade de depósitos de calose quanto o acúmulo de espécies reativas de oxigênio nos locais de ataque do fungo. Ademais, para a cultivar Sutagao, houve resposta tardia de genes relacionados à defesa, como *PR-1*, *PR-3* e *Pv-POD.* O alelo *Co-5²* contribui para induzir mecanismos de defesa na interação entre *Phaseolus vulgaris* e *C. lindemuthianum*.

PALAVRAS-CHAVE: *Phaseolus vulgaris* L., antracnose, depósitos de calose, defesa vegetal.

INTRODUCTION

The production of common bean (*Phaseolus vulgaris* L.) is affected by the *Colletotrichum lindemuthianum* (Sacc. and Magnus) anamorph of *Glomerella cingulata* f. sp. *phaseoli*, which is the causal agent of anthracnose (Fontenelle et al. 2017). As a management strategy, resistant cultivars carrying the anthracnose-resistant genes *Co* have been used (Garzón et al. 2007, Padder et al. 2017). These genes, which confer resistance to specific races of *C. lindemuthianum*, are clustered in the bean genome and encode 256 NBS-LRR (Nucleotide Binding Site - Leucine-Rich Repeat) and 200 RLK (Receptor Like Kinase) protein types involved in the activation of defense responses against *C. lindemuthianum* (Vaz Bisneta & Gonçalves-Vidigal 2020).

The *Co-4²* allele, a form of *Co-4* locus, and the *Co-5²* and *Co-3⁵* alleles have been mapped on the Pv08, Pv07 and Pv04 chromosomes, respectively, which contain clusters of anthracnose-resistant genes (Gilio et al. 2020, Vaz Bisneta & Gonçalves-Vidigal 2020). These alleles have been identified in the bean differential G2333, hence resistant to a large number of *C. lindemuthiaum* races (Young et al. 1998). The SAS13 molecular marker type SCAR (Sequence Characterized Amplified Region) is tightly linked to the *Co-4* locus and contains part of an

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open-reading frame (ORF) that encodes for a serine/threonine kinase (STK) (Melotto et al. 2004). SAS13 has been used to identify the *Co-4²* allele, which provides more resistance than the original *Co-4* and contains copies of the *COK-4* gene, which encodes Receptor-Like Kinases (RLKs) (Rodrigues et al. 2015). Additionally, there are few studies about the *Co-5* locus contribution to the induction of defense response in common bean. This locus contains a cluster of genes that confers specific resistance to different races, including the *C. lindemuthianum* race 7 frequently found in bean crops (Campa et al. 2009).

In the present study, a cross between the Sutagao bean cultivar, which is derived from the Cabrera bean (of Andean origin; susceptible to the pathogen), and the bean differential G2333 (of Mesoamerican origin; the *Co-4²*, *Co-5²*, *Co-3⁵* resistance alleles) was used. In previous studies, the Sutagao cultivar was exposed to different races of the pathogen (races 7, 15, 131, 133, 137 and 141) found in Colombia (Gallego et al. 2010) and were evaluated with the resistance scale proposed by Schoonhoven & Pastor-Corrales (1987), showing intermediate resistance to race 7, with a value of 4.4. However, the Sutagao cultivar has recently shown susceptibility to this race. Thus, this study focused on verifying the presence of *Co* genes inherited from the parent G2333 to the Sutagao cultivar (understanding that both materials are not isogenic lines), specifically markers of the *Co-4²* and *Co-5²* alleles previously identified in these materials (Garzón et al. 2007), but not the *Co-3⁵* allele, because there is no molecular marker associated with it, as well as their possible influence on inducing early defense against *C. lindemuthianum.*

MATERIAL AND METHODS

Seeds from the Sutagao cultivar, and their parents G2333 and Cabrera, were used in this study, in 2019. All bean seeds were sown in sterile soil and kept under greenhouse conditions at 18 ºC. *Colletotrichum lindemuthianum* race 7 was supplied by the International Center for Tropical Agriculture (CIAT, Colombia) and grown in sterile bean pods at 17 ºC, for 15 days (Castellanos et al. 2011).

To evaluate the presence of the *Co-4²* and *Co-5²* alleles, the DNA extraction was performed from leaves of 15-day-old plants (Miklas et al. 1996). One sample of each bean variety (the sample was composed of 5-10 detached leaves) was randomly selected and collected. This sampling was repeated three times. The amplification of SCAR markers was confirmed by PCR using specific primers for the SAS13 marker linked to the *Co-4²* allele (Forward: 5'-CAC GGA CCG AAT AAG CCA CCA ACA-3'; Reverse: 5'-CAC GGA CCG AGG ATA CAG TGA AAG-3') (Miklas & Kelly 2002) and for the SAB3 marker linked to the *Co-5²* allele (Forward: 5'-TGG CGC ACA CAT AAG TTC TCA CGG-3'; Reverse: 5'-TGG CGC ACA CCA TCA AAA AAG GTT-3') (Vallejo & Kelly 2001). The PCR reaction was composed of 0.1 µM of each primer, 1x PCR buffer [500 mM KCl, 100 mM Tris-HCl (pH 9.1 at 20 ºC) and 0.1 % Triton ™ X-100], 2.5 mM $MgCl_2$, 0.25 mM dNTPs, 1U Taq DNA polymerase (Vivantis Technologies Sdn Bhd) and 2 µL DNA. The amplification for each marker was carried out according to BIC (2010).

The anthracnose severity was evaluated on 15-day-old plants from the Sutagao cultivar and their parents G2333 and Cabrera. The plants were inoculated by entirely brushing both sides of the leaf with a spore suspension of *C. lindemuthianum* race 7 at 1.7 x 10^6 conidia mL^{-1} (Dufresne et al. 1998). The severity evaluation was carried out at 9 days post inoculation (DPI) on ten plants from each bean variety using a severity scale (Schoonhoven & Pastor-Corrales 1987), under greenhouse conditions of 18 ºC and relative humidity of 71 %.

In different experiments, the defense responses for hydrogen peroxide, callose deposits and gene expression were evaluated on (inoculated and non-inoculated) 15-day-old plants of the Sutagao cultivar and of the anthracnose-resistant bean G2333, using the inoculation method previously described. The detection of hydrogen peroxide (H_2O_2) was evaluated according to the protocol proposed by Thordal-Christensen et al. (1997). The leaves were randomly harvested at 6, 16 and 24 h post inoculation and at 3, 5 and 7 DPI. Two leaves per each evaluation time and variety were analyzed, and the experiment was repeated three times. Each leaf was stained with 3,3'-diaminobenzidine tetrahydrochloride (3,3'-DAB; 1 mg mL^{-1}; Sigma-Aldrich) and immersed in the solution. The 3,3'-DAB was infiltrated into leaves using a vacuum pump for 30 min. The leaves were later incubated in continuous shaking overnight at 19 ºC in the dark. The solution was removed, and the leaves were bleached overnight with absolute

ethanol in a water bath at 65 ºC. The samples were kept in 70 % ethanol at 4 ºC until observation. The hydrogen peroxide was located qualitatively in the *C. lindemuthianum* attack sites, and the plant tissue was scanned with a microscope at 40x. Photographs were taken with a ToupTek Photonics camera (UCMOS14000KPA, China) and calibrated with the ImageJ v1.51 software.

The detection of callose deposits was performed on leaves harvested at 3, 5 and 7 DPI. Two leaves per evaluation time and bean variety were randomly selected and collected. This experiment was repeated three times. Each leaf was stained with aniline blue at 0.01 % (Danies et al. 2015), visualized with an epifluorescence microscope with a UV filter (BP 360-370 nm excitation and BA 420 nm emission) at 10x, and 30 optical fields were randomly captured. The callose deposits were counted using the DotCount v1.2 software under an intensity threshold of 128, at a minimum point size of 1 and a maximum of 250 (Rodrigues et al. 2015). The variations of callose deposits between control and inoculated leaves (n = 30) at 3, 5 and 7 DPI were evaluated by a Generalized Linear Model (GLM) using the R software version 4.1.1. The assumptions for parametric statistics were verified and satisfied.

Quantitative RT-PCR was used to validate gene expression changes between the resistant G2333 and the susceptible Sutagao cultivar infected with *C. lindemuthianum* race 7. For this, the total RNA was extracted from two primary leaves of inoculated and non-inoculated plants harvested at 0, 2, 3, 5 and 7 DPI. The RNA extraction was performed following the CTAB-based protocol with lithium chloride, treated with DNase I (Thermo Scientific), and quantified by a Thermo Scientific™ NanoDrop™ OneC spectrophotometer. Then, cDNA synthesis was done with the SuperScript III First-Strand Synthesis System Kit and the Oligo (dT) primer (Invitrogen) using 1 µg RNA. qRT-PCR was performed with gene-specific primers (Table 1), and amplification was detected with SYBR Green I (Roche) performed on the Roche LightCycler® 480 System. For relative quantification, the Cp (crossing point) values of the *PvEF1-α* reference gene and the defense genes were compared considering the amplification efficiency of each gene. PCR reaction was performed in a volume of 11.4 µL composed of 5 µL of SYBR Green I, 0.2 µL of each primer (10 µM), 2 µL of cDNA and 4 µL of water. The reaction was performed under the following thermal profile: 95 ºC for 5 min, followed by 45 cycles of 95 ºC for 10 s, 61 ºC for 30 s and 72 ºC for 30 s. A melting curve was generated under the profile 95 ºC for 5 s, 65 ºC for 1 min and 97 ºC continuous. Finally, a cooling cycle was carried out at 40 ºC for 10 s. For each gene and evaluation time, two technical replicates were taken, the expression levels were determined using the $2^{-\Delta\Delta Ct}$ method (Livak & Schmittgen 2001), and the log2-transformed fold change of the defense genes was analyzed. Significant differences were identified according to the expression mean of each gene using the Tukey test ($p < 0.05$).

RESULTS AND DISCUSSION

The anthracnose-resistant G2333 and the Sutagao cultivar showed a band of 950 bp, corresponding to the allele *Co-4²* with molecular marker SAS13 (Vallejo & Kelly 2001, Miklas & Kelly 2002) (Figure 1A).

The SAS13 is tightly linked to the *Co-4* locus and contains part of an open reading frame (ORF) that encodes for a Kinase-Like Receptor (RLK)

Table 1. Primer sequences used for real-time qPCR analysis of defense-related genes of *Phaseolus vulgaris.*

Gene	Function	Oligonucleotide	Sequence (5'-3')	Reference
PvEF1-α	Elongation factor 1	*Pv-EF1α*-F	CGGGTATGCTGGTGACTTTT	Mayo et al. 2015
		Pv-EF1α-R	CACGCTTGAGATCCTTGACA	
PR-1	Antifungal	*PR1*-F	TGGTCCTAACGGAGGATCAC	Mayo et al. 2015
		PR1-R	TGGCTTTTCCAGCTTTGAGT	
PR-3	Chitinase	*PR3*-F	ATTGTTGTGCCAATCCCTTT	Oliveira et al. 2015
		PR3-R	CACCGCCATACAGTTCAAAA	
PR-4	Chitinase	*PR4*-F	CGCAGTGAGTGCATATTGCT	Mayo et al. 2015
		PR4-R	TGTTTGTCACCCTCAAGCAC	
Pv-POD	Peroxidase II	*Pv-POD*-F	TCCTTTTCAGCACTTTCACT	Oliveira et al. 2015
		Pv-POD-R	AGAAAGCAGTGTTCTTGTGG	

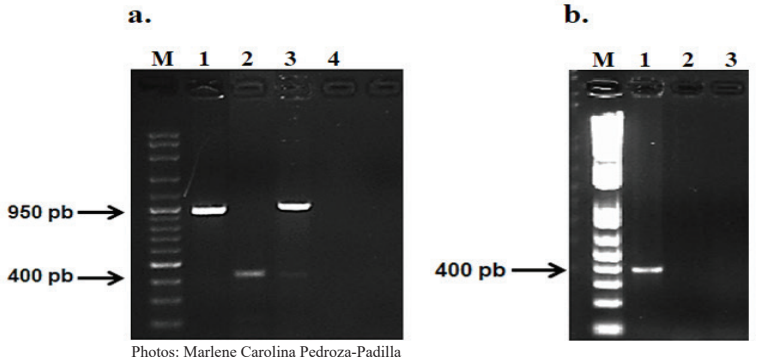

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Figure 1. SCAR molecular analysis for the *Co-4²* allele (SAS13) and the *Co-5²* allele (SAB3) by PCR in varieties of *Phaseolus vulgaris*. M: 1 kb ladder. A) SAS13 marker: lane 1 - anthracnose-resistant parent G2333; lane 2 - parent Cabrera; lane 3 - Sutagao cultivar; lane 4 - PCR negative control. B) SAB3 marker: lane 1 - anthracnose-resistant parent; lane 2 - parent Cabrera; lane 3 - Sutagao cultivar.

named COK-4 (Melotto et al. 2004), which is similar to the FERONIA protein from *Arabidopsis thaliana* and is involved in plant defense and development (Rodrigues et al. 2015, Azevedo et al. 2018). In addition, an unspecific band of 400 pb was amplified in the Cabrera parent and in the Sutagao cultivar, but it was absent in G2333 with marker SAS13 (Figure 1A). The SCAR molecular marker SAB3 associated with the *Co-5²* allele amplified a band of 400 bp in G2333, but it was absent in the Cabrera parent, and unexpectedly in the Sutagao cultivar as well (Figure 1B). The amplification that was absent with this marker in the Sutagao cultivar may be due to changes caused by recombination events in the recognized sequence by the molecular marker in the *Co-5* locus (Garzón et al. 2007). Although no genetic mapping of this region was carried out, the indirect effect of its absence was evaluated by anthracnose severity response and early defense induction against *C. lindemuthianum.*

To relate the presence of the *Co-4²* allele and the absence of *Co-5²* in the Sutagao cultivar, the anthracnose severity was compared with the parent G2333 (carrying *Co-4²* and *Co-5²*) and Cabrera (without *Co-4²* and *Co-5²*). The results indicated that G2333 was resistant to race 7 of the pathogen as the value of 1 was obtained, according to the scale of severity; while the Sutagao cultivar and its parent Cabrera were susceptible, showing values of 9 and 7, respectively, since these plants developed severe necrotic lesions (Figure 2).

Once severe anthracnose symptoms were detected in the Sutagao cultivar, the production of compounds related to plant defense responses was evaluated in both the Sutagao cultivar and the parent G2333. The H_2O_2 production was visualized by reddish brown halos around the appressoria in the pathogen attack sites after 24 h post inoculation in G2333, while, in the Sutagao cultivar, there was no accumulation of H_2O_2 by that time (Figure 3). At 3 DPI, larger halos were observed around the appressoria in G2333, as well as hypersensitivity response in the cells (Figure 3). Simultaneously, the fungus showed conidia, germ tubes and appressoria of degraded appearance in G2333, while, in the Sutagao cultivar, infection vesicles indicated the entry of the fungus into the cell (Figure 3). At 5 DPI, a pathogen invasion was evident due to the presence of primary and secondary hyphae in the Sutagao cultivar, and, at 7 DPI, the H_2O_2 accumulation increased, especially in regions with necrotic lesions, while, in the resistant parent G2333, it decreased, presenting smaller appressoria (Figure 3).

H_2O_2 is considered a second messenger that participates in both the PAMP-Triggered Immunity (PTI) and Effector-Triggered Immunity (ETI) to allow the expression of defense-related genes (Kadota et al. 2015), and for the release of Ca^{2+} to the cytosol, NADPH-oxidase activation, interaction with salicylic acid (SA) signaling pathways for generation of hypersensitivity response and the establishment of systemic defense (Kimura et al. 2017). H_2O_2 also regulates the transcription of defense genes to generate modifications in the cell wall in response to pathogens, favoring the lignin synthesis (Smirnoff & Arnaud 2019).

On the other hand, the resistant parent G2333 and the Sutagao cultivar showed callose deposits at

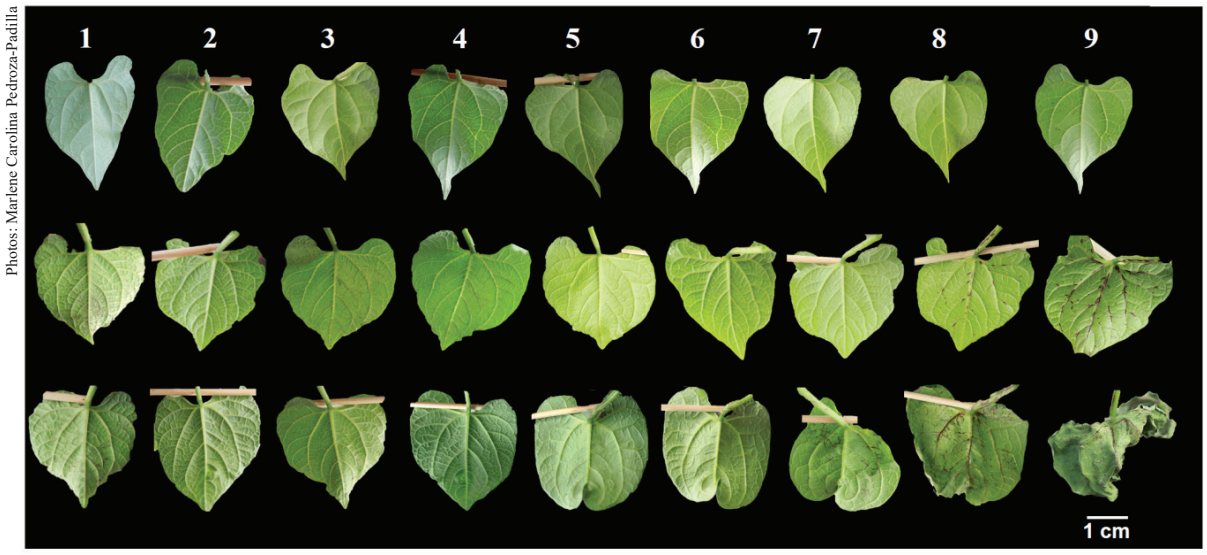


Figure 2. Anthracnose symptoms in *Phaseolus vulgaris* leaves inoculated with *Colletotrichum lindemuthianum* race 7. Top lane: leaves of anthracnose-resistant parent G2333; middle lane: leaves of susceptible parent Cabrera; bottom lane: leaves of susceptible Sutagao cultivar. Severity was evaluated at 9 days post inoculation (DPI) (Schoonhoven & Pastor-Corrales 1987). The sequence of numbers indicates the DPI.

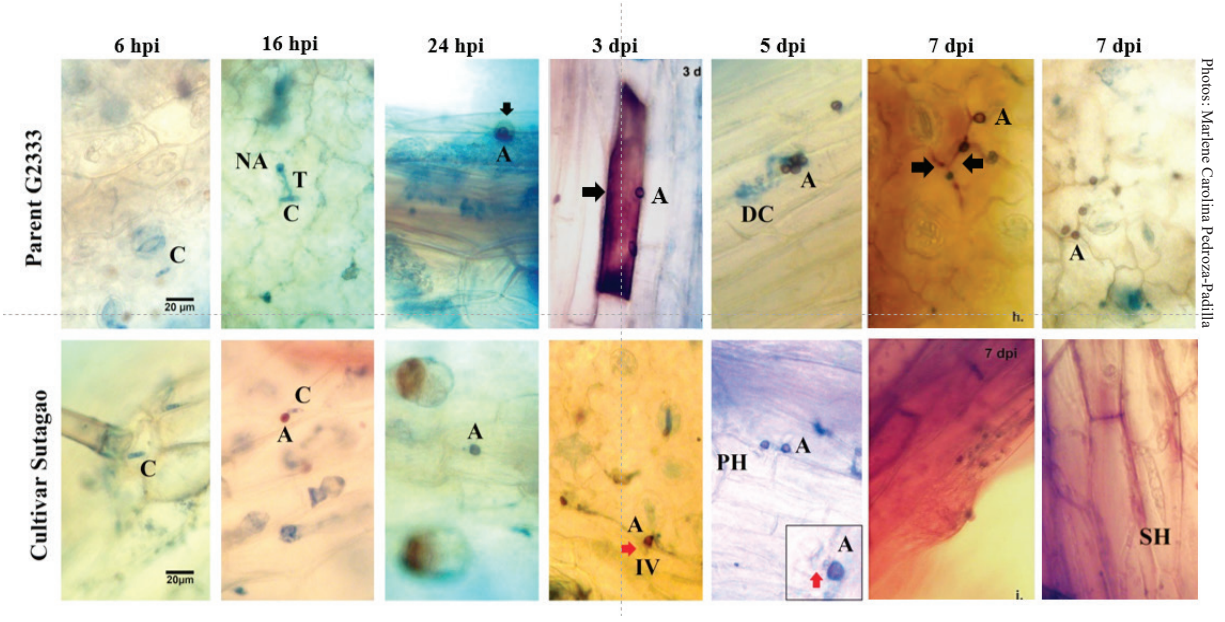
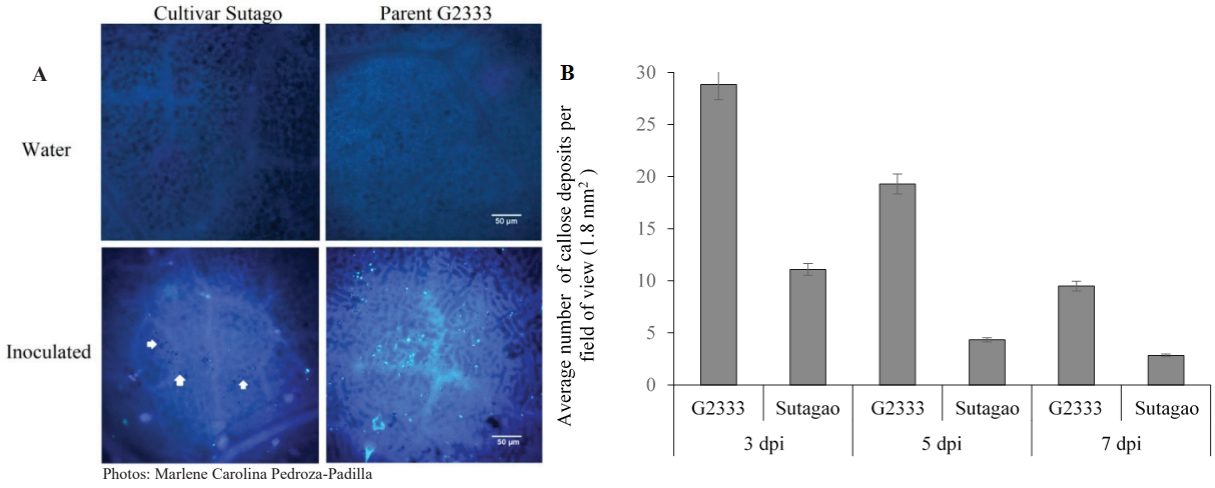


Figure 3. Accumulation of H_2O_2 during the interaction between *Phaseolus vulgaris* and *Colletotrichum lindemuthianum* race 7. Top lane: leaves of the anthracnose-resistant parent G2333; bottom lane: leaves of the susceptible Sutagao cultivar. The detection of H_2O_2 was visualized at 24 h post inoculation (HPI) in G2333 as brown to reddish brown halos at the fungus attack sites and at 3 days post inoculation (DPI) in cells with hypersensitivity responses. In the Sutagao cultivar, the detection of H_2O_2 at 6 HPI was not evident, but it was intense at 7 DPI in regions with necrosis. C: conidia; DC: degraded conidia; T: germ tube; A: appressoria; NA: non-melanized appressoria; IV: infective vesicle; PH: primary hypha; SH: secondary hypha; black arrow, hydrogen peroxide and red arrow: infective vesicle. Magnification: 40x; scale: 20 µm.

3, 5 and 7 DPI. However, G2333 exhibited a higher number of callose deposits than the Sutagao cultivar (Figure 4). Cell wall thickening (called papilla) becomes visible at the pathogenic attack site to limit the entry and growth of infectious structures (Voigt & Somerville 2009). These results indicate that, once pathogen penetration attempts were made, the G2333 was able to detect conserved PAMP-type



Photos: Marlene Carolina Pedroza-Padilla

Figure 4. Detection and counting of callose deposits in the anthracnose-resistant parent G2333 and in the susceptible Sutagao cultivar inoculated with *Colletotrichum lindemuthianum* race 7. A) Callose deposits in the leaf tissue at 3 days post inoculation (DPI). Top lane: control leaves; bottom lane: inoculated leaves. Green fluorescence dots indicate callose deposits, and black dots (white arrows) represent appressoria. Scale: 50 µm. B) Average number of callose deposits in G2333 and Sutagao. The General Linear Model (GLM) analysis revealed a significant effect of bean material ($p < 0.001$) on the number of callose deposits at each time. Bars represent the standard deviation.

molecules which early induced the accumulation of antimicrobial compounds, such as H_2O_2, and the reinforcement of the cell wall by callose, in order to block the entry of *C. lindemuthianum* (Kadota et al. 2015).

The relation between the expression of defense genes with the presence or absence of the *Co-5²* allele was evaluated by quantifying the relative expression of the *Pv-POD*, *PR-3*, *PR-1* and *PR-4* genes. The peroxidase gene (*Pv-POD*) expression was higher in the resistant parent G2333 than in the susceptible Sutagao cultivar at 1 DPI (Figure 5). However, Sutagao increased the expression of *Pv-POD* at 3 DPI, yet it was lower than that of G2333 at 1 DPI. The *PR-3* gene, which codes for chitinases, rose its expression level in G2333 at 1 DPI, increasing at 5 and 7 DPI, in contrast to the Sutagao cultivar, which showed low levels at the same times. The *PR-1* gene (antifungal activity) was highly expressed in G2333 more than in the Sutagao cultivar, with the highest expression thereof being detected at 1 and 3 DPI. The *PR-4* gene (chitinase) showed the highest expression at 5 DPI in Sutagao and at 7 DPI in G2333 (Figure 5). The peroxidase gene (*Pv-POD*) and the *PR-3* gene were expressed early at 1 DPI in G2333, coinciding with the detection times of H_2O_2. These results confirm that the defense processes were performed after the detection of the pathogen at 1 DPI, simultaneously with the expression of defense genes, accumulation of H_2O_2, chitinases and callose deposits under the appressoria. In contrast, the susceptible Sutagao cultivar, which had only the molecular marker associated with the *Co-4²* allele, showed no changes in the production of H_2O_2, callose deposits, and *Pv-POD* and *PR-3* gene expression at 1 DPI. Thus, these results, together with histological evaluations, indicate that *C. lindemuthianum* initiates the penetration at around 1 DPI for the establishment of its biotrophic hypha, formation of the infective vesicle and primary hyphae. The *C. higginsianum* pathogenic fungus manipulates the host defense from the appressorial stage (pre-penetration and penetration) to the biotrophic phase (O'Connell et al. 2012). In the Sutagao cultivar, *C. lindemuthianum* was not detected as early inducing defense responses, which favored the entry of the fungus and the development of the infective vesicle at 3 DPI, thus generating the anthracnose disease, unlike the parent G2333, which showed to be resistant to *C. lindemuthianum* race 7.

The *PR-1* gene related to antifungal activity was significantly expressed at 1 and 3 DPI in the resistant parent G2333, contributing to the defense against *C. lindemuthianum*. The expression of the *PR-1* and *PR-3* genes in the pathosystem barley and *Pseudomonas syringae* pv. *syringae* is linked to the SA hormone pathway activation, since *PR-1* is a marker gene in that pathway (Yang et al. 2015). Therefore, we consider that, in the resistant G2333,

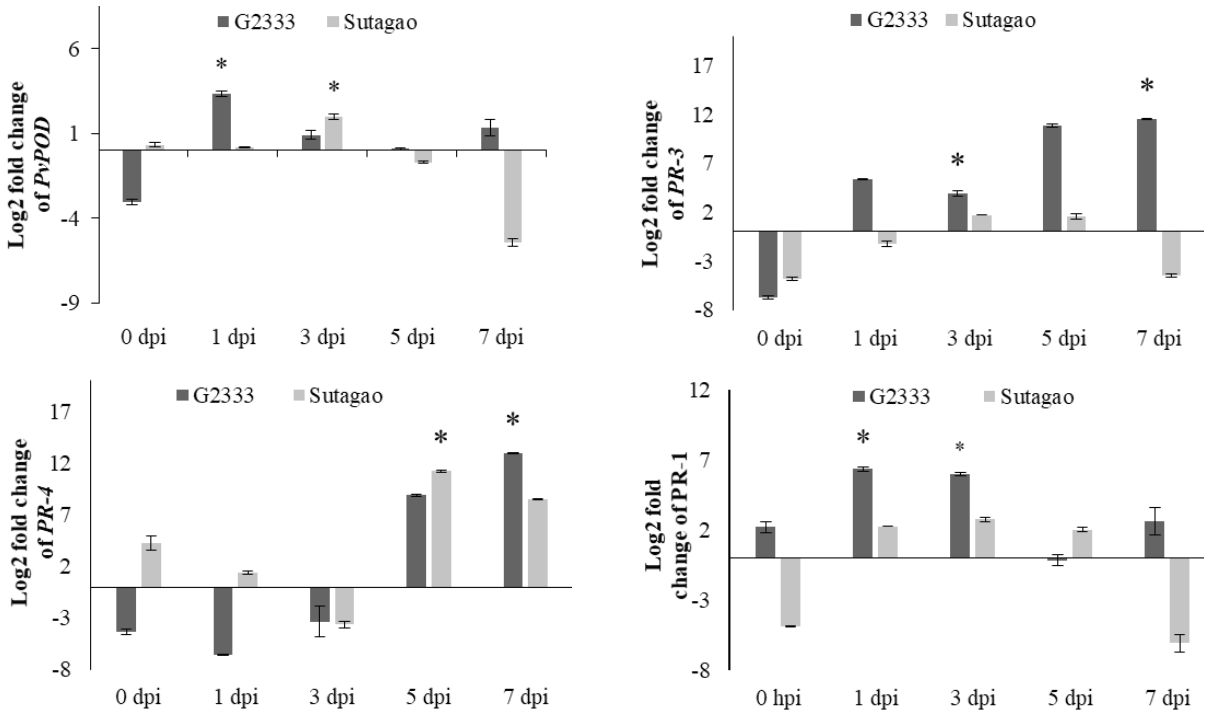


Figure 5. Log2 transformed fold change of defense genes from *Phaseolus vulgaris* in response to *Colletotrichum lindemuthianum* race 7. The expression analysis of *Pv-POD*, *PR-3*, *PR-1* and *PR-4* genes was carried out by real-time qPCR, and *PvEF1-α* was used as the reference gene. Leaves were collected at 0, 1, 3, 5 and 7 days post inoculation (DPI). The error bars represent the SD of n = 3. Asterisks indicate significant differences ($p < 0.05$).

the SA-mediated defense signaling pathway was induced early.

PR-4 is a marker gene of the jasmonic acid (JA), signaling a pathway which codes for chitinase. It is induced by abscisic acid (ABA), methyl jasmonate, and is repressed by SA, ethylene or H_2O_2 (Wang et al. 2011). In G2333, *PR-4* was highly induced at 7 DPI, indicating the activation of the JA hormone pathway, which contrasted with the *PR-1* expression at 3 DPI, showing antagonism between the hormone pathways SA and JA, since the induction of *PR-1* and *PR-4* occurred at different times during the interaction between *P. vulgaris* and *C. lindemuthianum.* Thus, the expression of *PR-4* in the resistant parent G2333 would be associated with a strengthening defense against the presence of the fungus (Ali et al. 2018). The induction of *PR-4* in the Sutagao cultivar at 5 DPI may be interpreted as a late activation of defense mechanisms not successful enough to limit pathogenic invasion. Finally, these results showed that the formation of chemical and physical barriers at infection sites, as well as the induction of some defense genes such as *Pv-POD*, *PR-3*, *PR-1* and *PR-4*, may be associated with the presence of the *Co-5²* allele. However, considering that the evaluated bean varieties are not isogenic lines, the contribution of other genes in the resistance to *C. lindemuthianum* race 7 is not excluded.

The absence of the *Co-5²* allele in the Sutagao cultivar may have contributed to its susceptibility to this race, contrary to the resistant parent G2333 carrying *Co-5²*, which includes a cluster of genes able to confer specific resistance to various races of *C. lindemuthianum*, among them race 7 (Campa et al. 2009). The presence of the *Co-4²* allele in the Sutagao cultivar did not guarantee resistance to the *C. lindemuthianum* race 7. This allele has 18 copies of the *COK-4* gene, which may be a PTI defense-response activator (Rodrigues et al. 2015). However, *COK-4* enhances the plant development and is a possible negative regulator of immunity in bean plants (Azevedo et al. 2018); thus, further studies are necessary to elucidate its role during the plant-pathogen interaction.

CONCLUSION

The *Co-5²* resistance allele in *Phaseolus vulgaris* contributes to induce a basal defense response against the *Colletotrichum lindemuthianum* race 7.

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