



Screening of Resistance Genes and Some Reactive Oxygen Specific Enzymes against *Xanthomonas axonopodis* Pv. *Phaseoli* and *Pseudomonas savastanoi* Pv. *Phaseolicola* in Bean Varieties

Tibebu Belete¹, Badel Uysal Sahin², Kubilay Kurtulus Bastas³

10.18805/ag.DF-682

ABSTRACT

Background: Common bacterial blight (CBB) and halo blight (HB) are among the most destructive bacterial diseases affecting common beans, leading to significant yield and quality losses. The use of resistant cultivars remains the most effective and sustainable management strategy. Identifying the presence of resistance genes and understanding the role of antioxidant enzymes before and after bacterial inoculation are crucial for developing resistant breeding lines.

Methods: This study investigated the presence of resistance genes against *Xanthomonas axonopodis* pv. *phaseoli* (Xap) and *Pseudomonas savastanoi* pv. *phaseolicola* (Psp) in eight bean varieties (Ozmen, Noyanbey, Akman, Alberto, XAN159, Vax1, Aras 98 and 36K). Additionally, the activities of antioxidant enzymes; Peroxidase (POX) and Catalase (CAT) were evaluated in two selected varieties (Aras 98 and 36K) following bacterial inoculation. Resistance genes (SAP6, BAC6, BC420 and R7313 for Xap; SR13, ST8, SH11 and SB10 for Psp) were screened using SCAR markers under greenhouse conditions. Four-week-old bean seedlings were inoculated with Xap and Psp isolates and leaf samples (1 g) were collected at 0, 12, 24, 48 and 72 hours post-inoculation for enzyme assays.

Result: Under greenhouse conditions, the lowest disease incidence and severity for CBB and HB were observed in XAN159, Vax1, Ozmen, Noyanbey and 36K ($p \leq 0.01$). The SAP6 and BAC6 resistance genes were present in all cultivars except Alberto and Aras 98, respectively, while the SR13 gene was absent in Noyanbey and Aras 98. POX activity increased by 198.40% in the 36K cultivar at 36 hours post-inoculation, whereas CAT activity reached its peak (0.4087 U/g FW) in the same cultivar at 72 hours after Psp inoculation ($p \leq 0.01$). The enhanced activities of antioxidant enzymes, along with the presence of specific resistance genes, appear to play a critical role in the defense mechanisms of resistant and susceptible bean genotypes against bacterial infection.

Key words: Bean, Enzyme, *Pseudomonas savastanoi* Pv. *Phaseolicola*, SCAR, *Xanthomonas axonopodis* Pv. *Phaseoli*.

INTRODUCTION

Common bean (*Phaseolus vulgaris* L.) is an important legume in the world as a source of protein, dietary fibre and minerals such as potassium, thiamine, vitamin B6 and folic acid in diets (Garden-Robinson and McNeal, 2013; Chekanai *et al.*, 2018; Belete *et al.*, 2022). Also, the presence of phytochemicals such as polyphenolic compounds in common bean prevents various human diseases and they show high antioxidant activity (Hayat *et al.*, 2014). The production of beans (green and dried) worldwide in 2017 was about 56 million tons and they were harvested on 38 million hectares (Anonymous, 2019). In Turkey, the sowing area and production of beans (green and dried) are 1,382,613 decares and 869,347 tons, respectively (Anonymous, 2019).

Bacterial diseases that cause yield and quality losses in bean production lead to severe epidemics in warm and humid conditions and yield losses of up to 45% depending on the durability of bean variety and environmental conditions (Singh and Schwartz, 2010; Félix-Gastélum *et al.*, 2016; Belete *et al.*, 2022).

The damage by halo blight (HB) caused by *Pseudomonas savastanoi* pv. *phaseolicola* (Psp) and common bacterial

¹Rwanda Institute for Conservation Agriculture (RICA), Bugesera Campus, Bugesera Rwanda, East Africa.

²Department of Plant Protection, Faculty of Agriculture, Igdir University, Campus Igdir, Turkey.

³Department of Plant Protection, Faculty of Agriculture, Selcuk University, Konya Turkey.

Corresponding Author: Tibebu Belete, Rwanda Institute for Conservation Agriculture (RICA), Bugesera Campus, Bugesera Rwanda, East Africa. Email: tibebelete@gmail.com

How to cite this article: Belete, T., Sahin, B.U. and Bastas, K.K. (2025). Screening of Resistance Genes and Some Reactive Oxygen Specific Enzymes against *Xanthomonas axonopodis* Pv. *Phaseoli* and *Pseudomonas savastanoi* Pv. *Phaseolicola* in Bean Varieties. *Agricultural Science Digest*. 1-7. doi: 10.18805/ag.DF-682.

Submitted: 07-10-2024 **Accepted:** 18-12-2025 **Online:** 08-01-2026

blight (CBB) caused by *Xanthomonas axonopodis* pv. *phaseoli* (Xap) in the bean plants is of great economic importance worldwide due to the significant losses it generates in the crop (Hall, 1994; Singh *et al.*, 1995; Belete

et al., 2022). HB and CBB are difficult to control and the effectiveness of existing strategies for the treatment of existing diseases is limited due to the absence of an effective chemical. This is because both HB and CBB are caused by bacterial pathogens for which no effective bactericides are currently available. The pathogens can survive in crop residues, seeds and volunteer plants, enabling them to persist from season to season and they spread rapidly under favorable environmental conditions. Consequently, chemical control options are largely ineffective, making integrated approaches such as genetic resistance, crop rotation and the use of disease-free seed more critical. Genetic resistance and the use of disease-free seed provide the most effective and viable control of these two diseases (Miklas *et al.*, 2014; Ferreira *et al.*, 2003). Plant resistance is highly effective in controlling crop loss from bacterial pathogen infection. Plants have evolved varied defense mechanisms to protect themselves against pathogen attacks (Waller *et al.*, 2005).

Antioxidant enzymes such as peroxidase (POX) and catalase (CAT) play an important role in the plant response mechanism to pathogen invasion by detoxifying superoxide and H₂O₂ (Bernal-Vicente *et al.*, 2015).

In this study, the focus was on the antioxidant activities of POX and CAT enzymes in eight bean varieties and the resistance genes in these varieties for *Xanthomonas axonopodis* pv. *phaseoli* (Xap) and *Pseudomonas savastanoi* pv. *phaseolicola* (Psp) were scanned using SCAR (sequence characterized amplified region) markers. SCAR markers are PCR-based DNA markers developed from specific genomic sequences, typically derived from RAPD or other dominant markers, that are converted into longer, specific primers flanking a known DNA sequence. They are highly specific, reproducible and co-dominant, enabling reliable detection of target genes or traits, such as disease resistance (Shaik *et al.*, 2020).

MATERIALS AND METHODS

Materials

Bean cultivars were procured from Dept. of Field Crop, Selcuk University (Ozmen, Noyanbey, Akman, Alberto) and Ataturk University (36K, resistant) and Eastern Anatolia Agricultural Research Institute in Turkey (Aras 98, susceptible) and International Centre for Tropical Agriculture (CIAT) (XAN159, Vax1). Bacterial strains, *Xanthomonas axonopodis* pv. *phaseoli* (Xap (120-x, 145-x)) and *Pseudomonas syringae* pv. *phaseolicola* (Psp (510-p, 522-p)) cultures were provided by Dr. M. F. Donmez, Dept. of Plant Protection, Iğdır University.

Methods

Enzyme assays

All bean cultivar seed samples used in the experiment were surface sterilized with 3% (v/v) sodium hypochlorite solution for 3 min and washed with distilled water. Plants were grown in 10 cm pots in a soil mix containing sand,

perlite and peat compost under greenhouse conditions. Greenhouse-grown 36K and Aras 98 bean seedlings were used to determine POX and CAT enzyme activities.

Only these two genotypes were selected for enzymatic analysis because they represent the contrasting extremes of disease reaction-36K being highly resistant and Aras 98 highly susceptible to both Xap and Psp. This contrast allows a clearer assessment of biochemical defense responses while keeping the experimental design manageable and statistically robust. The remaining six genotypes were included only in the SCAR marker analysis to determine the presence or absence of specific resistance genes.

Two-day-old cultures of Xap and Psp were harvested from NA plates, suspended in sterile deionized water and adjusted to a concentration of 10x CFU mL⁻¹ determined by a spectrophotometer at 660 nm and OD: 0.15.

Plants of the 36K bean genotype and Aras 98 bean variety were sampled at 0, 12th, 24th, 48th and 72nd hours post inoculation (hpi) and without pathogen inoculation. The samples were stored at -80°C prior to the enzyme assay (Teranishi *et al.*, 1974).

Fresh leaf material (1.0 g) from bacterial pathogen-treated and control bean plants was homogenized with a mortar and pestle in 6 mL of ice-cold 50 mM sodium potassium phosphate buffer, pH 7. After the homogenate was centrifuged at 10,000 g for 25 min, the supernatant (crude extract) was used as the source of POX and CAT enzymes. All steps were carried out at 0-4°C (Jebara *et al.*, 2005).

Peroxidase (POX; EC: 1.11.1.7) assay

The activity of peroxidase was determined by adding 100 µL of the crude enzyme preparation to 2 mL of a solution containing 10 mmol/L KH₂PO₄ (pH 7), 20 mmol/L guaiacol and 40 mmol/L H₂O₂. The change in absorbance at 470 nm was recorded for 1 min with a spectrophotometer (Lin and Kao, 1999).

Catalase (CAT; EC: 1.11.1.6) assay

The activity of catalase was determined by adding 100 µL of the enzyme extract to 900 µL of a solution containing 50 mM potassium phosphate buffer and 30 mM H₂O₂. The change in absorbance at 240 nm was recorded for 1 min with a spectrophotometer (Aebi, 1984).

Determination of resistance genes by SCAR markers in bean varieties

Plant material

Ozmen, Noyanbey, Akman, Alberto, XAN159, Vax1, Aras 98 and 36K bean seeds were planted into pots (diameter 15 cm) filled with a mixture of sterilized soil, sand and farmyard manure and grown at 25-35°C in the greenhouse.

DNA isolation

Total genomic DNA extractions were prepared from fresh bean leaves. 200-300 mg powdered plant materials for each bean varieties was transferred to a 2 mL eppendorf

tubes and 1 mL freshly prepared extraction buffer was added (Doyle and Doyle, 1990).

PCR amplifications

PCR amplification for SCAR marker primers (Table 1) from genomic DNA was performed in a total reaction volume of 20 µL containing 4 µL (5 ng/µL) of template bean DNA, 2 µL of 10X Taq polymerase reaction buffer, 7 µL of dH₂O, 2 µL of MgCl₂, 0.5 µL of each dNTP (dATP, dCTP, dGTP and dTTP), 0.2 µM primers and 0.5 µL of Taq DNA polymerase (Applied Biological Materials, ABM, Canada).

Statistical analysis

Enzyme activity data (POX and CAT) were subjected to statistical analysis to determine significant differences

between treatments, sampling times and bean cultivars. The experiments were arranged in a completely randomized design (CRD) with three biological replicates per treatment and time point. Data were analyzed using analysis of variance (ANOVA) and means were separated using Tukey's Honest Significant Difference (HSD) test at a significance level of $p \leq 0.05$.

RESULTS AND DISCUSSION

Enzyme assays

Pseudomonas syringae pv. *phaseolicola* (Psp) and *Xanthomonas axonopodis* pv. *phaseoli* (Xap) are the causal agents of halo blight (HB) and common bacterial blight (CBB) of beans, respectively and cause substantial yield losses in

Table 1: The SCAR markers used in PCR amplifications.

SCAR Markers	Size (bp)	Primers	Amplification Protocol	References
SR13/Psp	1150bp	5'GGACGACAAGGAACATATTCA 3' 5'GGACGACAAGGCTGCAAGAACCAT 3'	95°C 5 min for 1 cycle, 30 cycles of 60s at 94°C, 60s at 60°C and 60s at 72°C; followed by one cycle of 7 minutes at 72°C	Miklas <i>et al.</i> , 2000
ST8/Psp	1350bp	5'AACGGCGACATCAGTGTAAGG 3' 5'AACGGCGACAACCGACCATGTTTTAC 3'	95°C 5 min for 1 cycle, 30 cycles of 10 s at 94°C, 40s at 65°C and 120 s at 72°C; followed by one cycle of 7 minutes at 72°C	Miklas <i>et al.</i> , 2009
SH11/Psp	800bp	5'CTTCCGCAGTCGAGAGAT 3' 5'CTTCCGCAGTAGCAC C 3'	95°C 5 min for 1 cycle, 30 cycles of 10s at 94°C, 40s at 67°C and 120s at 72°C; followed by one cycle of 7 minutes at 72°C	Miklas <i>et al.</i> , 2009
SB10/Psp	525bp	5'CTGCTGGGACAATCACCAAGTC 3 '5'CTGCTGGGACTCTCTTAC3'	94°C 5 min for 1 cycle, 30 cycles of 10s at 94°C, 40s at 65°C and 120s at 72°C; followed by one cycle of 5 minutes at 72°C	Fourie <i>et al.</i> , 2004
SAP6/Xap	820bp	5'GTCACGTCTCCTTAATAGTA 3' 5'GTCACGTCTCAATAGGCAAA 3'	34 cycles of 10s at 94°C, 40s at 55 °C and 120s at 72°C; followed by one cycle of 5 minutes at 72°C	Miklas <i>et al.</i> , 200
BAC6/Xap	1250bp	5'TAGGCGGCGGCGCACGTTTTG 3' 5'TAGGCGGCGGAAGTGGCGGTG 3'	34 cycles of 10s at 94°C, 60s at 70°C and 120s at 72°C; followed by one cycle of 5 minutes at 72°C	Jung <i>et al.</i> , 1999
BC420/Xap	900 bp	5'GCAGGGTTCGAAGACACACTGG 3' 5'GCAGGGTTGCCCCAATAACG 3'	35 cycles of 30s at 94°C, 30s at 50°C and 60s at 72°C; followed by one cycle of 5 minutes at 72°C	Yu <i>et al.</i> , 2000
R7313/Xap	700 bp	5'ATTGTTATCGTCGACACG 3' 5'AATATTTCTGATCACACGAG 3'	34 cycles of 10s at 94 °C, 40s at 60°C and 120s at 72°C; followed by one cycle of 5 minutes at 72°C	Beattie <i>et al.</i> , 1998

Turkey (Kahveci and Maden, 1994). Since no absolute effective chemical control method exists for these bacterial diseases, breeding and deployment of resistant cultivars remains the most sustainable control approach. This study aimed to investigate the activity of two key antioxidant defense enzymes; catalase (CAT) and peroxidase (POX) in resistant and susceptible bean cultivars following inoculation with Psp and Xap.

The results revealed that POX activity was induced in both resistant (36K) and susceptible (Aras 98) cultivars after inoculation with either pathogen, but the intensity and timing of induction differed significantly. In 36K, POX activity increased sharply, peaking at 36 hours post-inoculation (hpi) with a 198.40% rise, suggesting a rapid oxidative stress response. In contrast, Aras 98 showed a slower induction, with maximum POX activity at 72 hpi (Fig 1). This temporal difference suggests that early and stronger POX induction may be a key feature of resistance, as rapid reinforcement of cell walls through lignification and the production of antimicrobial quinones (via POX activity) can restrict pathogen spread (Brisson *et al.*, 1994; Kawano, 2003).

Similarly, CAT activity patterns differed between cultivars and pathogens. The highest CAT activity ($0.4087 \text{ U g}^{-1} \text{ FW}$) was observed in 36K at 72 hpi with Psp. CAT activity in 36K was consistently higher than in control plants at 24 and 36 hpi with Xap, whereas Aras 98 showed generally reduced CAT activity compared to controls (Fig 2). Since CAT scavenges hydrogen peroxide (H_2O_2) to prevent oxidative damage to host cells (Mhamdi *et al.*, 2010), its elevated activity in 36K may reflect an efficient balance between reactive oxygen species (ROS) generation for defense signaling and ROS detoxification to avoid self-harm. Reduced CAT activity in Aras 98 could lead to oxidative stress-related cellular injury, increasing susceptibility.

Previous studies support these observations: Milosevic and Slusarenko (1996) and Chandrashekar and Umesha

(2012) reported that resistant tomato genotypes showed earlier and stronger CAT activity following bacterial infection, enabling better ROS regulation. Baysal *et al.* (2003) and Kavitha and Umesha (2008) also demonstrated higher POX activity in resistant tomatoes than in susceptible ones after pathogen challenge. Taken together, our data and previous reports suggest that the resistance mechanism in 36K involves a coordinated oxidative burst-rapid POX induction for pathogen suppression, followed by elevated CAT activity to mitigate oxidative damage. The catalase (CAT) enzyme is found in peroxisomes of almost all aerobic cells.

CAT is a protective antioxidant enzyme that prevents damage to cells. Catalase enzyme hydrogen peroxide decomposes into water and molecular oxygen (Nicholls *et al.*, 2000; Cavalcanti *et al.*, 2006). Peroxidase enzyme is involved in cell wall lignification process using hydrogen peroxide which is formed during oxidative combustion and also oxidizes phenols to quinones, which are toxic to the pathogen (Brisson *et al.*, 1994; Kawano, 2003).

Thus, our results agree with Chandrashekar and Umesha (2012), who observed an earlier increase in CAT enzyme activity in resistant tomato plants than in susceptible plants after pathogen inoculation. Similarly, Baysal *et al.*, (2003) and Kavitha and Umesha, (2008) were examined POX activity in resistant and susceptible tomato variety after bacterial pathogen inoculation. They found that POX activity increased higher in resistant variety than susceptible after bacterial pathogen inoculation.

SCAR analysis

Before investigating the resistant genes using SCAR markers, the bean varieties were tested under greenhouse conditions for their susceptibility reaction to the pathogens. According to the results, highly significant differences ($p \leq 0.01$) were observed among common bean cultivars in

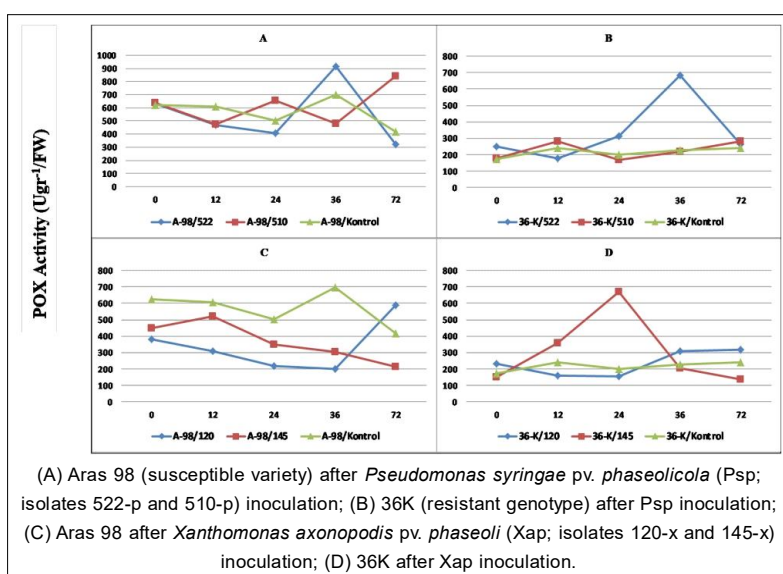


Fig 1: Peroxidase (POX) enzyme activity in common bean varieties following bacterial inoculation.

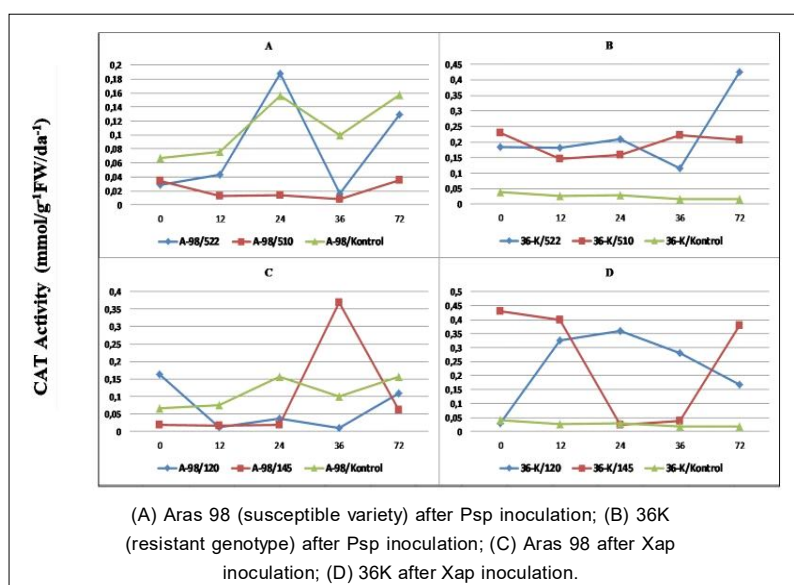


Fig 2: Catalase (CAT) enzyme activity in common bean varieties following bacterial inoculation.

terms of disease incidence and severity of Xap and Psp agents under greenhouse conditions. The lowest disease incidence and severity for both pathogens were observed in the varieties XAN159, Vax1, Ozmen, Noyanbey and 36K.

After testing the eight bean cultivars pathologically against Xap and Psp in the greenhouse, the presence of resistance genes was detected in most of the bean varieties. With the SCAR markers, an 820 bp band was obtained for SAP6 in all cultivars except Alberto, a 1250 bp band for BAC6 in all cultivars except Aras 98 and an 1150 bp band for the SR13 marker in all cultivars except Noyanbey and Aras 98 with PCR amplification. We did not obtain any results for resistance genes using the BC420, R7313, ST8, SH11 and SB10 markers in the examined bean cultivars. Similarly, Poyraz *et al.* (2017) screened ten resistance genes against Xap and Psp in 12 local bean cultivars using molecular markers (SCAR). They found a presence/absence of the tested resistance genes in bean varieties against Xap and Psp. Molecular markers are very important for the detection of resistance against plant diseases because, nowadays, plant breeding programs, including disease resistance, are incorporating molecular marker techniques like SCAR (Meziadi *et al.*, 2016).

Our studies indicated that the defense enzymes POX and CAT are actively involved in imparting resistance to halo blight caused by *P. s. pv. phaseolicola* and common bacterial blight (CBB) caused by *X. a. pv. phaseoli* in beans.

The development of resistant bean cultivars to halo and common blight diseases caused by bacterial pathogens will provide high added value to the economy of the country, reduce chemical input for farmers and have great benefits for the environment and human health.

CONCLUSION

This study showed that resistance to common bacterial blight (*Xanthomonas axonopodis* pv. *Phaseoli*) and halo blight

(*Pseudomonas savastanoi* pv. *phaseolicola*) in beans is governed by a combination of genetic and biochemical defense mechanisms. Resistant varieties, particularly 36K, exhibited earlier and stronger induction of POX and CAT activities following pathogen inoculation, indicating a more efficient oxidative defense response. In contrast, delayed and weaker enzyme responses were associated with susceptibility. SCAR marker analysis confirmed the distribution of major resistance genes among bean cultivars and generally corresponded with observed disease reactions. Therefore, integrating molecular markers with antioxidant enzyme analysis can strengthen breeding strategies aimed at developing durable bacterial disease-resistant bean cultivars.

Conflict of interest

The authors declare they have no conflict of interests.

REFERENCES

- Aebi, H. (1984). Catalase *in vitro*. *Methods in Enzymology*. **105**: 121-126.
- Anonymous (2019). FAO-Faostat Statistics Division. <http://www.fao.org/faostat/en/#data>. Accessed 24 September 2019.
- Baysal, Ö., Soylu, E.M. and Soylu, S. (2003). Induction of defence-related enzymes and resistance by the plant activator acibenzolar-S-methyl in tomato seedlings against bacterial canker caused by *Clavibacter michiganensis* ssp. *michiganensis*. *Plant Pathology*. **52**(6): 747-753.
- Beattie, A., Michaels, T.E. and Pauls, K.P. (1998). An efficient, reliable method to screen for common bacterial blight (CBB) resistance in *Phaseolus vulgaris* L. *Bean Improvement Cooperative*. **41**: 53-54.
- Belete, T., Bastas, K.K. and Ceyhan, E. (2022). Evaluation of dry bean cultivars in Turkey for resistance against common bacterial blight disease. *Legume Research*. **45**(6): 769-774. doi: 10.18805/LRF-674.

- Bernal-Vicente, A., Pascual, J.A., Tittarelli, F., Hernández, J.A. and Diaz-Vivancos, P. (2015). *Trichoderma harzianum* T-78 supplementation of compost stimulates the antioxidant defence system in melon plants. *Journal of the Science of Food and Agriculture*. **95(11)**: 2208-2214.
- Brisson, L.F., Tenhaken, R. and Lamb, C. (1994). Function of oxidative cross-linking of cell wall structural proteins in plant disease resistance. *The Plant Cell*. **6**: 1703-1712.
- Cavalcanti, F.R., Resende, M.L.V., Lima, J.P.M.S., Silveira, J.A.G. and Oliveira, J.T.A. (2006). Activities of antioxidant enzymes and photosynthetic responses in tomato pre-treated by plant activators and inoculated by *Xanthomonas vesicatoria*. *Physiological and Molecular Plant Pathology*. **68(4-6)**: 198-208.
- Chandrashekar, S. and Umesha, S. (2012). Induction of antioxidant enzymes associated with bacterial spot pathogenesis in tomato. *International Journal of Food, Agriculture and Veterinary Sciences*. **2**: 22-34.
- Chekanai, V., Chikowo, R. and Vanlauwe, B. (2018). Response of common bean (*Phaseolus vulgaris* L.) to nitrogen, phosphorus and rhizobia inoculation across variable soils in Zimbabwe. *Agriculture, Ecosystems and Environment*. **266**: 167-173.
- De Freitas, M.B. and Stadnik, M.J. (2012). Race-specific and ulvan-induced defense responses in bean (*Phaseolus vulgaris*) against *Colletotrichum lindemuthianum*. *Physiological and Molecular Plant Pathology*. **78**: 8-13.
- Doyle, J.J. and Doyle, J.L. (1990). Isolation of plant DNA from fresh tissue. *Focus*. **12(13)**: 39-40.
- Félix-Gastélum, R., Maldonado-Mendoza, I.E., Navarrete-Maya, R., Olivas-Peraza, N.G., Brito-Vega, H. and Acosta-Gallegos, J.A. (2016). Identification of *Pseudomonas syringae* pv. *phaseolicola* as the causal agent of halo blight in yellow beans in northern Sinaloa, Mexico. *Phytoparasitica*. **44**: 369-378. <https://doi.org/10.1007/s12600-016-0530-5>.
- Ferreira, C.F., Pereira, M.G., Dos Santos, A.D.S., Rodrigues, R., Bressan-Smith, R.E., Pio-Viana, A. and Daher, R.F. (2003). Resistance to common bacterial blight in *Phaseolus vulgaris* L. recombinant inbred lines under natural infection of *Xanthomonas axonopodis* pv. *phaseoli*. *Euphytica*. **134(1)**: 43-46.
- Fourie, D., Miklas, P. and Ariyaranthe, H. (2004). Genes conditioning halo blight resistance to races 1, 7 and 9 occur in a tight cluster. *Annual Report - Bean Improvement Cooperative*. **47**: 103-104.
- Garden-Robinson, J. and McNeal, K. (2013). All about beans: Nutrition, health benefits, preparation and use in menus. NDSU Extension Service, North Dakota State University.
- Hall, R. (1994). Compendium of Bean Diseases. APS Press, The American Phytopathological Society.
- Hayat, I., Ahmad, A., Masud, T., Ahmed, A. and Bashir, S. (2014). Nutritional and health perspectives of beans (*Phaseolus vulgaris* L.): An overview. *Critical Reviews in Food Science and Nutrition*. **54(5)**: 580-592.
- Jebara, S., Jebara, M., Limam, F. and Aouani, M.E. (2005). Changes in ascorbate peroxidase, catalase, guaiacol peroxidase and superoxide dismutase activities in common bean (*Phaseolus vulgaris*) nodules under salt stress. *Journal of Plant Physiology*. **162(8)**: 929-936.
- Jung, G., Skroch, P.W., Nienhuis, J., Coyne, D.P., Arnaud-Santana, E., Ariyaratne, H.M. and Marita, J.M. (1999). Confirmation of QTL associated with common bacterial blight resistance in four different genetic backgrounds in common bean. *Crop Science*. **39(5)**: 1448-1455.
- Kahveci, E. and Maden, S. (1994). Detection of *Xanthomonas campestris* pv. *phaseoli* and *Pseudomonas syringae* pv. *phaseolicola* by bacteriophages. *Journal of Turkish Phytopathology*. **23**: 79-85.
- Kawano, T. (2003). Roles of the reactive oxygen species-generating peroxidase reactions in plant defense and growth induction. *Plant Cell Reports*. **21(9)**: 829-837.
- Kavitha, R.S. and Umesha, S. (2008). Regulation of defense-related enzymes associated with bacterial spot resistance in tomato. *Phytoparasitica*. **36(2)**: 144.
- Lin, C.C. and Kao, C.H. (1999). NaCl-induced changes in ionically bound peroxidase activity in roots of rice seedlings. *Plant and Soil*. **216(1-2)**: 147.
- Meziadi, C., Richard, M.M.S., Derquennes, A., Thareau, A., Blanchet, S., Grati, A., Pflieger, S. and Geffroy, V. (2016). Development of molecular markers linked to disease resistance genes in common bean based on whole genome sequence. *Plant Science*. **242**: 351-357.
- Mhamdi, A., Queval, G., Chaouch, S., Vanderauwera, S., Van Breusegem, F. and Noctor, G. (2010). Catalase function in plants: A focus on *Arabidopsis* mutants as stress-mimic models. *Journal of Experimental Botany*. **61(15)**: 4197-4220.
- Miklas, P.N., Delorme, R., Stone, V., Daly, M.J., Stavely, J.R., Steadman, J.R. and Beaver, J.R. (2000). Bacterial, fungal and viral disease resistance loci mapped in a recombinant inbred common bean population ('Dorado'/XAN 176). *Journal of the American Society for Horticultural Science*. **125(4)**: 476-481.
- Miklas, P.N., Fourie, D., Wagner, J., Larsen, R.C. and Mienie, C. (2009). Tagging and mapping *Pse-1* gene for resistance to halo blight in common bean differential cultivar UI-3. *Crop Science*. **49(1)**: 41-48.
- Miklas, P.N., Fourie, D., Trapp, J., Davis, J. and Myers, J.R. (2014). New loci including *Pse-6* conferring resistance to halo bacterial blight on chromosome Pv04 in common bean. *Crop Science*. **54**: 2099-2108.
- Milosevic, N. and Slusarenko, A.J. (1996). Active oxygen metabolism and lignification in the hypersensitive response in bean. *Physiological and Molecular Plant Pathology*. **49(3)**: 143-158.
- Nicholls, P., Fita, I. and Loewen, P.C. (2000). Enzymology and structure of catalases. *Advances in Inorganic Chemistry*. **51**: 51-106.
- Poyraz, I., Şahin, B. and Atmaca, E. (2017). Detection of ten resistance genes against *P. syringae* pv. *phaseolicola* and *X. axonopodis* pv. *phaseoli* in twelve local bean varieties using SCAR markers. *Journal of the Institute of Science and Technology*. **7(2)**: 143-150.
- Shaik, S., Mahesh, H.B., Gangadhar, G. and Krishna, T.G. (2020). Molecular identification of bean genotypes for partial resistance to *Sclerotinia sclerotiorum* based on SCAR markers. *Journal of Plant Pathology*. **102(1)**: 369-375. <https://doi.org/10.1007/s42161-019-00432-1>.

- Singh, U.S., Singh, R.P. and Kohmoto, K. (1995). Pathogenesis and host specificity in plant diseases: Histopathological, biochemical, genetic and molecular bases. Pergamon Press.
- Singh, S.P. and Schwartz, H.F. (2010). Breeding common bean for resistance to diseases: A review. *Crop Science*. **50(6)**: 2199-2223.
- Teranishi, Y., Tanaka, A., Osumi, M. and Fukui, S. (1974). Catalase activities of hydrocarbon-utilizing *Candida* yeasts. *Agricultural and Biological Chemistry*. **38(6)**: 1213-1220.
- Waller, F., Achatz, B., Baltruschat, H., Fodor, J., Becker, K., Fischer, M. and Franken, P. (2005). The endophytic fungus *Piriformospora indica* reprograms barley to salt-stress tolerance, disease resistance and higher yield. *Proceedings of the National Academy of Sciences*. **102(38)**: 13386-13391.
- Yu, K., Park, S.J. and Poysa, V. (2000). Marker-assisted selection of common beans for resistance to common bacterial blight: Efficacy and economics. *Plant Breeding*. **119(5)**: 411-415.