Evaluation of common bean lines for their reaction to the common bacterial blight pathogen

EBRAHIM OSDAGHI¹, ALI ALIZADEH², MASOUD SHAMS-BAKHSH³ and MOHAMMAD REZA LAK⁴

¹Department of Plant Pathology, Tarbiat Modares University of Tehran, Iran ²Iranian Research Institute of Plant Protection, Tehran, Iran ³The Center for Agricultural Research in Markazi Province, Arak, Iran

Summary. Common bacterial blight (CBB) caused by *Xanthomonas axonopodis* pv. *phaseoli* (*Xap*) is an economically important disease of common beans (*Phaseolus vulgaris* L.). Since there is no satisfactory chemical control for CBB, the use of resistant cultivars is an important management strategy. In the present study, twenty nine lines and one cultivar of common bean were evaluated for their reaction to *Xap* under greenhouse and field conditions. The experiments were conducted in randomized complete blocks with three replications. Reaction to *Xap* was assessed as diseased leaf area (DLA) and the number of spots on the leaves. Data analysis indicated that cultivar Khomein and the Ks21479 and Ks31169 lines were the most susceptible, while Ks51103, BF13607 and BF13608 lines were the most resistant. The data obtained from greenhouse and field experiments were in agreement. None of the evaluated lines and/or cultivars was immune for CBB; however, the three resistant lines were identified for use in cultivation or as sources of resistance to CBB in plant breeding programs.

Key words: Xanthomonas axonopodis pv. phaseoli, Phaseolus vulgaris, Iran.

Introduction

Common bean (*Phaseolus vulgaris* L.), one of the most important crops in terms of both economy and nutrition, is cultivated in different regions of Iran including the Markazi, Lorestan and Isfahan provinces (Lak *et al.*, 2002b). Among the main causes for poor yields in common beans are fungal, viral and bacterial diseases (Ferreira *et al.*, 2003). Common bacterial blight (CBB), caused by *Xanthomonas axonopodis* pv. *phaseoli* (*Xap*), is a major seed-borne disease of common bean worldwide (Tar'an *et al.*, 2001; Miklas *et al.*, 2003) and can cause 10 to 40% loss in yield (Opio *et al.*, 1996). The amount of yield loss depends on the intensity of the disease, environmental conditions that

favor the onset and progress of the disease, and the degree of susceptibility of the cultivars (Asensio et al., 2006). In Iran, CBB was originally reported from Markazi province by Lak et al. (2002b) and during the past five years has become one of the major diseases of common bean in Iran leading to yield loss (Zamani, 2008). CBB has spread from Markazi to neighboring provinces such as Lorestan, Isfahan and Chahar-Mahale Bakhtiyari (Zamani, 2008). CBB is difficult to control as chemical control is reportedly not very effective (Zanatta et al., 2007). The best alternatives to manage CBB include use of clean, pathogen-free seed (Zanatta et al., 2007) and planting CBB-resistant cultivars (Webster et al., 1980; Rodrigues et al., 1999; Tar'an et al., 2001; Lak et al., 2002a; Miklas et al., 2003; Asensio et al., 2006). Cultural practices such as crop rotation, weed elimination and removal of plant debris also contribute to the management of CBB (Saettler, 1991). High levels of cultivar resistance would minimize yield losses, reduce bactericide use and production costs, and would facilitate

Corresponding author: M. Shams-Bakhsh E-mail: shamsbakhsh@modares.ac.ir

the use of an integrated disease management program and the production and distribution of pathogen-free seeds (Singh and Munoz, 1999).

Some loss may be attributed to pod lesions, which can result in seed infection during pod development and subsequent rotting and shriveling of the seed. Most yield loss, however, results from leaf lesions which lead to premature defoliation (Goodwin, 1992). Seeds infected with Xap are regarded as the primary source of inoculum initiating epidemics in established common bean fields (Jung et al., 1997). The seed-borne nature of the CBB pathogen (Darrasse et al., 2007) is a major barrier in commercial bean production and has significantly influenced the location of the common bean seed production industry in Iran (Lak et al., 2002b). In the Markazi province of Iran, where CBB severity can be high, production of certified common bean seed is no longer feasible or economical for producers, resulting in the displacement of previously successful seed production programs (Lak et al., 2002b). Incorporation of CBB resistance into high-yielding commercial common bean cultivars would greatly benefit growers and seed producers in the Markazi province by providing access to locally produced certified disease-free seed.

Resistance to CBB in common bean has been described as a quantitative trait with low to medium heritability (Silva et al., 1989), conditioned by between one to five genes with additive gene action (Tar'an et al., 2001). Efforts to pyramid CBB resistance genes from different sources have been initiated by Singh and Muñoz (1999) and Miklas et al. (2000). CBB resistance is also reportedly associated with plant architecture, indeterminate growth habit and late maturity (Tar'an et al., 2001). The complex nature of the resistance and the large environmental influence on symptom development make CBB resistance a difficult trait to screen for in plant breeding programs. Useful sources of resistance to CBB have been found in the related species P. coccineus (Yu et al., 1998; Welsh and Grafton, 2001) and P. acutifolius (Dursun et al., 1995; Yu et al., 1998; Singh and Munoz, 1999; Marquez et al., 2007), and interspecies crosses between P. vulgaris and either P. acutifolius or P. coccineus have frequently been used to transfer the resistance-related traits in common bean breeding programs (Tar'an et al., 2001). Despite the complexity, these resistance traits have been introduced into bean breeding lines. Resistance from tepary bean and runner bean has successfully been transferred to common bean. However, most of these original and derived sources of resistance are poorly adapted in tropical conditions (Silva et al., 1989).

CBB resistance was evaluated in seedlings and adult plants of *P. vulgaris* under both greenhouse and field condi-

tions by Webster *et al.* (1980), using plants of F4 families derived from two susceptible × resistant crosses. Over 90% of the variation among disease scores of field-grown families was attributable to the regression on scores of greenhouse-grown seedlings.

Most commercial cultivars of common bean grown in Iran appear to be highly susceptible to CBB, chemical control is either ineffective or uneconomical to bean growers, and the disease is continuing to spread to other areas in Iran (Dursun *et al.*, 2002). The National Center for Bean Research in Iran is the main source of seed for farmers all over the country and therefore identification of any cultivars/lines with resistance to the CBB pathogen held in their collection would be of great benefit. The purpose of the present study was to investigate the reaction of different lines and cultivars to CBB under both greenhouse and field conditions in order to identify those with resistance to CBB.

Materials and methods

Plant materials

Twenty nine lines of common bean including white, red, cranberry, cream and black-white types of seed were sourced from the National Center for Bean Research in Khomein, Markazi province. The cranberry cultivar Khomein, the most widely grown common bean in Iran (Jahan-nema, 2000), was also included. Names and characteristics of these lines and cultivar are provided in Table 1. All common bean lines and one cultivar were screened for their reactions to the CBB pathogen in both greenhouse and field experiments. Most of these cultivar/lines were grown by National Center for Bean Research in Khomein and sent to other farmers of neighboring provinces for planting.

Sampling and bacteria isolations

Bacterial strains of *Xap* were isolated from common bean leaves collected during surveys in the Markazi province. In these surveys, representative common bean fields were randomly selected and surveyed for CBB symptoms, and leaves with typical CBB symptoms (irregular necrotic lesions with yellow borders and water-soaked spots) were collected and dried between paper towels. For each leaf sample, tissues (c.16 mm²) were excised from the lesion margin, placed in a drop of distilled water on a microscope slide, and macerated. Loopfuls of macerate were streaked onto nutrient agar (NA) and the plates were incubated at 28°C for 24 h. Yellow, mucoid, xanthomonad-like colonies were selected from each leaf sample and subcultured on NA.

No.	Cultivar/Line Name	Greenhouse experiment		Field experiment		Sood Trme
		Mean of disease scales ±S.D.	Disease response ^a	Mean of disease scales ±S.D.	Disease response ^a	_ Seed Type
1	BF 13607	2±0.0	R	1.5 ± 0.0	R	Black-white
2	BF 13608	2±0.0	R	1.5 ± 0.0	R	Black-white
3	$\mathrm{Ks}\ 21115$	4±0.0	$_{ m HS}$	3.83 ± 0.16	S	Cranberry
4	Ks 21143	3.66 ± 0.16	S	4.16 ± 0.16	$_{ m HS}$	Cranberry
5	Ks 21400	3.33 ± 0.16	S	3.16 ± 0.16	S	Cranberry
6	Ks 21425	4±0.0	$_{ m HS}$	2.16 ± 0.16	\mathbf{MS}	Cranberry
7	Ks 21479	5 ± 0.0	$_{ m HS}$	3.16 ± 0.16	S	Cranberry
8	Ks 21480	3.33 ± 0.16	S	3.83 ± 0.16	S	Cranberry
9	$\mathrm{Ks}\ 21487$	4±0.0	$_{ m HS}$	3.33 ± 0.16	S	Cranberry
10	$\mathrm{Ks}\ 31104$	3.66 ± 0.16	S	3.16 ± 0.16	S	Red
11	$\mathrm{Ks}\ 31115$	3.66 ± 0.16	S	4.16 ± 0.16	HS	Red
12	Ks 31139	3±0.0	S	2.16 ± 0.16	\mathbf{MS}	Red
13	Ks 31162	3.66 ± 0.16	S	3.16 ± 0.16	S	Red
14	Ks 31163	3.66 ± 0.16	S	3.16 ± 0.16	S	Red
15	Ks 31164	3.33 ± 0.16	\mathbf{S}	4.5 ± 0.0	$_{ m HS}$	Red
16	$\mathrm{Ks}\ 31165$	3.66 ± 0.16	\mathbf{S}	4.33 ± 0.16	$_{ m HS}$	Red
17	Ks 31166	3±0.0	\mathbf{S}	3.33 ± 0.16	S	Red
18	$\mathrm{Ks}\ 31167$	3.33 ± 0.16	\mathbf{S}	4.33 ± 0.16	$_{ m HS}$	Red
19	Ks 31169	4.5 ± 0.0	$_{ m HS}$	2.66 ± 0.16	\mathbf{MS}	Red
20	Ks 41101	3.66 ± 0.16	\mathbf{S}	4.33 ± 0.16	$_{ m HS}$	White
21	Ks 41103	3±0.0	\mathbf{S}	4.33 ± 0.16	$_{ m HS}$	White
22	Ks 41104	3±0.0	\mathbf{S}	3.83 ± 0.16	S	White
23	Ks 41124	3±0.0	\mathbf{S}	3.16 ± 0.16	\mathbf{S}	White
24	Ks 41128	3±0.0	S	3.16 ± 0.16	S	White
25	Ks 41231	3±0.0	\mathbf{S}	2.83 ± 0.16	${ m MS}$	White
26	Ks 41232	3.66 ± 0.16	\mathbf{S}	3.83 ± 0.16	S	White
27	$\mathrm{Ks}\;41234$	3±0.0	\mathbf{S}	4.33 ± 0.16	$_{ m HS}$	White
28	$\operatorname{Ks}41235$	$4{\pm}0.0$	HS	3.33 ± 0.16	S	White
29	ks51103	2 ± 0.0	R	1.5 ± 0.0	R	Cream
30	Khomein	5 ± 0.0	HS	5 ± 0.0	$_{ m HS}$	Cranberry

Table 1. List of cultivar/lines used in both greenhouse and field experiments and disease response as mean of disease scales \pm S.D. of diseased leaf area (DLA) and of number of spots on the leaves.

^a Disease response: R, resistant; MS, moderately susceptible; S, susceptible; HS, highly susceptible.

Identification of bacterial isolates

Bacterial isolates were identified using standard bacteriological tests (Schaad et al., 2001). Xap-specific primers (X4c,5'-GGC AAC ACC CGA TCC CTA AAC AGG-3'and X4e, 5'-CGCCGG AAG CAC GAT CCT CGA AG-3') were used to confirm the identity of isolates (Audy et al., 1994). DNA was extracted as following: bacteria were grown overnight and centrifuged at 4000 g for 3 min, the pellet was resuspended in TE buffer (1 mM EDTA, 10 mM Tris-HCl, pH 8) and boiled for 10 min, then centrifuged at 12,000 g for 5 min. The supernatant was used for PCR. PCR amplification was performed in a 20 µl reaction volume containing 2 µL of extracted template DNA, 2 µLPCR buffer (10x, CinnaGen, Tehan, Iran), 0.6 µL MgCl2 (1.75 mM), 0.4 µL of deoxynucleotide triphosphates (10 mM), 1 µL of each primer and 0.25 µL Taq polymerase (1.25 U µL⁻¹, CinnaGen). PCR reactions were performed in a thermocycler (Eppendorf, Hamburg, Germany). The PCR was incubated at 95°C for 1 min, followed by 30 cycles of 95°C for 1 min, 65°C for 1 min and 72°C for 2 min, and a final extension at 72°C for 10 min. The Xap-19 strain obtained from the Center for Agricultural Research in Markazi Province-Iran (Lak et al., 2002b) was used as a positive Xap strain. The PCR reaction products were analyzed by electrophoresis on a 1.2% agarose gel in 1× TBE buffer (90 mM Tris-borate, 2 mM EDTA) followed by staining with ethidium bromide (0.5 μg mL⁻¹). DNA molecular weight markers (GeneRulerTM 1 kb DNA ladder, Fermentas) were used to determine the size of the amplified fragments.

Pathogenicity assays

Selected *Xap* isolates (Araxa1 and Araxa2), maintained in the collection of the Iranian Research Institute of Plant Protection, Tehran, Iran, were streaked onto plates of NA and grown for 48 hrs at 28°C. Cells were suspended in distilled water and adjusted to c. 10^8 CFU ml⁻¹ according to Mkandawire *et al.* (2004). Three seeds of Khomein cultivar of common bean were planted in 6-in. pots. The bacterial mixture was spread onto the first fully expanded trifoliate leaves of bean plants (c. 15 days after planting), and distilled water was spread onto leaves of plants serving as negative controls. The floor of the greenhouse was kept wet to generate humidity in order to favor development of CBB.

Greenhouse trials

Seeds of the different common bean lines and one cultivar were sterilized in 20% sodium hypochlorite for five minutes and washed with distilled water twice. The experimental unit consisted of three plants per 20 cm plastic pot containing parts of perlite, soil and peat (1:1:1

by volume), with three replications totalling 9 plants per treatment. Pots were arranged on a greenhouse bench in a randomized complete block design. The greenhouse temperature was maintained at 25°C±2 throughout, with 16 h light and 8 h dark. The Araxa1 isolate suspension, adjusted to 10⁸ CFU ml⁻¹ in distilled water, was prepared from 48 hour-old cultures grown on NA according to Mkandawire et al. (2004). When the trifoliate leaves of the plants were fully expanded (fifteen days old plants), bacterial suspension was sprayed onto the aerial parts of the plants. The plants were then covered by transparent nylon for three days after inoculation, as described by Dursun et al. (2002) and Lak et al. (2002b). The reaction of the bean plants to Xap isolates was assessed as diseased leaf area, as described by Souza et al. (2000), and number of spots on the leaves, as described by Lak et al. (2002b). Symptoms were assessed using the following scale: 0, symptomless; 1, negligible symptoms or slight marginal necrosis; 2, water-soaking, chlorosis, or necrosis (blight) in <25% of the inoculated area; 3, 25-50% blight; 4, 50-75% blight and 5, complete necrosis of leaves (Dursun et al., 2002; Lak et al., 2002b).

Field experiment

Field experiments were conducted at the Center of Agricultural Research, Markazi province field, Arak, during May 2008 to investigate the reaction of different common bean lines and one cultivar to Xap isolates. Seeds of common bean cultivar/lines were sown in plots measuring 2×2 m in three rows 2 m long spaced 45 cm apart. The plots were spaced 1 m apart and prepared and planted according to standard commercial practices. The experimental design was a randomized complete block with three replications. Plots were irrigated as needed for standard plant growth. No herbicides were used, but fertilizer (urea 46%, 150 kg hec⁻¹) was added as needed in the same amount for all bean lines. Two treatments were applied in the experiment: (i) inoculation with Xap and (ii) control (spread with distilled water). Inoculum of Araxa1 isolate was obtained from a 2-day-old culture grown on NA at 28°C. Plants were sprayed at time of flowering with a suspension containing about 108 CFU ml-1 delivered via a pressure sprayer at nozzle pressures, approximately 50 ml of suspension per plant. Plants were sprayed with water prior to inoculation in order to provide a favorable microclimate for bacterial infection. Plants were rated for disease reaction and CBB symptoms from 20-25 days after inoculation (Lak et al., 2002b). Ten plants per each treatment were selected for CBB symptom evaluation and CBB symptoms were assessed using the scale mentioned above.

Statistical analysis

For each of the greenhouse and field experiments, disease severity index was statistically analysed using analysis of variance (ANOVA; P=0.05). Duncan's multiple range tests were used to separate treatment means. A combined analysis was performed for each study, and the data presented are the combined results of the repeat experiments. MSTAT C computer software (Russell and Eisensmith, 1983) was used for statistical analyses.

Results

Bacterial isolation and identification

Bacterial strains isolated onto NA from leaves showing CBB symptoms and collected from common bean fields in Markazi province were identified as xanthomonad-like, based on yellow, convex, mucoid colony morphology, gram negative reaction, hypersensitive reaction on tobacco, potato soft-rotting negative, levan positive, inhibition of growth by 2.5% NaCl, catalase positive, oxidase weak, gelatin hydrolysis positive biochemical characteristics. As expected, a 700-bp DNA fragment was amplified with the X4c/X4e primer pair (Figure 1). All of our isolates obtained in field surveys and identified as pathogenic on common bean were *X. axonopodis* pv. *phaseoli*.

Pathogenicity assays

Two isolates of *Xap*, called Araxa1 and Araxa2, were used in the initial pathogenicity assays. Fifteen days after inoculation, characteristic symptoms of CBB (irregular necrotic lesions with yellow borders and water soaked spots) were observed. The Araxa1 elicited more severe symptoms on all common bean plants and was selected and used in both greenhouse and field experiments.

Cultivar/lines evaluation for CBB

Characteristic symptoms of CBB were observed on inoculated plants of all twenty nine lines and one cultivar of common bean that were used in this study 10–12 days (in greenhouse experiments) and 20–25 days (in field experiment) after inoculation, with different disease severity values ($P \le 0.01$). Symptoms of CBB developed on almost all inoculated plants, suggesting that none of the studied cultivar/lines were immune. Bacterial blight lesions were

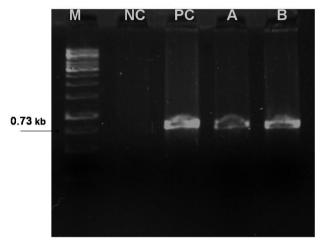


Figure 1. Ethidium bromide stained gel of PCR products directed by X4c and X4e primers, template DNAs were from *X. axonopodis* pv. *malvacearum* (NC, negative control), the Xap-19 strain of *X. axonopodis pv. phaseoli* (PC, positive control), Araxa 1 and Araxa 2 isolates (lane A and B respectively). Molecular weight standard (GeneRuler TM1 kb DNA ladder, Fermentas) was run in the left lane (M).

Table 2. Analysis of variance in greenhouse and field evolutionary experiments for common bean cultivar/lines resistance against common bacterial blight. Common bean cultivar/lines were planted in randomized complete blocks and data were obtained from means of 9 plants (3 plants per each replication).

Source	0	Greenhouse	Field		
Source	DF	$Mean \ of \ Squares^a$	DF	Mean of Squares ^a	
Replication	2	0.206	2	0.035	
Factor A	29	0.682^{**}	29	1.348^{**}	
Factor B	1	533.889**	1	537.339**	
A×B	29	0.682**	29	1.348^{**}	
Error	118	0.121	118	0.035	

^a Significance level according to Duncan's multiple range test: ** P=0.01.

located predominantly on the margins and near the base of the leaflets. The percent leaf area infected was significantly $(P \le 0.01)$ different between various cultivar/lines (Table 2). In the greenhouse, the CBB symptoms developed mostly on leaves, and pod infection was rarely observed even in the very susceptible cultivar Khomein. In the field experiment, however, both leaf and pod infection was observed.

Cultivar/lines comparison

Significant differences in CBB symptoms were observed between common bean lines and cultivar in both greenhouse and field experiments (Table 2). Three of the common bean lines (Ks51103, BF13607 and BF13608) were resistant to *Xap*. The remaining lines were assigned to three susceptibility groups (moderately susceptible, susceptible, and highly susceptible) based on the Average Disease Severity Rating (ADSR) according to Dursun *et al*. (2002), which includes Diseased Leaf Area (DLA) (Souza *et al.*, 2000) and numbers of spots on the leaves (Lak *et al.*, 2002b) in both greenhouse and field experiments. The Khomein cultivar, Ks21479 and Ks31169 lines were very susceptible to CBB (Table 2). Cultivar Khomein was the most susceptible in both greenhouse and field experiments (Table 1).

Comparison of field and greenhouse experiments

The relative CBB ratings for the various lines were the same for both field and greenhouse experiments, but most lines were more resistant when grown under field conditions. As noted before, cultivar Khomein was the most susceptible, and Ks51103, BF13607 and BF13608 were the most resistant. The resistance of these latter three lines was higher under field conditions than in the greenhouse (Table 1). Ks21479 was very susceptible in the greenhouse, but moderately susceptible in the field experiment. Ks31169 was also more resistant in the field. Greenhouse conditions are more favorable for disease development than field conditions due to optimised temperature and humidity.

Discussion

Twenty nine Iranian lines and one Iranian cultivar of common bean were evaluated for the first time for their reaction to the CBB pathogen under greenhouse and field conditions during the 2008 growing season, resulting in the identification of three common bean lines Ks51103, BF13607 and BF13608 as possible new sources of CBB resistance. However, although Ks51103, BF13607 and BF13608 lines were resistant to CBB and high-yielding, these lines are not marketable for Iranian farmers or con-

sumers but could be utilized in plant breeding for developing CBB resistant bean cultivars/lines for Iran.

There is some evidence that CBB resistance can be incorporated from lines into bean cultivars in common bean breeding programs. Ferreira *et al.* (2003) evaluated 109 recombinant inbred lines (F7) of *P. vulgaris* originating from the cross HAB-52 (susceptible-snap bean)×BAC-6 (resistant-common bean) and obtained increasing heritability results for disease index and variation index from 26.85% and 0.26%, respectively in F3, to 91.77% and 1.36%, respectively in F7. Rodrigues *et al.* (1999) combined three snap bean genotypes (Alessa, Hab 52 and Hab 198) and two dry bean genotypes (Bac-6 and A 794) and observed increasing resistance. Therefore, the results of this present study, plus those of Dursun *et al.* (2002), are important sources of information for the development of Iranian cultivars/lines of common bean resistant to CBB.

Resistance reactions are also influenced by environmental factors and farming methods. The source of inoculum is important. Fininsa and Tefera (2001) demonstrated that CBB incidence on common beans grown in infested debris-inoculated plots was 59% higher than incidence on beans from treated seed plots, and CBB on beans from untreated seed and from infested debris-inoculated plots was more severe than on beans from infested soil-inoculated plots. They also found that bean seed yield and quality were influenced by the inoculum source. Lower seedling emergence, stand count and seed yield were obtained from infested debris- and soil-inoculated plots. Gilbertson et al. (1988) investigated the role of seed inoculum versus debris infestation in disease development by Xap. They found that dry leaf debris remained a source of viable inoculum for up to six years. Prior inoculation appeared to have no effect on the disease reactions of subsequently inoculated leaves and pods (Ariyarathne et al., 1994), indicating that different plant parts may be inoculated at different times for assessing CBB resistance for plant breeding purposes.

Lak *et al.* (2002b) observed that irrigation systems such as sprinkler and flood irrigation influenced CBB symptom development and yield loss. Webster *et al.* (1983) showed that climate also influenced susceptibility to CBB by affecting plant growth and maturity. The common bean cultivar Jules and the PI 207262 line are moderately resistant to *Xap* when grown in temperate conditions, but were susceptible in field evaluations in the tropics (Webster *et al.*, 1983).

To conclude, we found that greenhouse and field experiments gave similar results. Therefore, glasshouse screening could be used for assessing CBB resistance in plant breeding programs instead of time-consuming and costly field experiments. The present study demonstrated the existence of resistance sources against CBB within Iranian common bean cultivar/lines that could potentially be used for breeding resistant cultivars. This is the first study and report of resistance against common bacterial blight in Iranian common bean cultivars/lines.

Acknowledgements

We thank the National Center for Bean Research in Khomein, Markazi province, for providing us with common bean seeds lots. Financial support of Tarbiat Modares University is acknowledged.

Literature cited

- Ariyarathne H.M., D.P. Coyne, A.K. Vidaver and K.M. Eskridge, 1994. Effect of prior inoculation of leaves of dry beans with the common blight pathogen on the disease reaction of subsequently inoculated leaves and pods and the reaction of detached versus attached pods. *HortScience* 29, 427–581.
- Asensio M.C., C. Asensio and S.P. Singh, 2006. Gamete selection for resistance to common and halo bacterial blights in dry bean intergene pool populations. *Crop Science* 46, 131–135.
- Audy P., A. Laroche, G. Saindon, H.C. Huang and R.L. Gilbertson, 1994. Detection of the bean common blight bacteria, *Xanthomonas campestris* pv. *phaseoli* and *X. c.* pv. *phaseoli fuscans*, using the polymerase chain reaction. *Phytopathology* 84, 1185–1192.
- Darrasse A., C. Bureau, R. Samson, C.E. Morris and M.A. Jacques, 2007. Contamination of bean seeds by *Xanthomonas axonopodis* pv. *phaseoli* associated with low bacterial densities in the phyllosphere under field and greenhouse conditions. *European Journal* of *Plant Pathology* 119, 203–215.
- Dursun A., D.P. Coyne, M.F. Mohamed and G. Jung, 1995. Inheritance of resistance to common bacterial blight in tepary beans. *HortScience* 30, 839–840.
- Dursun A., M.F. Donmez and F. Sahin, 2002. Identification of resistance to common bacterial blight disease on bean genotypes grown in Turkey. *European Journal of Plant Pathology* 108, 811–813.
- Ferreira C.F., M.G. Pereira, A.D.S.D. Santos, R. Rodrigues, R.E. Bressan-Smith, A. PioViana and R.E.F. Daher, 2003. Resistance to common bacterial blight in *Phaseolus vulgaris* L. recombinant inbred lines under natural infection of *Xanthomonas axonopodis* pv. phaseoli. Euphytica 134, 43–46.
- Fininsa C. and T. Tefera, 2001. Effect of primary inoculum sources of bean common bacterial blight on early epidemics, seed yield and quality aspects. *International Journal of Pest Management* 47(3), 221–225.
- Gent D.H., J.M. Lang and H.F. Schwartz, 2005. Epiphytic survival of Xanthomonas axonopodis pv. allii and X. axonopodis pv. phaseoli on leguminous hosts and onion. Plant Disease 89, 558–564.
- Gilbertson R.L., R.E. Rand, E. Carlson and D.J. Hagedorn, 1988. The use of dry-leaf inoculum for establishment of common bacterial blight of beans. *Plant Disease* 72, 385–389.

Goodwin P.H., 1992. Effect of common bacterial blight on leaf

photosynthesis of bean. *Canadian Journal of Plant Pathology* 14, 203–206.

- Jahan-nema F., 2000. Social and economic factors that affected the acceptation of Khomein common bean cultivar. *Agricultural and Economic Development* 8(31), 261–287.
- Jung G., P.W. Skroch, D.P. Coyne, J. Nienhuis, E. Arnaud-Santana, H.M. Ariyarathne, S.M. Kaeppler and M.J. Bassett, 1997. Molecular marker-based genetic analysis of tepary bean-derived common bacterial blight resistance in different developmental stages of common bean. *Journal of American Society for Horticultural Science* 122, 329–337.
- Lak M.R., M. Bahar and M. Shams-Bakhsh, 2002a. Comparison of serological methods for identification of common bacterial blight in bean seed in Markazi provinc. *Iranian Journal of Plant Pathology* 38, 193–206.
- Lak M.R., M. Shams-Bakhsh and M. Bahar, 2002b. Identification of the bacterial agent of bean leaf and pod blight in Markazi province. *Journal of Science and Technology of Agriculture and Natural Resource* 6(1), 231–243.
- Marquez M.L., H. Teránb and S.P. Singh, 2007. Selecting common bean with genes of different evolutionary origins for resistance to *Xanthomonas campestris* pv. *phaseoli*. *Crop Science* 47, 1367–1374.
- Miklas P.N., D.P. Coyne, K.F. Grafton, N. Mutlu, J. Reiser, D.T. Lindgren and S.P. Singh, 2003. A major QTL for common bacterial blight resistance derives from the common bean great northern landrace cultivar Montana. *Euphytica* 131, 137–146.
- Miklas P.N., J.R. Smith, R. Riley, K.F. Grafton, S.P. Singh, G. Jung and D.P. Coyne, 2000. Marker-assisted breeding for pyramided resistance to common bacterial blight in common bean. *Annual Report of Bean Improvement* 43, 39–40.
- Mkandawire A.B.C., R.B. Mabagala, P. Guzman, P. Geots and R.L. Gilbertson, 2004. Genetic diversity and pathogenic variation of common blight bacteria (*Xanthomonas azxonopodis* pv. *phaseoli* and *Xanthomonas axonopodis* pv. *phaseoli* var. *fuscans*) suggest pathogen coevolution with the common bean. *Phytopatology* 94, 593–603.
- Opio A.F., D.J. Allen and J.M. Teri, 1996. Pathogenic variation in *Xanthomonas campestris* pv. *phaseoli*, the causal agent of common bacterial blight in *Phaseolus* Beans. *Plant Pathology* 45, 1126–1133.
- Rodrigues R., N.R. Lea, M.G. Pereira and A. Lam-Sánchez, 1999. Combining ability of *Phaseolus vulgaris* for resistance to common bacterial blight. *Genetic and Molecular Biology* 22(4), 571–575.
- Russell D.F. and S.P. Eisensmith, 1983. MSTAT-C. Crop and Soil Science Department, Michigan State University, MI, USA.
- Saettler A.W., 1991. Common bacterial blight. In: Compendium of Bean Diseases. R.Hall (ed.) St. Paul, MN, USA, APS, 29–30.
- Schaad N.W., J.B. Jones and W. Chun, 2001. Laboratory Guide for Identification of Plant Pathogenic Bacteria, Third edition. APS, St. Paul, MN, USA, 379 pp.
- Silva L.O., S.P. Singh and M.A. Pastor-Corrales, 1989. Inheritance of resistance to bacterial blight in common bean. *Theoretical and Applied Genetic* 78, 619–624.
- Singh S.P. and C.G. Muñoz, 1999. Resistance to common bacterial blight among *Phaseolus* species and common bean improvement. *Crop Science* 39, 80–89.

- Souza A.A., R.L. Boscariol, D.H. Moon, L.E.A. Camargo and S.M. Tsai, 2000. Effects of *Phaseolus vulgaris* QTL in controlling hostbacteria interactions under two levels of nitrogen fertilization. *Genetics and Molecular Biology* 23(1), 155–161.
- Tar'an B., T.E. Michaels and K.P. Pauls, 2001. Mapping genetic factors affecting the reaction to *Xanthomonas axonopodis* pv. *phaseoli* in *Phaseolus vulgaris* L. under field conditions. *Genome* 44, 1046–1056.
- Webster D.M., T. Falls, S.R. Temple and G. Galvez, 1983. Expression of resistance to *Xanthomonas campestris* pv. *phaseoli* in *Phaseolus vulgaris* under tropical conditions. *Plant Disease* 67, 394–396.
- Webster D.M., S.R. Temple and H.F. Schwartz, 1980. Selection for resistance to *Xanthomonas phaseoli* in dry beans. *Crop Science*

20, 519–522.

- Welsh M.M. and K.F. Grafton, 2001. Resistance to common bacterial blight of bean introgressed from *Phaseolus coccineus*. *Hort-Sci*ence 36, 645–814.
- Yu Z.H., R.E. Stall and C.E. Vallejos, 1998. Detection of genes for resistance to common bacterial blight of beans. *Crop Science* 38, 1290–1296.
- Zamani Z., 2008. Genetic Diversity of Common Bacterial Blight of Bean (Xanthomonas axonopodis pv. Phaseoli). MS Thesis, Isfahan University of Technology, Isfahan, Iran.
- Zanatta Z.G.C.N., B.A. Moura, L.C. Maia and A.S. Santos, 2007. Bioassay for selection of biocontroller bacteria against bean common blight (*Xanthomonas axonopodis* pv. *phaseoli*). *Brazilian Journal of Microbiology* 38, 511–515.

Accepted for publication: August 31, 2009