

RESPONSE OF COMMON BEAN GENOTYPES TO PREVALENT *PSEUDOCERCOSPORA GRISEOLA* RACES CAUSING ANGULAR LEAF SPOT IN UGANDA

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ABSTRACT

Angular Leaf Spot (ALS) caused by *Pseudocercospora griseola* is responsible for 54% yield loss in Uganda's common beans. Host plant resistance is a safe and cost-effective management strategy for this disease. Identification of resistant common bean genotypes to prevailing races is vital to utilize the crop. Therefore, the objective of this study was to identify genotypes that are resistant to the commonly occurring and virulent *P. griseola* races in Uganda for breeding purposes. Twenty-seven common bean genotypes and three control varieties (Mexico54, MCM5001, and CAL96) were screened at field conditions for ALS resistance at testing site (National Agricultural Research Laboratories - Kawanda) under natural disease infection. The genotypes were also evaluated in the screen house using frequently occurring *P. griseola* races: 61:63, 1:6 and 21:39. Variability in the severity of ALS on both leaves and pods was significant whereas the difference between seasons and the interaction between the seasons and genotypes was only significant for yield. The disease severity scores were higher (mean of 3.2) on leaves than on pods (mean of 2.9). Ninety-three percent, 33.3% and 15% of the genotypes were resistant to *P. griseola* races 21:39, 1:6 and 61:63, respectively. A large-seeded genotype AFR703 was resistant to all the three *P. griseola* races. A medium seed size genotype AFR702 and three small seed genotypes (G148, G18842 and G6727) were resistant to both 21:39 and 1:6 but moderate resistance to 61:63 whereas a large-seeded genotype AND279 was resistant to both 61:63 and 21:39 but moderate to 1:6. All of these six genotypes (AFR703, AFR702, G148, G18842, G6727 and AND279) expressed moderate resistance to *P. griseola* races on leaves under field conditions. Thus, these common bean genotypes could be used as sources of ALS resistance for breeding programs to address the ALS constraint; and genes responsible for resistance have to be characterized.

Key words: Pathology, Disease, Resistance, Infection, BALSIT, Pathotypes, Prevalent, Races



INTRODUCTION

The common bean (*Phaseolus vulgaris* L.) is an important source of protein (45%), carbohydrate (25%), fiber, vitamins including B complex and minerals such as iron, zinc and sulfur [1-3]. Despite its contribution to the human diet, bean on-farm yields are lower (1.5 t/ha) than the potential yield of 2.5 – 3.5 t/ha under experimental condition [4, 5]. The low yields are attributed to several constraints such as diseases, insect pests, low soil fertility and periodic water stress [6]. Among the major foliar diseases, angular leaf spot (ALS), common bacterial blight (CBB), the bean common mosaic viruses (BCMV), bean common mosaic necrotic viruses (BCMNV) and bean rust (*Uromyces appendiculatus*) are the most devastating biotic constraints to bean production in Uganda [7, 8]. Angular leaf spot (caused by *Pseudocercospora griseola*) is responsible for 54% yield loss in Uganda [9] and 61% in Tanzania [10]. The severity of ALS depends on the variety, environmental conditions, earliness of the infection and pathogenicity of the isolates or pathotypes [9]. Disease development is favored by the temperature of 20 – 25°C, relative humidity of 95 – 100% and altitude of less than 1600 meters above sea level [11, 12].

Pseudocercospora griseola survives between seasons on infested seed and debris. The spores produced on infected debris or seed are rain-splashed and/or wind-blown onto healthy tissue after planting where they could germinate and infect susceptible tissue through natural pores [13]. There are two race categories of the *P. griseola* pathogen races (the Andean and the Mesoamerican) developed in each of Andean and Mesoamerican common bean gene pool separately [14]. The Mesoamerican races exhibit a much broader virulence spectrum by also infecting Andean beans although with a less severe effect [15]. The two races usually co-exist on infected leaves and cannot be differentiated based on symptoms or morphology [16].

Angular leaf spot disease symptoms appear on aerial plant parts such as leaves, petioles, stems and pods. However, symptoms are most recognizable on leaves. Lesions on leaves usually appear as brown spots with a tan or silvery center that are initially confined to tissue between major veins, which give an angular appearance to the lesions. The lesions result in defoliation as well as reduction in the photosynthetic area, leading to yield loss [17]. The infection can also reduce common bean quality by causing lesions on pods and seeds [18]. The pathogen can survive as spores in infected bean residue left on the soil surface although it does not persist for long when the remains of infected beans are covered up in the soil and decompose.



Pseudocercospora. griseola pathogen is a very variable pathogen as detected by molecular markers and the ALS differential cultivars [15, 19, 20]. Races 61:11 and 63:51 were detected and reported as the most virulent in Puerto Rico whereas six races were identified in western Kenya with race 63:63 being the most virulent [19]. A total of 23 races were identified in Ethiopia with races 63:59 and 19:33 being the most frequent [20] and 20 races were characterized in Tanzania with race 63:63 as the most virulent [10]. In Uganda, among the identified 12 races, 61:63, 5:55, 21:39, 17:23, 17:39 and 1:6 were the most virulent where 61:63 was able to overcome resistant genes in 11 known sources of resistance that constituted part of the differential set [7].

The presence of high pathogenic variability of *P. griseola* has been observed to lead to race variation among the different locations making it possible for a genotype to be resistant to ALS in one location and susceptible in another location. An example of this is, an Andean bean genotype CAL143 was resistant to all the races in Malawi, Rwanda and the Democratic Republic of Congo, intermediate to some Ugandan races and susceptible to race 63:21 in Uganda [21]. Intercropping, crop rotation and the use of varietal mixtures have been used in the management of the disease [22, 23]. However, the use of these measures has been limited by small plot sizes and there is lack of knowledge about proper variety mixing [23]. The use of chemical fungicides is another effective strategy but fungicides are costly to a small scale farmer and are unsafe for the environment and the users [10]. Genetic resistance is, therefore, considered the most applicable, environment friendly and cost-effective strategy for small-scale common bean farmer [11, 17].

The Bean Angular Leaf Spot International Test (BALSIT) nursery is a collection of the best ALS resistance sources identified from evaluations conducted mostly in Colombia and Brazil, and tested internationally across several locations including Africa [28]. Among others, Mexico54, Cornell 49-242, BAT332, Oura Negro, CAL143 and AND277 are some sources of resistance to *P. griseola* [21, 24-26]. However, due to the complexity and the variability of the ALS pathogen, these cultivars succumb to prevalent ALS races in certain locations and resistant to races in other locations. For example, the line BAT332 was found to be resistant in Colombia but was susceptible in Brazil whereas G15396A was resistant to Colombian races; 1:55 and 63:15 but susceptible to the Ugandan isolate 63:21[21]. This variability complicates the development of resistant cultivars, successful breeding for a broad ALS resistance should involve broader sources of genes of Andean and Mesoamerican origin [21]. Therefore, the objective of this study was to

identify genotypes that are resistant to the commonly occurring and the most virulent *P. griseola* races in Uganda: 1:6, 21:39 and 61:63.

MATERIALS AND METHODS

Description of the experimental site

The study was conducted at the International Center for Tropical Agriculture (CIAT) located at the National Agricultural Research Laboratories Institute (NARL). The institute is found in Kawanda, Nabweru Sub-County of Wakiso district in Uganda, about 13 kilometers from Kampala. The experimental site is located at longitude 45°N and latitude 48°E, 1300 meters above sea level, 21.7°C average temperature and 1242 mm average rainfall.

Experimental materials

Twenty-seven genotypes from the BALSIT nursery and three control genotypes (MCM5001, Mexico54, and CAL96) were evaluated (Table 1). Mexico54, a Mesoamerican resistant check possesses the "Phg-2" gene that controls resistance to ALS disease, MCM5001 and CAL96 are Mesoamerican and Andean susceptible checks for ALS, respectively. MCM5001 bears the "I" and "bc3" gene for BCMV/BCMNV resistance.

Screening for resistance to ALS under field conditions

The genotypes were evaluated in the field under natural disease infection for resistance to ALS. The trial was set up at CIAT, Kawanda field, where 80 seeds per genotype were planted in a randomized complete block design (RCBD) at a spacing of 50 cm between the rows and 10 cm between the seeds in two replications in the second rainy season (September- December, 2012) and in the first rainy season (March-June, 2014). Three control genotypes, Mexico54 (resistant check for ALS), MCM5001 (a susceptible check for ALS), and CAL96 (susceptible check for ALS) were included in the experiment. Weeding was done twice in each season (when the crop was at third trifoliolate formation (V3 growth stage) and at the pod formation (R7stage)) with no fertilizer or fungicide or pesticide applied. Besides the yield data at harvest, disease severity data for ALS was collected on leaves and pods of ten randomly selected plants at pod formation using a 1-9 CIAT standard rating scale, where, 1= No visible symptoms, 3 = small lesions which are covering 2% of the leaf area, 5 = several small lesions covering 5% of the leaf area with sporulation, 6, 7 = abundant large sporulating lesions, 9 = large sporulating lesions covering 25% leaf area. Scores 1–3 mean resistance, 4–6 intermediate and 7–9 susceptibility.



Evaluating the genotypes for resistance to ALS under screen house conditions

Disease inoculum was prepared from monosporic cultures of three most virulent *P. griseola* races: 61:63, 1:6 and 21:39 which were collected from infected fields of different parts of Uganda and maintained on petri dishes containing 1000 mL V8 media manufactured by the Campbell soup company, Camden, New Jersey, USA (200 ml V-8 juice, 3 gm CaCO₃, 20 gm Bacto agar, and 800ml double distilled water) at the CIAT laboratory in Uganda [7]. To regenerate the cultures, pure isolates were sub-cultured onto fresh media and 100 µl of sterile water was added onto each plate to make spore suspension. The spore suspension was sub-cultured on media containing V8 agar media and incubated for 14 days at 24°C to allow more sporulation. To prepare the inoculum, the plates on which isolates were grown were flooded with 100 µl of sterile distilled water, and the surface was scraped with a glass rod to release the conidia. The dislodged conidia were filtered through a sterile cheese cloth and the suspension was collected and diluted in distilled sterile water to obtain an inoculum containing 3-4×10⁴ spores per ml [24]. The experiment was laid out in the screen house as a randomized complete design (RCD) with three replications during the seasons 2012b and 2014b for races 12:39 and 1:6, 2017a and 2017b for race 61:63. "a" is the first rainy season in which the first planting of the year is done (March-June) and "b" is the second rainy season which is also the second planting season of the year (September - December). Each replication consisted of 27 BALSIT genotypes as treatments, one resistant control genotype (Mexico54), and two susceptible control genotypes (MCM5001 and CAL96). In preparation for planting, 60 five-liter buckets per replication were assembled, each bucket was three-quarter way filled with sterilized soil that consisted of a mixture of forest soil, lake sand, and animal manure in a ratio of 3:1:1. Each genotype was planted in 2 five-liter buckets, and 5 seeds were planted in each bucket, making 10 seeds per genotype per replicate. At 21 days after planting, the plants were tagged, labeled with numbers, and inoculated by hand spraying the inoculum onto and below the first trifoliate leaf until runoff. Immediately after inoculation, the inoculated plants were covered with white polyethylene bags for three days to create a high relative humidity as this is one of the requirements for the development of this pathogen [29]. Evaluation for disease severity on tagged plants was done every three days starting at 10 days after inoculation up to 22 days [24].

Data analysis

Data were subjected to the Analysis of Variance (ANOVA) using GenStat version 13 to obtain means of disease severity which were separated using the least significant differences (LSD, P = 0.05). The genotypes were then classified



according to their reactions to the disease infection with those scoring (1-3) classified as resistant, (4-6) as intermediate, and (7-9) susceptible [30]. The Area Under Disease Progress Curve (AUDPC) was computed from ALS disease severity scores collected from the screen house using a midpoint rule method [31].

$$\text{AUDPC} = \sum_{i=1}^{n-1} \left(\frac{y_i + y_{i+1}}{2} \right) (t_{i+1} - t_i)$$
, where “t” is the time in days of each evaluation, “y” is the disease percentage representing the infected foliage at each evaluation, and n is the number of evaluations.

RESULTS AND DISCUSSION

The phenotypic response of common beans to ALS under field conditions

There was significant variability in the severity of ALS on both leaves ($P = 0.002$) and pods ($P = 0.0004$) under the natural field conditions. Much as these genotypes expressed resistant or intermediate reactions, their disease scores were in ranges that indicated development and presence of lesions in that none was immune. The disease severity scores of ALS on leaves ranged from 2.0 - 5.0 with a mean of 3.2, and the scores of ALS on pods ranged from 2.0 - 4.0 with a mean of 2.9. Sixteen genotypes expressed resistant response on both leaves and pods, four genotypes expressed resistant response on leaves and moderate on pods while six expressed resistant responses on pods but moderate on the leaves. None of the genotypes was susceptible on either leaves or pods (Table 3). Such an observation was also observed by Correa-Victoria and Pastor-Corrales [32], who reported that many common bean genotypes which had resistance to ALS in several locations in Latin America and Africa were characterized by at least small disease lesions without complete immunity. Pastor-Corrales and Jara [33,34] also reported no immunity even in the resistant checks. The resistant check (Mexico54) expressed a resistant response on both leaves and pods whereas both susceptible check (CAL96) expressed resistant and moderate resistant response on leaves and pods, respectively. Generally, higher severity scores were noted on leaves compared to pods since different genes control response in leaves and pods and moreover, reaction on the former is more influenced by the environment [35].

There was significant difference between seasons, and the interaction between the seasons and genotypes for yield (Table 2). There was also a significant variation ($P < 0.001$) in yield among the genotypes. Though rainfall precipitation amounts were not recorded in this study, comparably, the yield for 2014a among all genotypes was higher than yield obtained in 2012b suggesting that season differences could have had an influence on yield. A high yield was from genotype



G162667 FPF (1134 kg/ha) and genotypes G19115 (2194 Kg/ha) for seasons 2012b and 2014a, respectively and the low yielders genotype AFR735 (72.9 kg/ha) and AFR702 (166 kg/ha) for seasons 2012b and 2014a, respectively. The susceptible control, CAL96, for ALS produced higher seed yield (675 kg/ha) than the resistant check, Mexico54 (596 kg/ha) indicating that other factors for instance disease complexes and variety genetic potential other than ALS infection alone could have had an influence on the yield (Table 3). Additionally, much as CAL96 is an ALS susceptible line, it is known to be among the high yielding lines and also a check for high yields among other varieties when provided with favorable conditions.

Phenotypic response of common beans to *P. griseola* races under screen house conditions

The variability between the genotypes was highly significant ($P < 0.001$) for disease severity caused by the three *P. griseola* races, 1:6, 21:39 and 61:63 (Table 3). Variable reaction of the genotypes was observed in that a genotype could exhibit a resistant reaction to one or two of the races and an intermediate or susceptible response to the other(s). Twenty-five genotypes (93%) expressed resistance to race 21:39 with a severity score of 2.0-3.0. The checks Mexico54, CAL96, and MCM5001 had scores of 2, 4 and 9 indicating resistance, moderate resistance and susceptibility, respectively. Nine (33.3%), 13 (48%) and 5 (19%) genotypes, respectively, exhibited resistant, moderate and susceptible response to the *P. griseola* race 1:6. The control genotypes Mexico54, CAL96 and MCM5001 expressed resistant, susceptible and moderate response to this race, respectively (Table 3). Four (15%), 10 (37%) and 13 (48%) genotypes expressed resistant, moderately resistant, susceptible to race 61:63, respectively (Figure 2). Susceptible controls, CAL96 and MCM5001, expressed a moderate reaction to this race whereas the resistant control, Mexico54 was found to be resistant to race 61:63. CAL 96, a susceptible check against Angular Leaf spot was resistant to 21:39 but highly susceptible to 1:6 and Vice-versa for MCM5001. This kind of response could have been caused by gene pool differences (Mesoamerican vs Andean) given the fact that 21:39 is more pathogenic on genotypes of Mesoamerican origin whereas 1:6 is more pathogenic on the genotypes of Andean origin [18].

The analysis of variance for AUDPC among the genotypes indicated that the genotypes had significant differences in the AUDPC values ($P < 0.001$) for the *P. griseola* races. The AUDPC values were in a range of 22–81, 14–42 and 24–68 for 1:6, 21:39 and 61:63, respectively. Mexico54, a resistant control expressed the lowest AUDPC value across all three races. Six genotypes (AFR702, AFR703,

AND279, G148, G18842, and G6727) expressed a resistance (ALS score 1-3) to intermediate reaction (ALS score 4 - 6) across the three ALS races (Table 3). Resistance among these lines could depend on the presence of the resistance genes available such as the *Phg*-2, *phg*-5.

Generally, higher severity scores for ALS were recorded in the screen house compared to the field, a scenario similar to that noted by Mulumba and Nankya [34]. This was probably because the field was under natural infection with pathogen pressure differences. Differences in pathogenicity of *P. griseola* population in the field and the races used in the screen house could have also had an effect [33]. Additionally, the difference could have also been due to the different stages at which the genotypes were evaluated, for instance the field evaluation was at pod formation whereas it was at V3 growth stage in the screen house. Generally, the differential reaction of the genotypes in the field and screen house is attributed to the genetic diversity in that some genotypes have resistance genes which may be absent in others. For instance, Mexico54 has *Phg* – 2 loci and G 5686 a *Phg* – 4 loci that have been proved to be responsible for the resistant reaction [25]. The BALSIT genotypes were evaluated and found to be resistant to ALS isolates from Colombia but with intermediate reaction to races in Zaire and Rwanda [36]. Similarly, an Andean bean genotype CAL143, which has gene *Phg* – 5 was resistant to all the races in Malawi, Rwanda and Democratic Republic of Congo (DRC), intermediate to some Ugandan races but susceptible to race 63:21 in Uganda [21]. Genotype G15396A was immune to Colombian races: 1:55 and 63:15 whereas it was susceptible to the Ugandan isolate 63:21. G6727 which expressed resistance to the three isolates in this study was reported resistant to 63:21 and susceptible to 15:39 in earlier studies [33]. As noted in the previous studies in Uganda, Democratic Republic of Congo (DRC), Kenya and Tanzania [11, 34, 37, 38], the ALS resistant check, Mexico54 of Meso-American origin-maintained resistance to all three races indicating the usefulness of the *Phg*-2 gene to current races. Despite its resistance levels, Mexico54 is less preferred as a parent in breeding procedures against ALS because of the intensive backcross program that would be needed to restore the necessary pod quality and determinacy in crosses in which it is involved. Moreover, the most available sources of ALS resistance are small-seeded, an attribute not preferred by many farmers since most African bean growers grow Andean beans [19, 39]. Sources of ALS resistance of Andean origin are limited, for instance CAL143 is one of the large-seeded sources of ALS resistance but was found to be susceptible in Uganda. However, some genotypes were resistant both in the field and to all the races used in the screen house indicating broad resistance in them.

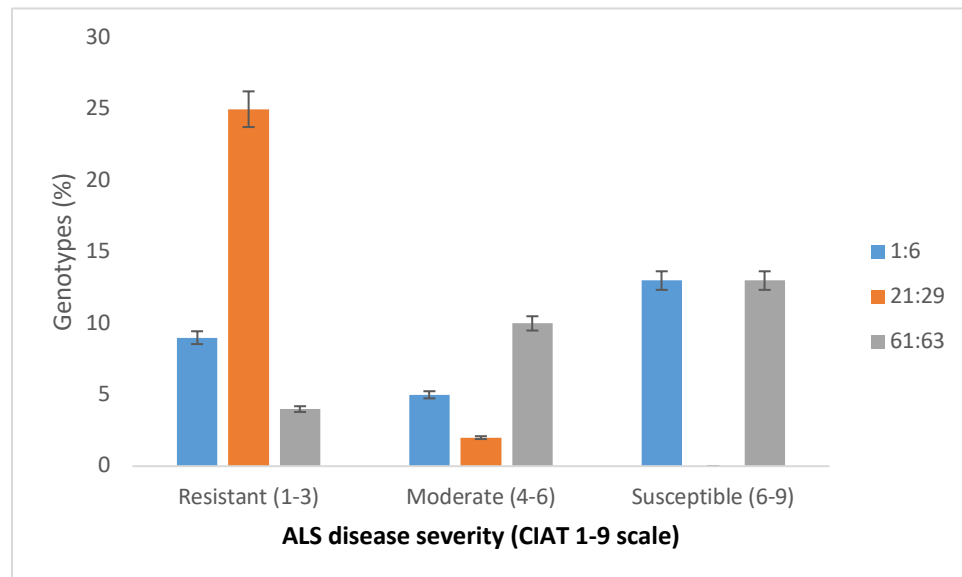


Figure 2: Response of some of the selected genotypes from the BALSIT nursery to the three *P.griseola* races in the screen house at CIAT-Kawanda, Uganda

CONCLUSION, AND RECOMMENDATIONS FOR DEVELOPMENT

The study identified two large-seeded genotypes (AFR703 and AND279) and four small-seeded bush bean genotypes of different seed colors namely: AFR702, G18842, G148, and G6727 that could be used to improve resistance within market classes. These genotypes could also be included in farmers' varietal mixtures to manage the disease since adding high yielding ALS resistant varieties (25%) to local farmer bean mixtures can reduce ALS incidence and result in yields equivalent to those attained from a high yielding resistant variety grown singly. The identified lines are potential sources of resistance that can be pyramided with other resistant lines, a strategy for obtaining more durable resistance. Characterization of genes responsible for resistance in these genotypes is recommended.

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Table 1: Description of selected common bean genotypes from the BALSIT nursery evaluated for resistance to the natural field inoculum and the three *Pseudocercospora griseola* races in Uganda

| Common bean genotype | Primary color | Brilliance | 100 seed weight (grams) | Size |
|----------------------|---------------|------------|-------------------------|--------|
| A791 | Red | Shiny | 37.3 | Medium |
| AFR702 | Red | Shiny | 34.8 | Medium |
| AFR703 | Red | Semi-shiny | 42.2 | Large |
| AFR735 | Pink | Shiny | 20.4 | Small |
| AND279 | Red | Shiny | 40.7 | Large |
| BAT496 AND | Cream | Dull | 24.5 | Small |
| BM12722-127VEF 2000 | Cream | Dull | 21.4 | Small |
| BM12722-132VEF 2000 | Red | Semi-shiny | 50.0 | Large |
| BM12722-77 VEF 2000 | Red | Dull | 42.3 | Large |
| CNF5558 | Brown | Dull | 23.5 | Small |
| DFA70 | Brown | Dull | 23.1 | Small |
| G148 | Red | Shiny | 18.9 | Small |
| G4691 | Red | Shiny | 21.6 | Small |
| G5207 | Red | Shiny | 19.7 | Small |
| G6727 | Red | Shiny | 20.0 | Small |
| G7004 | Pink | Shiny | 20.4 | Small |
| G7005 | Red | Shiny | 30.9 | Medium |
| G7874 | Brown | Dull | 19.4 | Small |
| G8152 | Pink | Dull | 38.3 | Medium |
| G9282 | Black | Dull | 21.5 | Small |
| G11405 | Red | Shiny | 43.7 | Large |
| G162667 FPF | Black | Dull | 25.0 | Small |
| G18842 | Brown | Shiny | 34.6 | Medium |
| G18970 | Red | Shiny | 34.4 | Medium |
| G19115 | Red | Shiny | 24.4 | Small |
| G19833 | Purple | Shiny | 21.4 | Small |
| G20523 | Cream | Dull | 23.9 | Small |
| Controls | | | | |
| CAL96 | Red mottled | Shiny | 46.7 | Large |
| Mexico54 | Grey | Dull | - | Small |
| MCM5001 | Cream mottled | Dull | 18.1 | Small |

Small size (< 25g/100 seeds), Medium (25-40g/100 seeds), and big size (>40g/100 seeds)

Table 2: Mean squares of the analysis of variance of the disease severity of the selected BALSIT nursery and control genotypes in reaction to ALS under field and screen house conditions

| SOV | DF | ALS Severity | | | | | AUDPC Values | | | YLDHA |
|----------------|-----|--------------|-------|---------|--------|--------|--------------|----------|----------|--------------|
| | | ALSL | ALSP | 1:6 | 21:39 | 61:63 | 1:6 | 21:39 | 61:63 | |
| Season (S) | 1 | 58.2 | 0.9 | | | | | | | 19499227.0** |
| Genotype (G) | 29 | 1.4** | 1.1** | 16.5*** | 3.9*** | 7.4*** | 871.3*** | 109.4*** | 779.2*** | 348979.4* |
| G.S | 29 | 0.5 | 0.3 | | | | | | | 183526.0*** |
| Residual | 68 | 0.4 | 0.4 | 0.9 | 0.13 | 0.9 | 56 | 8 | 59.8 | 37118.8 |
| Total | 127 | 1.1 | 0.5 | 8.7 | 2.1 | 6 | 447.8 | 57.7 | 296.4 | 290725 |
| SED (P = 0.05) | | 0.4 | 0.4 | 0.4 | 0.2 | 0.8 | 7.6 | 2.8 | 6.3 | 136.2 |
| CV (%) | | 19.4 | 22.4 | 14.8 | 10.3 | 8.5 | 16 | 11 | 7.6 | 29.1 |

SOV = source of variation, G.S = Genotype. Season, DF = degrees of freedom, ALSL = Angular leaf spot severity on leaves, ALSP = Angular leaf spot symptoms on pods, YLDHA = Clean yield in Kg/ha, * = significant at $P \leq 0.05$, ** = significant at $P \leq 0.01$

Table 3: Angular leaf spot severity for the BALSIT nursery common bean genotypes evaluated CIAT – Kawanda, Uganda under field and screen house conditions

| Genotype | ALSF | | | ALSP | | | YLDHA | | Severity | | | AUDPC | | |
|----------------------|-------|-------|------|-------|-------|------|-------|-------|----------|-------|-------|-------|-------|-------|
| | 2012b | 2014a | Mean | 2012b | 2014a | Mean | 2012b | 2014a | 1:6 | 21:39 | 61:63 | 1:6 | 21:39 | 61:63 |
| A791 | 2 | 5 | 3 | 4 | 4 | 4 | 174 | 880 | 9 | 2 | 4 | 63 | 23 | 25 |
| AFR702 | 3 | 4 | 3 | 3 | 2 | 3 | 81 | 166 | 4 | 2 | 3 | 38 | 23 | 28 |
| AFR703 | 3 | 5 | 4 | 3 | 3 | 3 | 132 | 179 | 3 | 2 | 2 | 35 | 26 | 24 |
| AFR735 | 3 | 3 | 3 | 4 | 2 | 3 | 73 | 965 | 8 | 2 | 7 | 81 | 23 | 41 |
| AND279 | 3 | 5 | 4 | 4 | 4 | 4 | 160 | 941 | 4 | 2 | 4 | 40 | 22 | 24 |
| BAT496 AND | 3 | 3 | 3 | 3 | 3 | 3 | 208 | 1265 | 4 | 2 | 8 | 36 | 22 | 57 |
| BM12722-132- VEF2000 | 3 | 4 | 3 | 4 | 3 | 4 | 82 | 1429 | 9 | 2 | 5 | 74 | 25 | 26 |
| BM-12722-127 VEF2000 | 3 | 5 | 4 | 3 | 3 | 3 | 178 | 1494 | 9 | 2 | 2 | 72 | 25 | 33 |
| BM-12722-77 | 3 | 5 | 4 | 3 | 3 | 3 | 151 | 842 | 6 | 2 | 2 | 55 | 24 | 24 |
| CNF5558 | 2 | 3 | 2 | 2 | 2 | 2 | 141 | 1358 | 2 | 2 | 8 | 23 | 22 | 59 |
| DFA70 | 2 | 3 | 2 | 3 | 3 | 3 | 373 | 991 | 3 | 2 | 8 | 34 | 25 | 68 |



| | | | | | | | | | | | | | | |
|------------|---|---|---|---|---|---|------|------|---|---|---|----|------|------|
| G11405 | 3 | 4 | 3 | 3 | 3 | 3 | 93 | 472 | 9 | 6 | 7 | 75 | 42 | 47 |
| G148 | 3 | 5 | 4 | 4 | 3 | 3 | 142 | 625 | 2 | 2 | 5 | 26 | 24 | 38 |
| G162667FPF | 2 | 2 | 2 | 2 | 2 | 2 | 1134 | - | 9 | 2 | 6 | 65 | 22 | 34 |
| G18842 | 5 | 5 | 5 | 3 | 2 | 2 | 106 | 346 | 3 | 2 | 4 | 30 | 24 | 31 |
| G18970 | 3 | 4 | 3 | 3 | 3 | 3 | - | 922 | 7 | 4 | 8 | 28 | 25.8 | 35.3 |
| G19115 | 2 | 5 | 3 | 2 | 3 | 3 | 177 | 2194 | 7 | 2 | 7 | 62 | 24 | 53 |
| G19833 | 2 | 3 | 2 | 3 | 3 | 3 | 258 | 1899 | 9 | 2 | 8 | 64 | 26 | 67 |
| G20523 | 2 | 3 | 3 | 3 | 2 | 2 | 124 | 1823 | 2 | 2 | 7 | 24 | 22 | 45 |
| G4691 | 2 | 5 | 3 | 3 | 4 | 3 | 147 | 887 | 5 | 2 | 8 | 46 | 28 | 57 |
| G5207 | 2 | 3 | 2 | 2 | 2 | 2 | 127 | 583 | 2 | 2 | 7 | 22 | 25 | 49 |
| G6727 | 3 | 5 | 4 | 3 | 4 | 3 | 230 | 893 | 2 | 2 | 4 | 25 | 23 | 28 |
| G7004 | 3 | 3 | 3 | 3 | 3 | 3 | 210 | 486 | 2 | 2 | 7 | 22 | 22 | 54 |
| G7005 | 3 | 4 | 3 | 3 | 3 | 3 | 235 | 535 | 8 | 2 | 7 | 63 | 24 | 42 |
| G7874 | 3 | 3 | 3 | 4 | 4 | 4 | 140 | 1114 | 7 | 3 | 5 | 52 | 32 | 32 |
| G8152 | 3 | 4 | 3 | 3 | 3 | 3 | 154 | 395 | 7 | 2 | 6 | 56 | 27 | 43 |



| | | | | | | | | | | | | | | |
|----------------------|---|---|---|---|---|---|-----|------|---|---|---|----|----|----|
| G9282 | 2 | 5 | 4 | 2 | 3 | 3 | 102 | 777 | 9 | 2 | 4 | 78 | 22 | 28 |
| Controls | | | | | | | | | | | | | | |
| CAL96 (susceptible) | 3 | 4 | 3 | 4 | 4 | 4 | 106 | 212 | 9 | 4 | 5 | 84 | 26 | 29 |
| MCM5001(susceptible) | 2 | 2 | 2 | 4 | 2 | 3 | 85 | - | 3 | 9 | 6 | 39 | 57 | 30 |
| Mexico54 (resistant) | 2 | 4 | 3 | 4 | 4 | 4 | 676 | - | 2 | 2 | 3 | 13 | 14 | 26 |
| Means | 2 | 3 | 3 | 2 | 2 | 2 | 634 | 1000 | 5 | 2 | 6 | 48 | 25 | 40 |

ALSL = Angular leaf spot symptoms on leaves, ALSP = Disease severity scale of 1-9 was used; (1-3) = Resistant; (4-6) = intermediate and (7-9) = susceptible, YLDHA= Clean yield in Kg/ha



REFERENCES

1. **Wortmann CS** Atlas of common bean (*Phaseolus vulgaris* L.) production in Africa: CIAT; 1998.
2. **Broughton WJ, Hernandez G, Blair M, Beebe S, Gepts P and J Vanderleyden** Beans (*Phaseolus spp.*)—model food legumes. Plant and soil. 2003; 252(1): 55-128.
3. **Romero-Arenas O, Damián-Huato M, Rivera-Tapia J, Báez-Simón A, Huerta-Lara M and E Cabrera-Huerta** The nutritional value of beans (*Phaseolus vulgaris* L.) and its importance for feeding of rural communities in Puebla, Mexico. International Research Journal of Biological Sciences. 2013; 2(8): 59-65.
4. **FAOSTAT**. 2017 [Available from: <http://www.fao.org/faostat/en/#data/QC>, Accessed 16 September, 2019.
5. **UBOS**. Uganda National Bureau of Statistics-Statistical Abstract 2018. Available from: https://www.ubos.org/wpcontent/uploads/publications/05_2019STATISTICAL_ABSTRACT_2018.pdf Accessed 25th October, 2019.
6. **Beebe SE, Rao IM, Mukankusi CM and RA Buruchara** Improving resource use efficiency and reducing risk of common bean production in Africa, Latin America, and the Caribbean. International Center for Tropical Agriculture; 2012.
7. **Ddamulira G, Mukankusi C, Ochwo-Ssemakula M, Edema R, Sseruwagi P and P Gepts** Distribution and Variability of *Pseudocercospora griseola* in Uganda. *Journal of Agricultural Science*. 2014; **6(6)**: 16.
8. **Alladassi B, Nkalubo S, Mukankusi C, Kayaga H, Gibson P and R Edema** Identification of common bean genotypes with dual leaf and pod resistance to common bacterial blight disease in Uganda. *African Crop Science Journal*. 2018; **26(1)**: 63-77.
9. **Paparu P, Katafiire M, Mcharo M and M Ugen** Evaluation of fungicide application rates, spray schedules and alternative management options for rust and angular leaf spot of snap beans in Uganda. *International Journal of Pest Management*. 2014; **60(1)**: 82-89.



10. **Mongi RJ** Breeding for resistance against angular leaf spot disease of common bean in the Southern Highlands of Tanzania 2016.
11. **Kijana R, Abang M, Edema R, Mukankusi C and R Buruchara** Prevalence of angular leaf spot disease and sources of resistance in common bean in eastern Democratic Republic of Congo. *African Crop Science Journal*. 2017; **25(1)**:109-22.
12. **Mwang'ombe AW, Wagara I, Kimenju J and R Buruchara** Occurrence and Severity of Angular leaf spot of common bean in Kenya as influenced by geographical location, altitude and agroecological zones. *Plant Pathology Journal*. 2007; **6(3)**: 235-241.
13. **Sengooba Tand J Mukiibi** Studies on inoculum sources of angular leaf spot of beans caused by *Phaeoisariopsis griseola* in Uganda. *International Journal of Pest Management*. 1986; **32(4)**: 288-291.
14. **Crous PW, Liebenberg MM, Braun U and JZ Groenewald** Re-evaluating the taxonomic status of *Phaeoisariopsis griseola*, the causal agent of angular leaf spot of bean. *Studies in Mycology*. 2006; **55**: 163-173.
15. **Mahuku GS, Henríquez MA, Munõz J and RA Buruchara** Molecular markers dispute the existence of the Afro-Andean group of the bean angular leaf spot pathogen, *Phaeoisariopsis griseola*. *Phytopathology*. 2002; **92(6)**: 580-589.
16. **Guzmán P, Gilbertson R, Nodari R, Johnson W, Temple S and D Mandala** Characterization of variability in the fungus *Phaeoisariopsis griseola* suggests coevolution with the common bean (*Phaseolus vulgaris*). *Phytopathology*. 1995; **85(5)**: 600-607.
17. **Willocquet L, Allorent D and S Savary** Quantitative analysis of two important epidemiological features of the common bean: *Phaeoisariopsis griseola* pathosystem. *Fitopatologia Brasileira*. 2004; **29(6)**: 676 - 609.
18. **Ddamulira G, Mukankusi C, Ochwo-Ssemakula M, Edema R, Sseruwagi P and P Gepts** Gene pyramiding improved resistance to angular leaf spot in common bean. *Journal of Experimental Agriculture International*. 2015; **9**: 1-12.

19. **Leitich RK, Omayio D, Mukoye B, Mangeni B, Wosula D and W Arinaitwe** Pathogenic variability of angular leaf spot disease of common bean in Western Kenya. *International Journal of Applied Agricultural Sciences*. 2016; **2(6)**: 92 - 98.
20. **Rezene Y, Tesfaye K, Clare M and P Gepts** Pathotypes characterization and virulence diversity of *Pseudocercospora griseola* the causal agent of angular leaf spot disease collected from major common bean (*Phaseolus vulgaris* L.) growing areas of Ethiopia. *J Plant Pathol Microbiol*. 2018; **9(8)**: 445
21. **Aggarwal VD, Pastor-Corrales MA, Chirwa RM and RA Buruchara** Andean beans (*Phaseolus vulgaris* L.) with resistance to the angular leaf spot pathogen (*Phaeoisariopsis griseola*) in southern and eastern Africa. *Euphytica*. 2004; **136**: 201 - 210.
22. **Lemessa F, Sori W and M Wakjira** Association between angular leaf spot (*Phaeoisariopsis griseola* (Sacc.) Ferraris) and common bean (*Phaseolus vulgaris* L.) yield loss at Jimma, Southwestern Ethiopia. *Plant Pathology Journal*. 2011; **10**: 57 - 65.
23. **Olango N, Tusiime G, Mulumba J, Nankya R, Fadda C and D Jarvis** Response of Ugandan common bean varieties to *Pseudocercospora griseola* and Angular leaf spot disease development in varietal mixtures. *International Journal of Pest Management*. 2017; **63(2)**: 119-27.
24. **Keller B, Manzanares C, Jara C, Lobaton JD, Studer B and B Raatz** Fine-mapping of a major QTL controlling angular leaf spot resistance in common bean (*Phaseolus vulgaris* L.). *Theoretical and Applied Genetics*. 2015; **128(5)**: 813-26.
25. **Namayanja A, Buruchara R, Mahuku G, Rubaihayo P, Kimani P and S Mayanja** Inheritance of resistance to angular leaf spot in common bean and validation of the utility of resistance linked markers for marker assisted selection out side the mapping population. *Euphytica*. 2006; **151(3)**: 361-9.
26. **SARTORATO A** Identification of *Phaeoisariopsis griseola* pathotypes from five States in Brazil. *Fitopatologia Brasileira*. 2002; **27(1)**: 78-81.
27. **Nay MM, Souza TL, Raatz B, Mukankusi CM, Gonçalves-Vidigal MC and AF Abreu** A review of angular leaf spot resistance in common bean. *Crop Science*. 2019; **59**: 1376.

28. **Beebe S and MP Corrales** Breeding for disease resistance. Common beans: research for crop improvement. 1991: 561-617.
29. **Wagara I, Mwangómbe A, Kimenju J, Buruchara RA and P Kimani** Reaction of selected common bean genotypes to physiological races of *Phaeoisariopsis griseola* occurring in Kenya. *African Crop Science Journal*. 2011; **19(4)**: 343-55.
30. **Schoonhoven AV and M Pastor-Corrales** Standard system for the evaluation of bean germplasm. 1987.
31. **Campbell CL and LV Madden** Introduction to plant disease epidemiology: John Wiley & Sons.; 1990.
32. **Correa-Victoria F, Pastor-Corrales M and A Saettler** Angular leaf spot. Bean production problems in the tropics. 1989; 59:75.
33. **Pastor-Corrales MA, Jara C and SP Singh** Pathogenic variation in, sources of, and breeding for resistance to *Phaeoisariopsis griseola* causing angular leaf spot in common bean. *Euphytica*. 1998; **103**: 161-71.
34. **Mulumba J, Nankya R, Adokorach J, Kiwuka C, Fadda C and P De Santis** A risk-minimizing argument for traditional crop varietal diversity use to reduce pest and disease damage in agricultural ecosystems of Uganda. *Agriculture, Ecosystems & Environment*. 2012; **157**: 70-86.
35. **Borel JC, Ramalho MAP, Abreu ÂdFB and LGS Maia** Genetic control of the angular leaf spot reaction in common bean leaves and pods. *Scientia Agricola*. 2011; **68(6)**: 661-4.
36. **Smithson JB and P Trutmann** African Bean Pathology Workshop (1, 1987, Kigali, Rwanda). Proceedings. 1992.
37. **Chilagane LA, Tryphone GM, Protas D, Kweka E, Kusolwa PM and S Nchimbi-Msolla** Incorporation of resistance to angular leaf spot and bean common mosaic necrosis virus diseases into adapted common bean (*Phaseolus vulgaris L.*) genotype in Tanzania. *African Journal of Biotechnology*. 2013; **12(27)**: 4343-4350.
38. **Kimno S, Kiplagat O, Arunga E and E Chepkoech** Evaluation of selected French bean (*Phaseolus vulgaris L.*) genotypes for resistance to angular leaf spot (*Pseudocercospora griseola*) in Western Kenya. *Journal of Experimental Agriculture International*. 2016; **13(4)**: 1-6.



39. **Liebenberg M and Z Pretorius** A review of angular leaf spot of common bean (*Phaseolus vulgaris* L). *African Plant Protection*. 1997; **3(2)**: 81-106.
40. **Trutmann P and MM Pyndji** Partial replacement of local common bean mixtures by high yielding angular leaf spot resistant varieties to conserve local genetic diversity while increasing yield. *Annals of Applied Biology*. 1994; **125(1)**: 45-52.

