Physiological Races of *Colletotrichum lindemuthianum*, the cause of Bean Anthracnose in Major Bean Growing Regions of Southern and Central Ethiopia

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Abstract

Background: Bean anthracnose (*Colletotrichum lindemuthianum* (Sacc. & Magnus) Briosi & Cavara) is one of the major diseases of common bean (*Phaseolus vulgaris* L.) in Ethiopia causing up to a 63% yield loss. However, information regarding the race variability of the pathogen in Ethiopia is limited.

Objectives: The current study was initiated with the objective of characterizing races of *C. lindemuthianum* prevailing in major bean producing areas in southern and Central Ethiopia.

Materials and Methods: Thirty isolates of *C. lindemuthianum*, collected from potential bean growing districts of Damot Gale, Halaba Special, Melkassa, Hawassa Zuria, and Boricha in 2017 were inoculated on to 12 differential cultivars in a greenhouse using a completely randomized design.

Results: The results revealed the presence of 17 physiological races (pathotypes) of the pathogen, of which only three were previously reported from Ethiopia. Race 9 was found to be the most dominant one across the surveyed areas. Four races (2073 from Halaba Special, 2225 from Damot Gale, 2260 from Melkassa, and Hawassa Zuria, and 3047 from Boricha districts) were able to infect the highly resistant differential cultivar G2333, indicating that the Ethiopian *C. lindemuthianum* populations might be composed of highly virulent races. The cultivars Michelite, Mexico 222, and PI 207262 showed the most susceptible reaction to the tested races, while no cultivar was immune to the pathogen races. The results have demonstrated the existence of highly variable isolates of *C. lindemuthianum* that cause bean anthracnose in Ethiopia.

Conclusions: The Ethiopian bean anthracnose pathogen (*Colletotrichum lindemuthianum*) has high variability. The pathogen seems to be widely distributed in all studied bean growing areas with highly virulent as well as less virulent races. It is also suggested that the *C. lindemuthianum* population in Ethiopia could possibly be composed of highly virulent races that can cause much damage even to resistant/tolerant germplasm. The results provides important useful insights into future breeding programs for developing host resistance against bean anthracnose in the country. Identifying the race should continue using techniques that are more advanced and by including additional isolates across different agro-ecological settings in the country.

Keywords: *Colletotrichum lindemuthianum*; Differential cultivars; *Phaseolus vulgaris;* Physiological races; Virulent

1. Introduction

Bean anthracnose caused by *Colletotrichum lindemuthianum* (Sacc. & Magnus) Briosi & Cavara, is one of the most important seed borne diseases of common bean (*Phaseolus vulgaris* L.) in the world (Amin Mohammed, 2013; Amin Mohammed *et al*., 2014; Fitsum Sileshi *et al*., 2014). Depending on the cultivar and environmental conditions, anthracnose infections can drastically reduce crop yields (Perseguini *et al*., 2016). Fernandez *et al.* (2000) reported that infection of susceptible cultivars, like Mexican-142 and Awash-1, under favorable environmental conditions leads to 100% grain yield loss. In Africa, the disease is common in Burundi, Democratic Republic of Congo, Ethiopia, Kenya, Rwanda, Tanzania and Uganda (Batureine, 2009). In Ethiopia, the disease is a major production constraint in potential bean-growing regions (Fitsum Sileshi *et al*., 2014), causing a yield loss of up to 63% (Tesfaye Beshir, 1997).

Colletotrichum lindemuthianum exists in many physiological forms in all bean-growing regions of the world and infects bean genotypes from both bean gene pools, i.e. Andean and Mesoamerican (Mahuku and Riascos, 2004). According to CIAT (1997), the fungus is known to have races that vary across countries, regions, locations and varieties. The highest diversity and variation have been reported from Latin America, which is the center of origin of common beans (Pastor-Corrales *et al*., 1995). East African regions are considered as the secondary center of diversity for common bean, and due to co-evolution of the *C. lindemuthianum*

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pathogen and its host, the regions are expected to have a high variability of *C. lindemuthianum* (Moses *et al.*, 2016). The existence of a high pathogenic variability and emergence of new pathogen races results in continuous breakdown in host resistance (Bigirimana and Hofte, 2001).

Knowledge on race variability of the pathogen population is a prerequisite for developing durable resistance in bean varieties (Batureine, 2009). Co-evolution of the pathogen and its host in the Andean and Mesoamerican gene pools provides a useful means of identifying appropriate sources of resistance, since common bean genotypes originating from one gene pool are more likely to express resistance to pathogenic races than another gene pool (Allen *et al.*, 1998; Pastor-Corrales, 2004). Different race characterization has been carried out in Africa and the rest of the world. For example (Alzate Marin *et al.,* 2000 and Mahuku and Riascos, 2004) identified a total of 50 and 90 races of *Colletotrichum lindemuthianum* pathotypes, respectively, in Brazil between 1994 and 2002 from Andean and Mesoamerican bean varieties. To date about 1590 isolates of *C. lindemuthianum* inoculated on 12 bean differential cultivars have resulted in the identification of 182 races worldwide (Padder *et al.*, 2017).

In Africa, from the 12 isolates collected from the major bean-growing areas in Burundi nine races were identified (Bigirimana *et al.*, 2000). On the other hand, out of the 50 isolates collected from potential bean agro ecological zones of Tanzania, 42 races were identified (Masunga *et al.*, 2020). But in Ethiopia, although some works have been done on *C. lindemuthianum* variability and the race identification, information is still limited and also the work was done long ago. In 1992, race studies were initiated and isolates characterized from different bean-growing regions in the country Bako, Ambo, Ziway, Awassa, Areka, Adami Tulu, Meki, Jimma and Alem Tena and fifteen races of *C. lindemuthianum* were identified (Tesfaye Beshir, 2005). Studies of pathogenic variation among 20 isolates of the anthracnose pathogen led to the identification of at least nine races (Tesfaye Beshir, 1995). Tesfaye Beshir (2003) identified eight races of *C. lindemuthianum* and compared them to six races brought from Southern Africa.

However, for breeding anthracnose resistant varieties, studying and identifying the available races of *C. lindemuthianum* on major bean growing areas of the country, which is helpful in planning suitable breeding strategies for varieties with durable resistance over time and space is very important. Therefore, this research was aimed at studying variability in *C. lindemuthianum* and identifying races of the pathogen occurring in major bean growing regions in southern and central Ethiopia.

2. Materials and Methods

2.1. Disease Survey

A field survey was conducted in 2017 main cropping season to isolate *Colletotrichum lindemuthianum* in selected districts of Oromia National Regional State (central) and Southern Nations, Nationalities, and People's Region (SNNPR) (southern) Ethiopia. Based on production potential and importance of bean anthracnose, five representative districts, namely Damot Gale, Halaba Special, Melkassa, Hawassa Zuria, and Boricha were selected for the study (Figure 1). The surveyed areas have elevations ranging between 1436 and 1958 meters above sea level.

During the survey, two to three farmers' fields were randomly selected from each district. In each sample field, five quadrats (each 1 m^2) were sampled and disease incidence and severity were assessed for every quadrat by moving diagonally across each field from one end to the other in an 'X' pattern. From each quadrat (plot), the number of plants assessed and number of plants with anthracnose symptom were counted. Disease incidence was recorded as the percentage of plants showing anthracnose symptoms in each quadrat, and the averages of the five quadrats were calculated for each field.

$$\text{Disease incidence (\%)} = \frac{\text{Number of anthracnose infected plant(s)per quadrat}}{\text{Total number of plants assessed per quadrat}} \text{ x } 100$$

Severity of bean anthracnose was recorded on the symptom of the disease on the leaves based on CIAT (1987) standard system for evaluation of bean anthracnose scoring scales (Table 1).

Table 1. Symptom evaluation of common bean anthracnose severity scale according to CIAT (1987).

Disease scale	Description of common bean plant parts affected
1	No visible disease symptom
3	Presence of very few and small lesions, mostly on the primary vein of the leaf's lower side or on the pod, that covers approximately 1% of the surface area
5	Presence of several small lesions on the petiole or on the primary and secondary veins of the leaf's lower side. On the pods, small (less than 2 mm diameter) round lesions, with or without reduced sporulation, cover approximately 5% of the pod surface areas
7	Presence of numerous enlarged lesions on the lower side of the leaf. Necrotic lesions can also be observed on the upper leaf surface and on petioles. On the pods the presence of medium sized (>2 mm in diameter) lesions are evident but also some small and larger lesions generally with sporulation and that cover approximately 10% of pod surface area may be found
9	Severe necrosis on 25% or more of the plant tissue is evident as a result of lesions on the leaves, petioles, stem, branches, and even on the growing point which often results in death of the plant tissues. The presence of numerous, large, sporulating and sunken cankers can result in pod malformation, low seed number, and death of the pod

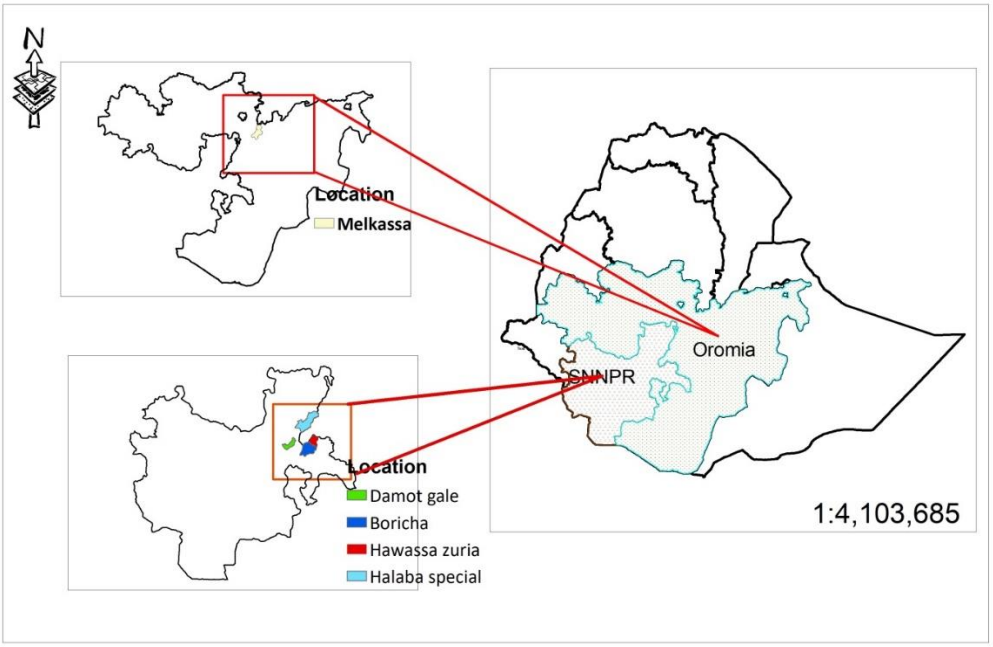

Figure 1. Map showing survey districts for bean anthracnose in Oromia (central) and SNNP region (southern) Ethiopia.

2.2. Sample Collection

Sample collection was conducted at flowering and grain filling stage. In each location, samples depicting typical bean anthracnose symptoms were randomly considered, and from each identified representative plants, three leaves were collected. The leaves collected were immediately wrapped up in paper towels to blot excess moisture and placed in paper bags after labeling with necessary information in pencil. Finally, specimens were kept in a refrigerator at 4 ^{o}C until processing for analyzing the races of the pathogen.

2.3. Isolation of the Pathogen

Isolation of the pathogen was conducted at Melkassa Agricultural Research Center (MARC) Plant Pathology Laboratory. Leaf samples were washed under running tap water and 2–5 mm long leaf pieces or patches were cut using a pair of scissors that was surface-sterilized with 2.5% sodium hypochlorite (NaOCl) solution for 2–3 minutes and rinsed three times with sterile distilled water. Since collected samples could possibly carry more than one strain of the pathogen or even more pathogens,

efforts were made to spatially resolute a spot with the typical anthracnose symptom from a single leaf, and then pieces were cut out to maintain the genetic purity of *C. lindemuthianum* isolates. Then, the pieces were placed on sterilized paper towel for 5–10 minutes; five such pieces were aseptically transferred into a 9 cm Petri Dish with potato dextrose agar (PDA). The cultures were incubated for 10 days at 20 °C under continuous fluorescent light. Sub-culturing was done by taking a 3 mm mycilial plug from the PDA, which was visually free from any contamination, and transferring it into new Petri Dishes containing fresh PDA. Then continuous sub-culturing was made until pure cultures with no contamination were obtained (Bean pathogens practical guide for laboratory). Finally, 30 isolates were purified and obtained from the survey areas for race identification.

2.4. Inoculum Preparation for Race Identification

Inoculum was prepared following the methods described by Batureine (2009). Two-week-old pure cultures were flooded with 50 ml of sterile distilled water and the spores scraped-off using a fine brush. Flooding was repeated three times, each time using fresh sterile distilled water to get most of the conidia from the culture. Then the suspension was poured into a beaker and mixed thoroughly. Spore suspensions from all cultures of the same isolate were mixed and passed through double layer cheesecloth to remove fragments of mycelia and culture medium. The standard concentration of the spore suspension for each obtained isolate was adjusted to $1.2x10^6$ spores per ml of sterile distilled water using hemocytometer (Batureine, 2009). One drop of Tween 20 was added per 100 ml spore suspension as surfactant and mixed thoroughly before inoculation.

2.5. Planting Common Bean Differentials

Differentials are sets of plant cultivars used to define race/pathotypes of pathogens based on known susceptible and resistant reactions. Common bean seeds of 12 differential cultivars (Table 2) obtained from the International Center for Tropical Agriculture (CIAT) were surface-sterilized with 0.1% NaOCl solution for three minutes, washed or rinsed thoroughly with sterile distilled water and dried at room temperature (25 $\pm$ 2 ^oC). Then the seeds were sown in plastic pot filled with 3 kg mix of sun-dried sterile top soil, manure and sand in 2:1:1 ratio, respectively, and pots placed in greenhouse. Five seeds were sown per pot in completely randomized design, and each plant was considered as a replicate for each common bean differential cultivars and for each isolate of the pathogen.

2.6. Inoculation

Fourteen-day-old common bean seedlings were used for inoculation. All the 12 differential cultivars were inoculated separately with the prepared separate isolate suspension using a hand sprayer on both the abaxial and adaxial surfaces of the leaves until suspension runoff at a time. A control (plants inoculated with sterile distilled water) was included for each set of the differentials inoculated with an isolate. In order to maintain the relative humidity of approximately 95% for the infection to take place, each inoculated plants were covered with transparent polythene sheets for five days after inoculation. After removal of polythene sheet, pots were kept in the greenhouse and maintained at the temperatures of 18–33 ^oC and relative humidity ranging from 72 to 95%.

2.7. Race Analysis

The reaction [susceptible reaction (+) and resistant reaction (-)] of each common bean differential cultivar to each isolate was assessed 15 days after inoculation, i,e. 30 days after planting. For the purpose of consistency, only the primary leaves of each plant were evaluated. After evaluation of all differential cultivars, race identification was done as the sum of binary numbers of all differential cultivars showing susceptible reaction to the particular isolate. A binary number is equal to 2^n, where n is equivalent to the place of the cultivar within the differential series order as presented in Table 2. The sum of all binary numbers of cultivars with susceptible reactions (i.e., $y = 2^n + 2^{(n+1)} + 2^{(n+2)} + \ldots\ldots + 2^{(n+11)}$; where $n = 0$) gives a specific race number or name (y) (Kelly and Vallejo, 2004).

Table 2. Standard differential cultivars of common bean used to characterize *Colletotrichum lindemuthianum,* their binary codes, resistance genes, growth habit and gene pool.

Differential cultivar	Seed type	Notation	Binary code	Resistance gene	Gene pool	Growth habit
Michelite	S	0	1	Co-11	MA	II
MDRK	L	1	2	Co-1	A	I
Perry Marrow	L	2	4	Co-1^3	A	II
Cornell 49-242	S	3	8	Co-2	MA	II
Widusa	L	4	16	Co-1^5	A	I
Kaboon	L	5	32	Co-1^2	A	II
Mexico 222	S	6	64	Co-3	MA	I
PI 207262	S	7	128	Co-4^3, Co-9	MA	III
TO	S	8	256	Co-4	MA	I
TU	S	9	512	Co-5	MA	III
AB 136	S	10	1024	Co-6, Co-8	MA	IV
G2333	S	11	2048	Co-4^2, Co-5^2, Co-7	MA	IV

Note: *S = Small seeded; L = Large seeded; MA = Middle American; A = Andean*; *I = Determinate; II = Indeterminate bush; III = Indeterminate bush with weak main stem and prostrate branches; and IV = Indeterminate climbing habit* (Awale *et al*., 2007).

3. Results and Discussions

3.1. Disease Survey

Bean anthracnose incidence in the study areas ranged from 33 to 67% (Figure 2). The highest anthracnose incidence (67%) was recorded for Hawassa Zuria and Boricha districts, followed by Damot Gale (59%), while the lowest (33%) was recorded for Melkassa. The results are in contrast to the finding of Habtu Assefa *et al.* (1996) who reported high level of bean anthracnose in the Rift Valley region including Melkassa and low level of bean anthracnose in Sidama areas where Hawassa Zuria is located. The change in climate and the change in the type of varieties produced in the area may have led to such deviations in bean anthracnose incidence.

The mean anthracnose severity scores across the surveyed areas ranged from 2.5 to 6.0. The highest (6.0) severity was recorded for Damot Gale and Boricha districts and the lowest (2.5) was recorded for Melkassa. These results are not in accord with the result of Tesfaye Beshir (2005) who reported higher anthracnose severity in the western zone and Rift Valley areas of the country, suggesting a possible shift in bean anthracnose intensity across the country. Seed born nature of the disease (Genchev *et al.*, 2010) and uncontrolled exchange of common bean seeds for planting across the country might lead to such deviations in the incidence and severity of bean anthracnose.

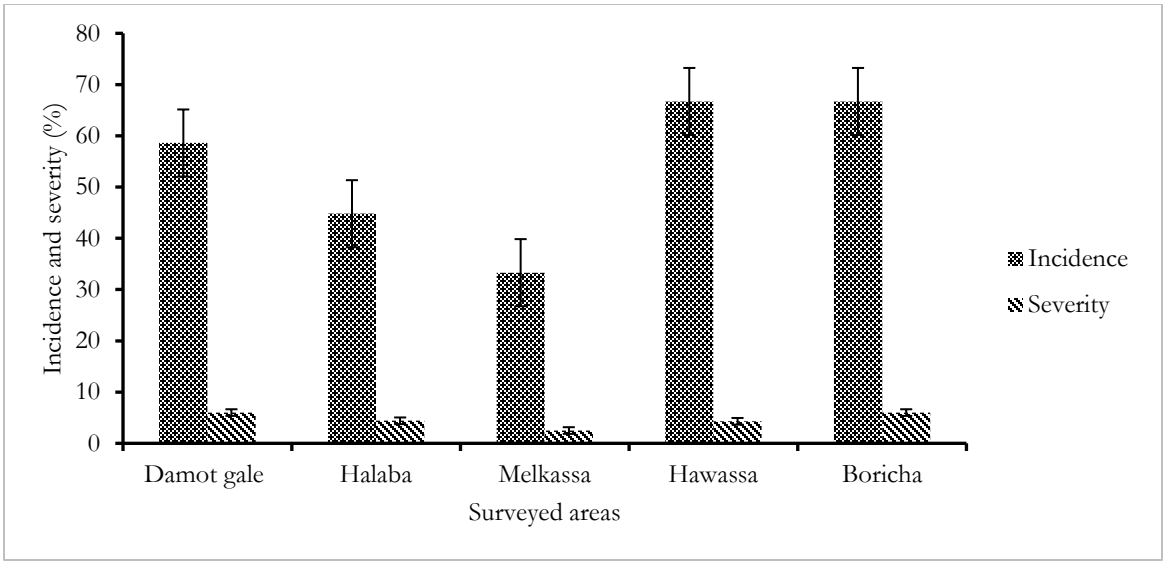

Figure 2. Incidence and severity of common bean anthracnose in the surveyed areas, Southern and Central Ethiopia.

3.2. Race Analysis

In the study area, the reactions of bean differential cultivars to the selected isolates varied. Out of the 30 isolates of *C. lindemuthianum* collected, a total of 17 physiological races (pathotypes) were identified based on race designation (Table 4), which was reported as an indicator of the presence of variability of the pathogen (Batureine, 2009). Out of 17 races identified, only three races, races 128, 898 and 1011 were earlier reported from Ziway, Bako and Areka district in Ethiopia, respectively (Tesfaye Beshir, 2005).

Race 9, which was identified from Boricha, Hawassa and Halaba districts, was the most frequently isolated physiological race, followed by the races 272, 1011 and 2260. Similarly, it was also reported to be the most widespread race in Argentina (Ansari *et al.,* 2004). Moreover, the same authors indicated that race 9 was isolated from four different countries. On the other hand, Gonçalves *et al.* (2008) stated that race 73 was the most common and widespread race in Santa Catarina State in Brazil.

Geographically, four (385, 3047, 9 and 587), two (2260 and 272), 12 (272, 321, 9, 1172, 898, 128, 465, 73, 1250, 34, 1011 and 2260), two (2225 and 1011), and two (2073 and 9) races were identified from Boricha, Melkassa, Hawassa, Damot Gale, and Halaba areas, respectively (Table 3). This suggests an abundance of *C. lindemuthianum* race variability in the southern and central bean producing regions in the country. The differences among the pathogen populations in different areas might reflect the differences in the agricultural practices employed and common bean germplasm used, which would affect the selection and adaptation processes in the different agro-ecologies (Batureine, 2009).

Of the 17 C. *lindemuthianum* races identified, four races 2073, 2225, 2260 and 3047 were able to infect the highly resistant differential cultivar G2333. Susceptibility of this cultivar to the isolates of C. *lindemuthianum* has been rarely reported (Mahuku *et al.*, 2002). Thus, the result

strongly suggests that C. *lindemuthianum* populations in Ethiopia could possibly be composed of highly virulent races that may cause much damage even to resistant/tolerant bean cultivars. Beside the differential cultivar G2333, race 3047 (Isolate from Boricha) also successfully infected the differential cultivars TU, TO, PI 207262, Mexico 222, Kaboon, Perry Marrow, MDRK and Michelite. Hence, this C. *lindemuthianum* race is a very aggressive isolate in the area. Furthermore, race 2260 managed to break the resistance of the differential cultivars Perry Marrow, Widusa, Mexico 222 and PI 207262. While race 2225 managed to break the resistance of the differential cultivars Michelite, Widusa, Kaboon and PI 207262, and race 2073 managed to break the resistance of the differential cultivars Michelite, Cornell 49-242 and Widusa (Table 4).

Table 3. The number of isolates and races of *Colletotrichum lindemuthianum* identified in each district using bean anthracnose differentials.

Region (zone)	District	No. of isolates	Identified races
Southern Nations, Nationalities and	Boricha	5	4
People's Region (SNNPR)	Hawassa zuria	19	12
	Damot gale	2	2
	Halaba	2	2
Oromia National Regional State	Melkassa	2	2

None of the differential cultivars was resistant to all of the isolates tested. Nevertheless, the differential cultivar AB 136 was a relatively more resistant cultivar than the other, considering the number of races, only two races, i.e. 1172 and 1250 that infected it. This observation is similar with findings of Sicard *et al.* (1997) and Gonzalez *et al.* (1998) who reported that the differential cultivar AB 136, containing resistance genes Co-6 and Co-8 as resistant to many isolates of *C. lindemuthianum* originating from different parts of the world. The differential cultivars Perry Marrow, G 2333 and TU were susceptible to three, four and four races, respectively, and ranked second and third most resistant cultivars to the pathogen races in the current experiment (Figure 3). A similar result was reported by Cláudia *et al.* (2002), where the cultivars AB 136 and G 2333 were found resistant to races collected from Paraná State of Brazil. Likewise, the differential cultivar TU also reported to be susceptible to four isolates of C. *lindemuthianum* in another study (Ansari *et al.,* 2004).

On the other hand, the differential cultivars Michelite and PI 207262 were found to be the most susceptible differential cultivars showing susceptible reaction to 59% of all *C. lindemuthianum* races, followed by Mexico 222, TO and Widus, which were susceptible to 53%, 41% and 41% of all collected races, respectively (Figure 3). Consistent with the results of this study, Cláudia *et al.* (2002) and Ansari *et al.* (2004) reported that Michelite, Mexico 222, Widusa and Cornell 49242 cultivars were the most susceptible differential cultivars. The differential cultivars Michelite and Mexico 222 showed susceptible reactions to all of the 10 isolates collected from Grosso State, Brazil (Gonçalves *et al.*, 2010). Gonzaz (2016) also reported that, the bean differential cultivar Michelite was susceptible to all C. *lindemuthianum* isolates collected from Arumeru, Karatu, Mbulu and Babati rural areas in Tanzania.

Table 4. Reaction of 12 common bean differential cultivars to *Colletotrichum lindemuthianum* isolates under greenhouse conditions.

Isolates	Common bean differential cultivars												Race name
	Michelite	MDRK	Perry Marrow	Cornell 49242	Widusa	Kaboon	Mexico 222	PI 207262	TO	TU	AB 136	G 2333	
Boricha205	+	-	-	+	-	-	-	-	-	-	-	-	9
Block 2(2)	+	-	-	+	-	-	-	-	-	-	-	-	9
Halaba p-2	+	-	-	+	-	-	-	-	-	-	-	-	9
HRC 318	-	+	-	-	-	+	-	-	-	-	-	-	34
HRC 8	-	+	-	-	-	+	-	-	-	-	-	-	34
HRC 308	-	+	-	-	-	+	-	-	-	-	-	-	34
HRC 9	+	-	-	+	-	-	+	-	-	-	-	-	73
HRC 12	+	-	-	+	-	-	+	-	-	-	-	-	73
HRC 315	-	-	-	-		-	-	+	-	-	-	-	128
Seed Multi	-	-	-	-	+	-	-	-	+	-	-	-	272
Block 1(2)	-	-	-	-	+	-		-	+	-	-	-	272
Block 1(3)	+	-	-	-	-	-	+	-	+	-	-	-	321
Block 1(1)	+	-	-	-	-	-	+	-	+	-	-	-	321
Boricha207(1)	+	-	-	-	-	-	-	+	+	-	-	-	385
Boricha207(3)	+	-	-	-	-	-	-	+	+	-	-	-	385
HRC 314	+	-	-	-	+	-	+	+	+	-	-	-	465
HRC 319	+	-	-		+	-	+	+	+	-	-	-	465
Boricha204	+	+	-	+	-	-	+	-	-	+	-	-	587
HRC 305	-	+	-	-	-	-	-	+	+	+	-	-	898
Wolaita p-7	+	+	-	-	+	+	+	+	+	+	-	-	1011
HRC 6	+	+	-	-	+	+	+	+	+	+	-	-	1011
Block 2(3)	-	-	+	-	+	-	-	+	-	-	+	-	1172
HRC 7	-	+	-	-	-	+	+	+	-	-	+	-	1250
HRC 1	-	+	-	-	-	+	+	+	-	-	+	-	1250
HRC 310	-	+	-	-	-	+	+	+	-	-	+	-	1250
Halaba p-3	+	-	-	+	+	-	-	-	-	-	-	+	2073
Wolaita p-6	+	-	-	-	+	+	-	+	-	-	-	+	2225
Kechachule 2	-	-	+	-	+	-	+	+	-	-	-	+	2260
HRC 2	-	-	+	-	+	-	+	+	-	-	-	+	2260
Boricha207(2)	+	+	+	-	-	+	+	+	+	+	-	+	3047

Note: (+) = *susceptible reaction*, and (-) = *resistant reaction.*

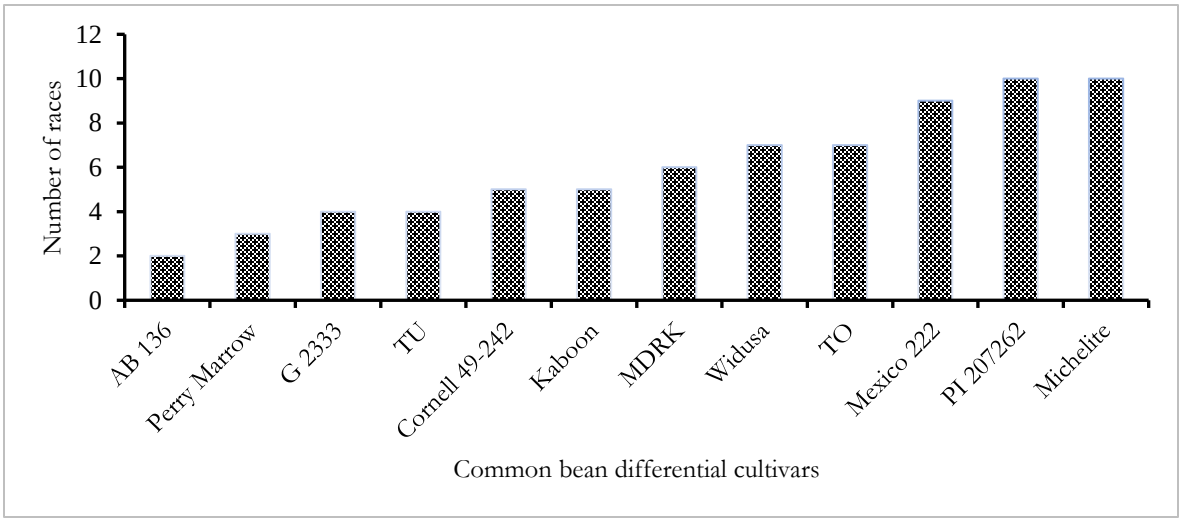

Figure 3. Reaction of 12 common bean differential cultivars to 17 races of *Colletotrichum lindemuthianum* tested under greenhouse conditions.

Of the 17 *C. lindemuthianum* races identified in the current work, race 3047 and race 1011 were the most 'cosmopolitan' races, infecting nine and eight differential cultivars, respectively, whereas race 9, 34 and 128 were 'narrow host ranged', infecting only two cultivars, out of the total 12 differential cultivars (Figure 4).

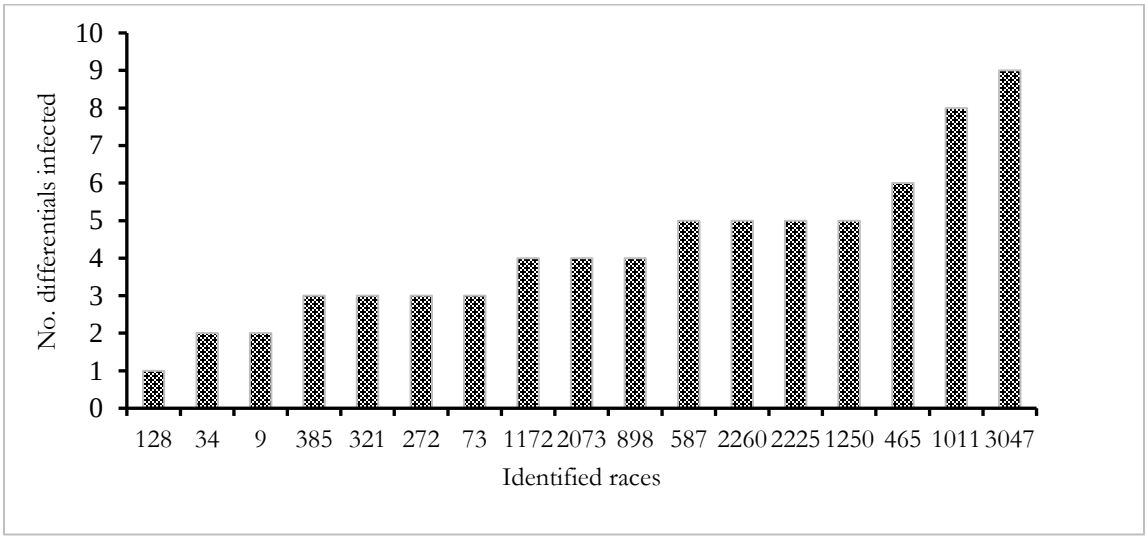

Figure 4. Virulence of races of *Colletotrichum lindemuthianum* collected from south and central Ethiopia as evaluated using common bean differential cultivars under greenhouse conditions.

With reference to bean gene pool, among the 17 identified races, 11 races (272, 465, 587, 898, 1011, 1172, 1250, 2073, 2225, 2260 and 3047) were pathogenic to the common bean differential cultivars having gene pool from both Mesoamerican and Andean origin. It is common to see races infecting differential cultivars having both gene pools as reported by Gonzaz (2016). Race 34 was pathogenic only to the differential cultivars from Andean gene pool, whereas races 9, 73, 128, 321 and 385 were pathogenic only to Mesoamerican gene pool. In line with this result, race 73, which infected only the Mesoamerican origin in this study, was also shown to be pathogenic to the differential cultivars from Mesoamerican origin (Cláudia *et al.*, 2002). Most of the isolates collected from Argentina and Central America were predominantly pathogenic to the Mesoamerican cultivars, while isolates collected from Africa, Europe, the Dominican Republic and South America showed a higher frequency of pathogenicity to the Andean

cultivars than to the Mesoamerican differential cultivars (Ansari *et al*., 2004).

4. Conclusions

The results of the study have demonstrated that the Ethiopian bean anthracnose pathogen (*Colletotrichum lindemuthianum*) has high variability in major bean growing areas of south and central Ethiopia. The pathogen seems to be widely distributed in all studied bean growing areas with highly virulent as well as less virulent races. The results also suggest that the *C. lindemuthianum* population in Ethiopia could possibly be composed of highly virulent races that can cause much damage even to resistant/tolerant germplasm. The differential cultivar AB 136 showed a relative high resistance as it was found to be susceptible only to two races. However, Michelite and PI 207262 were found to be the most susceptible differential cultivars, both showing susceptible reactions to 10 races. Race 34 was the only race that was pathogenic only to differential cultivars from Andean gene pool, whereas races 9, 73, 128, 321 and 385 were pathogenic only to differential cultivars from Mesoamerican gene pool. It is suggested that the Ethiopian population of *Colletotrichum lindemuthianum* should be regularly monitored for emergence of new races, and race identification studies need to be continued with advanced techniques like molecular method by including more isolates across different agro-ecologies of Ethiopia.

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6. References

Allen, D.J., Buruchara, R.A. and Smithson, J.B. 1998. Diseases of Common bean. The pathology of food and pasture legumes. CAB International. Pp. 179–265.

Alzate-Marin A.L., Menarim H., José M.C., Everaldo G.B. and Maurilio A.M. 2000. Identification of a RAPD marker linked to the Co-6 Anthracnose resistant gene in Common bean cultivar AB 136. *Genetics and Molecular Biology,* 23(3): 633–637.

Amin Mohammed. 2013. An Overview of distribution, biology and the management of common bean anthracnose. *Journal of Plant Pathology and Microbiology*, 4: 1–4.

Amin Mohammed, Amare Ayalew and Nigusie Dechassa. 2014. Effect of integrated management of bean anthracnose (*Colletotrichum lindemuthianum*) through soil solarization and fungicide application on epidemics of the disease and seed health in Hararghe highlands, Ethiopia. *Journal of Science and Sustainable Development*, 4: 182–182.

Ansari, K.I., Palacios, N., Araya, C., Langin, T., Egan, D. and Doohan, F.M. 2004. Pathogenic and genetic variability among *Colletotrichum lindemuthianum* isolates of different geographic origins. *Plant Pathology,* 53: 635–642.

Batureine, M.J. 2009. Diversity of *Colletotrichum lindemuthianum* and reaction of common bean germplasm to anthracnose disease. MSc. Thesis, Makerere University, Uganda.

Bigirimana, J., Fontaine, R. and Höfte, M. 2000. Bean Anthracnose: Virulence of *Colletotrichum lindemuthianum* isolates from Burundi, Central Africa. *Plant Disease*, 84(4): 491. http://hdl.handle.net/1854/LU-133099.

Bigirimana, J. and Hofte, M. 2001. Bean anthracnose: Inoculation methods and influence of plant stage on resistance of *Phaseolus vulgaris* cultivars. *Journal of Phytopathology,* 149: 403–408.

CIAT (International Center for Tropical Agriculture). 1997. Bean program annual report 1995. Working document No. 163. Centro Internacional de Agriculture Tropical, Cali, Colombia.

Cláudia, T., Maria Celeste, G.V., Pedro Soares, V.F., William Mário de, C.N. and João Batista, V. 2002. Characterization of *Colletotrichum lindemuthianum* races in Paraná State, Brazil. *Crop Breeding and Applied Biotechnology,* 2(1): 55–60.

Fernandez, M., Casares, A., Rodr´ıguez, R. and Fueyo, M. 2000. Bean germplasm evaluation for anthracnose resistance and characterization of agronomic traits: A new physiological strain of *Colletotrichum lindemuthianum* infecting *Phaseolus vulgaris* L. in Spain. *Euphytica,* 114: 143–149.

Fitsum Sileshi, Amin Mohammed, Thangavel, S. and Mulugeta Negeri. 2014. Field management of anthracnose (*Colletotrichum lindemuthianum*) in common bean through foliar spray fungicides and seed treatment bioagents. *Science, Technology and Arts Research Journal,* 3(2): 19–25.

Genchev, D., Christova, P., Kiryakov, I., Beleva, M. and Batchvarova, R. 2010. Breeding of common bean for resistance to the physiological races of anthracnose identified in Bulgaria. *Biotechnology,* 24(2): 1814–1823.

Gonçalves-Vidigal, M.C., Thomazella, C., Elias, H.T. and Vidigal Filho, P.S. 2008. Characterization of

Colletotrichum lindemuthianum isolates using differential cultivars of common bean in Santa Catarina state, Brazil. Department of Agronomy, University of Maring, Brazil 43–44.

Gonçalves-Vîdigal, M.C., Nunes, M.P.B.P., Cruz, A.S., Alves, M.A., Sousa, L.L. and Vidigal Filho, P.S. 2010. Characterization of *Colletotrichum lindemuthianum* isolates from Matogrosso State, Brazil. USDA Publications, Reports of Bean Improvement Cooperative and National Dry Bean Council Research Conference. http://handle.nal.usda.gov/10113/IND44207099.

Gonzalez, M., Rodriguez, R., Zavala, M.E., Jacobo, J.L., Hernandez, F., Acosta, J., Martinez, O. and Simpson, J. 1998. Characterization of Mexican isolates of *Colletotrichum lindemuthianum* by using differential cultivars and molecular markers. *Journal of Phytopathology,* 88: 292–299.

Gonzaz, K.K. 2016. Identification of *Colletotrichum lindemuthianum* and introgression of its resistance gene(s) to common bean (*Phaseolus vulgaris* L.) adapted in Tanzania. MSc. Thesis. Sokoine University of Agriculture, Morogoro, Tanzania.

Habtu Assefa, Sachet, I. and Zadoks, J.C. 1996. A survey of cropping practices and foliar diseases of common beans in Ethiopia. *Crop Protection*, 15(2): 179–186.

Kelly, J.D. and Vallejo, V.A. 2004. A Comprehensive review of the major genes conditioning resistance to anthracnose in common bean. *HortScience*, 39: 1196–1207.

Mahuku, S.G., Jara, C.E., Cajiao, C. and Beebe, S. 2002. Sources of resistance to *Colletotrichum lindemuthianum* in the secondary gene pool of *Phaseolus vulgaris* and in crosses of primary and secondary gene pools. *Plant disease,* 86: 1383–1387.

Mahuku, S.G. and Riascos, J.J. 2004. Virulence and molecular diversity within *Colletotrichum lindemuthianum* isolates from Andean and Mesoamerican bean varieties and regions. *European Journal of Plant Pathology,* 110: 253–263.

Masunga M., Nchimbi-Msolla S., Robert M. and Luseko A. 2020. Races of *Colletotrichum lindemuthianum* (Sacc. & Magnus) Briosi & Cavara in major bean growing regions in Tanzania. *African Journal of plant science*, 4(8): 308–314

Moses, J.K., Aston, E., Vincent, K., Stanley, T.N., Pamela, P., Clare, M., and Phinehas, T. 2016. Pathogenic variation of *Colletotrichum lindemuthianum* causing anthracnose of beans (*Phaseolus vulgaris*) in Uganda. *International Journal of Phytopathology,* 05(03): 89–98.

Padder B.A., Sharma P.N., Awale H.E. and Kelly J.D. 2017. *Colletotrichum lindemuthianum*, the causal agent of bean anthracnose (offered review). *Journal of Plant Pathology,* 99(2): 317–330.

Pastor-Corrales, M.A., Otoya, M.M. and Molina, A. 1995. Resistance to *Colletotrichum lindemuthianum* isolates from Middle America and Andean South America in different common bean races. *Plant disease,* 79: 63–67.

Pastor-Corrales, M.A. 2004. Review of co-evolution studies between pathogens and their common bean hosts: Implication for the development of disease-resistant beans. Annual report of bean improvement cooperative 47: 67–68.

Perseguini, J.M.K.C., Oblessuc, P.R., Rosa, J.R.B.F., Gomes, K.A., Chiorato, A.F. and Carbonell, S.A.M. 2016. Genome-wide association studies of anthracnose and angular leaf spot resistance in common bean (*Phaseolus vulgaris* L.). *PLOS ONE,* 11(3): e0150506.

Sicard, D., Michalakis, Y., Dron, M. and Neema, C. 1997. Genetic diversity and pathogenic variation of *Colletotrichum lindemuthianum* in the three centers of diversity of its host *Phaseolus vulgaris*. *Journal of Phytopathology,* 87: 807–813.

Tesfaye Beshir. 1995. Studies on the importance, pathogenic variation and management of bean anthracnose in Ethiopia. CIAT African workshop series (CIAT); no. 37, Pp. 33–43.

Tesfaye Beshir. 1997. Loss assessment study on haricot bean due to anthracnose. *Pest Management Journal of Ethiopia*, 1: 69–72.

Tesfaye Beshir. 2005. *Colletotrichum lindemuthianum* on beans in Ethiopia. Geographical distribution, importance, pathogenic variation, and management. Pp. 176-181. *In*: PABRA Millennium Workshop, 2001, Arusha, Tanzania.