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Review Article

Pathogenicity and Approaches for Management of Anthracnose in Common Bean (*Phaseolus vulgaris*) in Africa

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Abstract

Common bean plays significant role for human health globally and consumption of common bean is high in Africa as compared to other regions of the world. Despite common bean's potential in Africa, productivity remains low due to diseases, drought and poor crop management. Anthracnose disease plays major role in reducing common bean grain yield in Africa. It is caused by seed-borne fungal pathogen Collectotrichum lindemuthianum leading to 100% yield loss. Limited and fragmented information on fungal infection, pathogenicity and management of common bean anthracnose in Africa affects decisions regarding anthracnose management. This review has been produced to collect information regarding anthracnose disease and its management in beans in Africa, which will be of great value to bean stakeholders. C. lindemuthianum can survive up to five years in infected seeds. During this time, seed is the main source of inoculum, infection and transmission of pathogen to new locations. Other sources and mechanisms of transmission include infected residues, farm tools, water, wind, and disturbance of moist foliage by animals, insects and people. Anthracnose is a hemibiotrophic pathogen, first establishing biotrophic interactions with common bean plant before switching to necrotrophism, causing significant yield loss. Mechanical force, chemical weapons, toxins and growth regulators facilitate pathogenesis. Use of anthracnose-resistant varieties is recommended to control common bean anthracnose followed by integrated anthracnose management. Future research in Africa should focus on why farmers rely heavily on local bean cultivars as seed and should use tricot as tool to screen anthracnose-resistant varieties and evaluate anthracnose management options for increased productivity, nutrition and income. © 2022 Friends Science Publishers

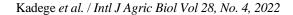
Keywords: Anthracnose; Biotrophic; Common bean; Disease resistance; Necrotrophic; Tricot

Introduction

Common bean (*Phaseolus vulgaris* L.) is the most important grain legume, with 28.9 million tonnes produced globally on approximately 33 million hectares (FAOSTAT 2019). In Africa, the common bean is produced on 7.8 million hectares, which is equivalent to approximately 25% of the global area of common bean production (Nadeem *et al.* 2021). Common bean is consumed by more than 100 million households in Africa (Mukankusi *et al.* 2019). The top five African countries producing common bean are

Tanzania, Uganda, Kenya, Burundi and Ethiopia (FAOSTAT 2019). Common bean contains important nutrients for human health such as carbohydrates (50–60 mg kg⁻¹), dietary fiber (75–80 mg kg⁻¹), energy (50–70 mg kg⁻¹), proteins (18.5–25 mg kg⁻¹), iron (18.8–82.4 mg kg⁻¹), magnesium (19–26 mg kg⁻¹), potassium (43–300 mg kg⁻¹) and zinc (32.6–70.2 mg kg⁻¹) (Rubyogo *et al.* 2019; Punia *et al.* 2020). Common bean is a healthy food, the consumption of which can reduce incidence of diseases such as cancer and diabetes, due to its low fat content and lack of cholesterol (Robinson 2019).

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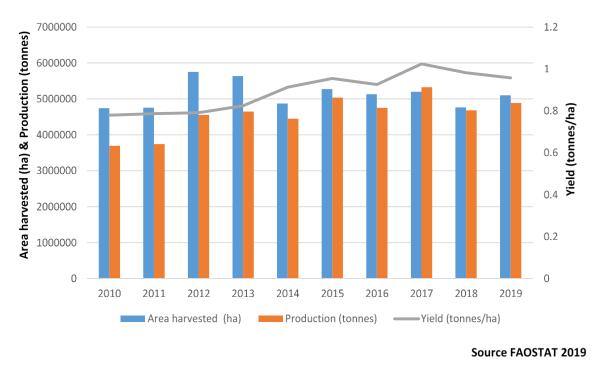


Fig. 1: Common bean production (tonnes) and area under bean cultivation (hectares) in Africa in 2010–2019

Bean consumption in Africa is high, reaching up to 66 kg person⁻¹ y⁻¹, while the global average is 2.51 kg person⁻¹ y⁻¹ (Nadeem *et al.* 2021). This indicates the importance of beans as a food crop in Africa compared with other regions. However, on-farm productivity remains low (0.8 t ha⁻¹) (Fig. 1), compared to a potential reported productivity of 2.5–5 t ha⁻¹ (Muthoni *et al.* 2017). Low productivity is attributed to both biotic and abiotic factors, including diseases, insect pests, poor seed quality, drought, heat, low soil fertility and poor crop management. Of these factors, disease, particularly anthracnose caused by *Colletotrichum lindemuthianum* is an important bean yield inhibitor (Padder *et al.* 2017; Mlemba 2021).

The C. lindemuthianum, first discovered in Lima bean (*Phaseolus lunatus*) samples from Germany in 1875 by Lindemuth and described by Saccardo (1878). Since then, it has spread and is now distributed worldwide including in Africa, Canada, Europe, Latin America and the USA (Ansari et al. 2004). In Africa, the disease is of particular concern in Burundi, D.R. Congo, Ethiopia, Kenya, Rwanda, Tanzania and Uganda (Farrow and Muthoni 2020). Anthracnose is most serious in temperatures of about 17°C, with relative humidity above 92% and soil pH of 5.8-6.5 (Padder et al. 2017). Bean anthracnose attacks leaves, stems, pods and seeds, causing dark brown necrotic lesions that decrease leaf photosynthetic activity. Reduced photosynthesis results in leaf senescence, stunted bean growth and eventual death. Yield loss of up to 100% due to anthracnose has been reported in Africa (Masunga et al. 2020).

In comparison, angular leaf spot causes yield loss of 80% and bean common mosaic virus causes yield loss of 40% (Mwaipopo *et al.* 2017) In Africa; some of the commercial bean varieties are susceptible to anthracnose disease (Muthoni *et al.* 2017), therefore, farmers in major bean-producing regions rely heavily on growing local cultivars. It is not clear whether local cultivars are preferred over commercial varieties based on resistance to anthracnose or due to differences in the ease of disease management.

Anthracnose can be managed by crop rotation, planting resistant varieties, foliar application of plant extracts, seed treatment and foliar application of contact or systemic fungicides. However, common bean production in Africa is vulnerable to anthracnose due to poor management and the prevalence of diverse races of the anthracnose pathogen that render the majority of varieties susceptible. Genes in common bean that confer resistance to anthracnose have been documented (Ferreira et al. 2013). However, common bean breeders are unsure which gene to deploy in resistance breeding programs. Pathogen variability is documented (Munda et al. 2009; Palaciog 1u et al. 2021) and marker-assisted selection is used in developing resistant varieties (Meziadi et al. 2016; Padder et al. 2017). Nevertheless, great variability in pathogenicity makes management of anthracnose disease in Africa difficult (Uwera et al. 2021). This is due diversity and virulence extensive of С. to *lindemuthianum*, where a single gene can affect stability

of resistance in the bean plant and a complementary gene conditions pathogen virulence. Information on fungal infection and pathogenicity of *C. lindemuthianum* is very limited and fragmented. Therefore, the aim of this review is to discuss mechanisms for anthracnose infection and pathogenicity and to design suitable disease management strategies. This information will facilitate stakeholders working on common bean in Africa to better manage anthracnose disease to allow for increased productivity, nutrition and income.

Mechanisms of infection

Infection is the process by which a pathogen establishes contact with and acquires nutrients from susceptible host cells or tissue. The process of anthracnose infection begins when a C. lindemuthianum conidium (spore) lands on the leaf, stem or pod of a bean, adheres to the plant cuticle and germinates (Pellier et al. 2003; Alkemade et al. 2021). Conidia are disseminated by splashes from rain and quickly attach to the aerial parts of a plant to infect it. Under humid conditions, the conidium germinates and develops a spherical structure, the appressorium, which is essential for epidermal cell penetration (Sharma and Kulshrestha 2015). The appressorial surface adhering to the cuticle is flattened and a pore forms on the flat surface. Subsequently, an infection peg emerges through this pore, pierces the bean leaf cuticle and cell wall and directly mediates entry into the host epidermal cell. Oxidase, cutinase and lipases are secreted from the infection peg to degrade the plant cuticle and wax layers (Pawlowski and Hartman 2016).

Fungal spores germinate when they come into contact with the bean plant, then a germ tube elongates to form an appressorium for penetration (Chethana et al. 2021). Germ tube elongation and differentiation occurs in response to environmental signals like surface hardness, hydrophobicity, plant signals and surface topography (Tucker and Talbot 2001). If appropriate environmental signals are not received, the germ tube remains undifferentiated and will eventually stop growth upon nutrient depletion. If appropriate physical and chemical signals are detected by the germ tube, a complex morphogenetic program is induced, causing appressorium formation which results in indentation in the cell wall. The morphogenetic events from spore attachment to appressorium formation are motivated by host plant signals like cutin monomers, ethylene and topographic signals, and environmental factors like temperature and substrate hydrophobicity. Finally, an infection peg protrudes from the appressorium, penetrating through the cell wall where infection hyphae grow and develop into infection vesicles. C. lindemuthianum is considered a hemibiotrophic fungus (Dubrulle et al. 2020), spending part of the infection cycle as a biotroph and the other as a necrotroph.

Phases of infection

Biotrophic phase: The biotrophic phase is the first stage of infection, where broad primary hyphae grow out of the infection vesicle (Padder *et al.* 2017; Ciofini *et al.* 2022). During this phase, the fungus grows between the cell wall and the plasma membrane of host cells without causing death. At this stage, the pathogen establishes interactions with the host plant, producing surface proteins that are important for adhesion and invasion. After successful penetration, the infection vesicle and primary hyphae are formed inside the living host's epidermal cells and invaginate the host cell's plasma membrane. Biotrophic fungal pathogens contain sophisticated structures like appresoria, infection pegs and haustoria used for nutrient absorption and secretion of effector proteins.

The pathogen's primary hyphae penetrate through cell walls by mechanical force. The hyphae grow near the infection vesicle and follow the plant cell walls in such a way that half of the hyphal circumference is in connection with the cell wall at all times (Suparman et al. 2018). C. *lindemuthianum* forms infection structures for successful attachment, host recognition, penetration, pathogenesis and proliferation. The structures are regulated by gene expression and complex regulatory pathways to facilitate compatible interactions between plant tissue and the pathogen (Padder et al. 2017). Lytic enzymes, carbohydrates and proteins are developed for virulence and haustoria for nutrient absorption and metabolism (Gebrie 2016; Pradhan et al. 2021). Once the fungal effector has bypassed the plant's defense mechanisms, the plant will no longer resist, reducing its production of defense signaling molecules such as salicylic acid. Depending on environmental conditions, the biotrophic phase ends 2-3 days after inoculation (Suparman et al. 2018). Thereafter, the fungus switches to the necrotrophic phase, which corresponds with the onset of anthracnose symptoms.

Necrotrophic phase: The necrotrophic phase is the second stage of infection, comprising many thin hyphae branching off from the primary hyphae and moving freely through the bean plant in all directions, penetrating cell walls and membranes. During the necrotrophic phase, the fungus differentiates secondary hyphae, which are thinner than primary hyphae and grow extensively, leading to the disorganization and death of infected host cells (Suparman et al. 2018; Alkemade et al. 2022). At this stage, the pathogen produces a cell wall-degrading enzyme that kills host cells by hydrolysis. Growth and multiplication of the fungal pathogen is favored by certain weather conditions. If optimal rainfall, temperature and relative humidity occur, the pathogen can invade the bean plant to its maximum potential regardless of plant defense and as a consequence anthracnose develops (Wang and Kerns 2017). The pathogen spreads into the leaves, stem, pods and seeds (Alkemade et al. 2022) by direct growth through cells as intracellular mycelia; subsequently, it invades the xylem. If successful, *C. lindemuthianum* grows and continues branching within the infected host plant tissue until the plant dies. The necrotrophic phase is completed 6–7 days after the beginning of the infection cycle.

Switching from the biotrophic to the necrotrophic phase is facilitated by the *CLTA1* gene. This encodes a protein that coats the hyphae to form a pseudo cell wall to avoid recognition by the common bean plant (Dufresne *et al.* 2000). Consequently, the pathogen produces phytotoxins which kill the plant cells, preventing them from responding in a synchronized means to resist infection. Toxins cause pores to form in the mitochondria through which small molecules leak, ceasing adenosine triphosphate (ATP) synthesis and causing cell death (Dufresne *et al.* 2000). Finally, the toxin induces reactive oxygen species in the bean plant which cause membrane breakdown and nutrient leakage.

Mechanisms of pathogenicity

Pathogenicity is the ability of a pathogen to interfere with the essential functions of a host plant or animal, thereby causing a disease. The mechanism of C. lindemuthianum pathogenesis involves the use of: (1) mechanical forces which include the formation of appressoria and penetration of the host cuticle and cell walls; (2) chemical weapons including enzymes like amylases, cellulases, cutinases, hemicellulases, lignases, lipases, pectinases and proteases; (3) non-host specific and host-specific toxins and (4) growth regulators including abscisic acid, auxins. cytokinins, ethylene and gibberellins (Chethana et al. 2021). Moisture is an important environmental factor influencing the formation of appressoria and development of anthracnose. Moisture affects infection, dispersal, spore germination, anthracnose establishment and development. The pathogen is inactive during the dry season, becoming active when favorable conditions are encountered. It detects and responds to host cues like chemical signals, electrical stimuli, pH, host surface chemistry and surface hardness on penetration (Sharma and Gautam 2019).

Anthracnose is common in African farmers' bean fields and has wide pathogenic and molecular variability. The disease is becoming more noticeable in Africa due to climate change. A total of 160 races of *C. lindemuthianum* have been described in Africa (Table 1). Common bean cultivars grown in Africa have significant diversity and adaptation to different climatic and agronomic conditions, and many of the several Andean and highly virulent Mesoamerican *C. lindemuthianum* races have been characterized in Africa.

During infection of common bean, the pathogen secretes extracellular protein and glycoproteins, which contribute to pathogenicity. Nevertheless, the amount of protein and glycoproteins produced is unknown. Extracellular protein establishes a molecular dialogue between the parasite and host. Hydrolytic enzymes, such cutinase and pectinases, are produced when as anthracnose is attached to the common bean plant, during establishment, development and colonization (Oeser et al. 2002; Li et al. 2007). The production of cellulolytic and pectinolytic enzymes is determined by the degree of cell wall penetration during pathogenesis and the level of enzyme inhibition by the host; this eventually interferes with disease development. The mechanism of pathogenhost interaction involves a series of stages from initial attachment of C. lindemuthianum, to infection, disease development and colonization, described in the following sections.

Pathogen attachment to the bean plant: Attachment of C. lindemuthianum to the bean leaf surface is an essential pre-infection event that determines infection success. A conidium is used as the propagule, adhering to the plant surface with the role of host recognition and subsequent fungal development. The propagule contains a mucilaginous substance, a mixture of water-insoluble polysaccharides, glycoproteins lipids and fibers, which when moistened become sticky and facilitate the pathogen's adherence to the plant (Chethana et al. 2021). Once adhesion to a leaf or stem has occurred, the pathogen can become established. If adhesion is disrupted by nontoxic synthetic compounds, the spore will neither infect leaves nor stem and there will be no disease development. Temperature changes can alter the adhesion properties of conidia (Sela-Buurlage et al. 1991). Fluctuations in temperature influence respiration and metabolic rate, both of which impair adhesion (Mercure et al. 1995), though the mechanism of this influence is unknown. The adhesion of conidia declines beyond 30 davs.

High turgor pressure develops on melanized appressoria walls which supports penetration. Penetration hyphae accumulate a cytoskeleton at their tip which secretes degrading enzymes including cellulases, cutinase, lignases and pectinases to facilitate penetration of the cuticle and plant cell wall (Sharma and Gautam 2019). Infection hyphae differentiate within the bean plant and during differentiation, degrading enzymes are synthesized to facilitate successful establishment, which leads to development of disease symptoms.

Pathogen establishment: Once *C. lindemuthianum* passes through the external protection of the bean plant, it lives within the host for some time, to obtain nutrients and produce toxins that cause disease symptoms. The pathogen completes parasitic colonization of the plant by reprogramming the defense electron structure of host cells through a range of disease effector proteins (Chethana *et al.* 2021). Apoplastic effectors are secreted in the plant by extracellular targets and surface receptors. Cytoplasmic effectors translocate inside bean plant cells *via* an

infection vesicle that invigilates in the living host. Effectors facilitate infection or activate disease reaction. The pathogen produces cytokinins on the leaf surface, which is the primary site for pathogen infection of the common bean plant (Sharma and Gautam 2019).

Anthracnose development: Seed-borne infection plays a significant role in disease development. Seasonality affects the persistence of anthracnose. Disease development, spread and severity index coincide with frequent heavy rain and moderate temperatures (Table 2). Heavy rain spreads *C. lindemuthianum*, stimulating and releasing fungal spores embedded in gelatinous acervuli (Mugambi 2013). *C. lindemuthianum* requires cool temperatures for growth, infection and development. High temperatures do not affect spread of anthracnose disease, but prolonged high temperatures increase disease severity by the disease spreading slowly for long time.

During disease development, a brick red to purplish discoloration is observed on the veins on the lower surface of the leaf (Fig. 2). Anthracnose disease symptoms extend on the upper surface of the leaf and at the base of the stem, progressing upwards and producing dark brown to black lesions along the veins. Disease symptoms are also observed on bean pods, causing dark red sunken spots (Fig. 2) and finally on bean seeds. In severe infections, young pods shrivel and dry prematurely. When many pods are infected, the number of seeds infected increases and grain yield and seed quality decreases (Mohammed 2013).

Anthracnose resistance mechanism: Common bean has a mechanism that may defend against anthracnose. The crop contains phytochemicals such as catechol, polyphenols and salicylic acid which act as proteinase and polygalacturonase inhibitors and antioxidants. These phytochemicals restrict/interfere with pathogen nutrition and retard anthracnose development, contributing to disease resistance. Once these phytochemicals are no longer sufficient to stop infection development, plant cells increase levels of antifungal phenolic compounds, producing fungitoxic quinones at the infection site. These toxins increase active oxygen species, making the bean plant cell an unfavorable medium for further pathogen development (Weidner et al. 2018). The ability to increase phytochemical production differs depending on bean variety and the environment in which the bean is grown (Ghasemzadeh et al. 2018), which leads to differences in anthracnose resistance.

Management of anthracnose disease

Use of resistant varieties: Cultivation of resistant varieties is the most effective and efficient method of anthracnose management (Negera and Dejene 2018; Palaciog Iu *et al.* 2021; Uwera *et al.* 2021), because the major transmission and survival structure for the anthracnose pathogen is the seed, in which the pathogen

can survive for up to five years. Movement of infected seed between sites increases the chance of spreading anthracnose from one location to another. To avoid this, farmers are advised to use improved bean varieties (Table 3).

Use of resistant varieties is the most economical and effective means to control anthracnose (Paulino et al. 2022), as it ensures protection against the disease, saving time, energy and money that would otherwise be spent on other control measures. Resistant varieties are easy to use, completely avoid the disease cycle, are better for the environment as demand for agrochemicals is reduced and ensure production of healthy beans (Mohammed 2013; Negera and Dejene 2018; Prabha et al. 2021). Use of resistant varieties significantly increases grain yield, by 225 kg ha⁻¹ (Mukankusi *et al.* 2019). Although anthracnose resistance provides economical control, farmers' adoption of improved, resistant varieties is limited. Most African farmers use farm-saved bean seed from previous harvests or purchase seed from neighbouring farmers or at local village markets (Sperling et al. 2021). As a consequence, anthracnose levels are high. Responsible authorities (seed regulatory authorities, seed companies and agro-dealers), should ensure timely and local availability of improved bean varieties at an affordable price.

Due to the high degree of genetic and physiological variability of C. lindemuthianum, management using single-gene resistance is not so much effective, as resistance is not controlled by single gene. For instance, four differential cultivars, G2333 (Co-42, Co-52, Co-7=Co-35), Cornell 49-242 (Co-2), Tu (Co-5) and AB136 (Co-6, Co-8), were reported to confer broad-spectrum resistance to anthracnose in Brazil and Uganda, yet succumb to disease caused by some C. lindemuthianum races. With high pathogen diversity and frequent emergence of new pathotypes, researchers should continue identifying new sources of resistance to bean anthracnose disease. In Africa, many common bean farmers work in diverse environments, exposed to different climatic and agronomic conditions. Different agroecosystems can be favourable for different varieties of common beans. To recommend resistant varieties to anthracnose, researchers are encouraged to conduct triadic comparison of technologies (TRICOT) in evaluation of anthracnose-resistant varieties, which will help to evaluate new varieties on a farm level.

Tricot is a simple format that engages many farmers in evaluation, from the initial point of trial establishment onwards, providing feedback on what has been observed from the experiment. Tricot combines farmer-generated trials and preferences with many seasons of data collection on cropping systems and household farming, allowing in-depth analysis at low cost. The tool engages many available management options and involves many farmers. It allows each farmer to evaluate three randomly Table 1: Races of C. lindemuthianum characterized in Africa from 1991 to 2021

Country	Races of C. lindemuthianum	Total no of races	Abundant race	Highly virulent race	References
Burundi	9, 69, 84, 87, 141, 246, 358, 384, 385, 401, 448, 449, 485, 515, 576, 768	10	401, 485	401, 485	Bigirimana <i>et al.</i> (2000); Kamiri <i>et al.</i> (2021)
Ethiopia	9, 34, 73, 128, 272, 321, 385, 465, 587, 898, 1011, 1172, 1250, 2073, 2225, 2255, 2260, 3047	18	9, 272, 1011, 2260	2073, 2225, 2260, 3047	Gezahegn et al. (2021)
Kenya	1, 2, 17, 23, 38, 55, 485	7	38, 55	38	Nunes et al. (2021)
South Africa	3, 6, 7, 49, 65, 80, 81, 83, 89, 263, 323, 390, 593	13	3, 7, 81, 83, 89, 263	7, 81, 83, 89, 263	Koch (1996); Muth and Liebenberg (2009); Nunes <i>et al.</i> (2021)
Tanzania	0, 2, 9, 12, 28, 31, 38, 39, 60, 62, 63, 91, 98, 101, 105, 112, 128, 129, 133, 155, 166, 167, 182, 191, 192, 274, 277, 287, 316, 344, 398, 524, 618, 661, 716, 770, 776, 832, 849, 944, 958, 1176, 1271, 1478, 1510, 1515, 1678, 1696, 1805, 1891, 2061, 2434, 2566, 2614, 3068, 3264, 3610	57	0, 2	3610	Mwalyego (1991); Ansari <i>et al.</i> (2004); Masunga <i>et al.</i> (2020)
Uganda	5610 0, 1, 2, 3, 4, 6, 14, 17, 19, 23, 42, 55, 81, 102, 128, 130, 227, 262, 264, 268, 320, 352, 375, 382, 386, 452, 481, 503, 511, 704, 713, 767, 784, 1023, 1024, 1094, 1169, 1175, 1334, 1471, 1527, 1536, 1538, 1791, 1834, 1856, 1857, 1989, 2023, 2039, 2045, 2047, 2079, 2479, 3086, 4033, 4095	57	167, 2047, 4095	1024, 1536, 1538, 1856, 1857, 1989, 2023, 2039, 2045, 2047, 3086, 4033, 4095	Nkalubo 2006; Mwesigwa 2008; Kiryowa <i>et al.</i> (2016)
Zambia	37,39, 53, 65, 73, 84, 207, 247, 255, 342, 382, 407, 485, 510,566	13	247	247	Zulu 2005; Nalupya <i>et al.</i> (2021)

Table 2: Climate variables during the common bean cropping season (meteorological station located at Arusha airport). Source: TARI Selian

Year	Mean rainfall March-June (mm)	Mean temperature (°C) March–June		Mean RH (%) March-June	
		Max.	Min.		Yield loss (%)
2015	836.1	26.5	15.6	87.1	64
2016	753.4	26.4	15.3	86.5	60
2017	924.7	26.5	15.5	87.4	65
2018	1286.7	26.9	16.1	94.7	95
2019	1132.3	26.6	15.7	91.3	67
2020	1199.9	26.7	15.9	93.1	71
2021	557.9	26.3	15.4	86.6	42



Fig. 2: Symptoms of anthracnose disease at the Tanzania Agricultural Research Institute (TARI), Selian

assigned genotypes from a large set of genotypes. The tool reduces bias and risk, by recording performance data across different growing seasons and locations (Etten *et al.* 2018, 2019). In addition, the tool allows sharing data through ClimMob software for meta-analysis to validate and improve recommendations based on existing data. Tricot has been used in different countries such as Ethiopia, India and Nicaragua, with promising results across various technologies (Etten *et al.* 2019). Through the tricot approach, many farmers in Tanzania are being engaged to test a wide range of common bean varieties for anthracnose resistance and adaptation to different environments on farms where the disease is prevalent. More than 1500 on-farm tricot trials have been

Country	Variety				
Burundi	LM9220492, MLB122-94B, RWR 2091, CODMLB003, IZ0201543, MAC44, MAC70, MUHORO, RWV1129, RWV 1272				
DR Congo	ECAPAN 021, G16157, TY 3396 -12, PRELON, K 132, MAHARAGI SOYA, RJB – 1, VCB81013, NUA 99				
Eswatini	MASAI – RED, NUA 45, ZEBRA, KAMIESBERG, MN 12685–15, MR 13557–17-7, MR 14215-9, RCB 265, WERNA				
Ethiopia	GLP-2, KATB1, Awash-1, SER-119, SER-125, Fedis, Babile, Hirna, BRC-ACC.NO-4, SARI-1				
Ghana	G53, G90, ROBA-1, SMR 53				
Kenya	KAT-RM-001, KAT-SR 01, KAT-SW-12, KAT-SW-13, KCB 13-02, KCB 13-09, KCB 13-11, MN6				
Lesotho	CAL143				
Madagascar	RI 5-3, AND 932-B1, EMP 250-5-1, RI 5-5				
Malawi	KK03/KK25/68/S-F, KK25/MAL/112/S-F, KK25/MAL/19/S-F, KK25/NAG/184/S-L, MAL/KK25/9/S-F, MAL/KK25/443/S-L,				
	NAG/KK25/168/S-L, BF 13607-9, SER 124, SER 83				
Mozambique VTT 924/4-4, VTTT 925/9-/-2					
Rwanda	RWV1348, RWV 2269, RWV 3317, SB - 273				
South Africa KAMIESBERG, PAN 9213, PAN 9216, RS 7					
Tanzania	TARIBEAN 1, TARIBEAN 2, TARIBEAN 3, TARIBEAN 4 TARIBEAN 6, Selian 14, Selian 15, Calima Uyole, Fibea, Rosenda, Pasi,				
	Uyole 16, Uyole Nyeupe				
Uganda	NABE 4, NABE 10, NABE 15, NABE 16, NABE 18, NABE 19, NABE 20, NABE 21, NABE 26C, NABE 27C, NABE 29C, NAROBEAN				
	1, NAROBEAN 2, Moore 88002, MAC 44, NYIRAMUHONDO				
Zambia	SPS2-4P-24, C30-920				
Zimbabwe	Gloria, NUA45, SUG 131, SWEET VIOLET, CHERRY, SC SUPERIOR				

established in an incomplete block design in the northern, southern highland and western zones of Tanzania. The process offers practical learning to farmers, and provides interpretable and meaningful results for real environments on different farms. The The approach works efficiently, and many stakeholders feel it is useful (Etten *et al.* 2018, 2019).

Cultural control: Produce bean seeds in areas that are not anthracnose hotspots (Yesuf and Sangchote 2005; Mohammed 2013; Etana 2022). Plant certified disease free seed grown in non-hotspot areas to anthracnose. Plant beans following the recommended planting dates (Girma et al. 2022) to avoid the extraordinary conditions favoring anthracnose development. Plant beans following the recommended spacing of 50 cm between rows and 20 cm between plants with two seeds per hole, or 50 cm between rows and 10 cm between plants with one seed per hole, to avoid foliar drying (Bush 2009). Timely weeding is required to ensure efficient air circulation and to decrease moisture in the bean plant. Over-irrigation should be avoided to reduce wet conditions which promote disease infestation. Weekly field scouting for anthracnose symptoms is encouraged in order to identify anthracnose as soon as it appears and allow the implementation of control measures to avoid spreading the disease (Batureine 2009; Etana 2022). Disinfect seed storage facilities with a 10% bleach solution equivalent to 0.5% sodium hypochlorite and Dettol to prevent contamination (Buruchara et al. 2010). Incoporate bean residues under the soil immediately after harvest to reduce fungus survival during winter (Yousef 2021). Additionally, Rotate common bean field with cereals and solanaceous crops every two to three years to minimize further pathogen survival (Buruchara et al. 2010; Etana 2022).

Use phosphate fertilizer to the bean field to reduce the incidence and severity of anthracnose (Gadaga *et al.* 2017). In Brazil, spraying potassium phosphate (KI) and manganese phosphate (Mn) reduced area under the disease progress curve (AUDPC) by 78.3 and 77% respectively (Gadaga et al. 2017). Potassium phosphate formulations have also been reported to reduce anthracnose severity by 68% in the USA (Costa et al. 2019). Sodium silicate should be evaluated and promoted for use on common bean fields in Africa to reduce severity of anthracnose. Application of sodium silicate in Brazil increased silicon concentration in bean leaves by 58%, decreased AUDPC by 62% and increased grain yield by 51% (Polanco et al. 2014). Despite these recommendations and implementation of some similar practices by bean farmers in Africa, anthracnose infection continues to threaten farmers' fields. Future research should investigate the optimum burying depth for bean residues and optimum burying period. Recommended spacing of bean plants and avoidance of mono-cropping should be encouraged and timely weeding in and around the field should be emphasized. Weekly field scouting for disease symptoms should be encouraged. A literature search encountered limited information on the application of phosphate fertilizer and sodium silicate in Africa. Information from other countries including Brazil, evokes the possibility of using phosphate fertilizer and sodium silicate to reduce anthracnose severity on common beans and consequently achieve greater gains in yield. Future research in Africa should explore the potential of phosphate fertilizer and sodium silicate to control common bean anthracnose.

Biological control: Use of plant growth promoting rhizobacteria (PGPR) such as species of *Pseudomona* and *Bacillus* (Sharf *et al.* 2021) and various species of fungi namely *Trichoderma* spp. (Javaid *et al.* 2021; Khan *et al.* 2021), *Penicillium* spp. and *Aspergillus* spp. (Khan and Javaid 2022a, b) as biological control agents against many fungal plant pathogens is gaining much importance nowadays. Spore suspension of *Trichoderma viride* can

be applied as a seed dip and soil drench to control C. lindemuthianum (Bankole 1996). Furthermore, bean seeds can be smeared with cultures of Gliocladium virens, T. hamatum or T. harzianum before sowing to inhibit pathogen infection (Padder et al. 2010). Bean seeds can be inoculated with rhizosphere bacteria from genera such as Arthrobacter, Bacillus, Pseudomonas and Serratia to control anthracnose (Duangkaew and Monkhung 2021). Extracellular metabolites like antibiotics, lytic enzymes, siderophores, and volatile compounds produced by rhizobacteria (Bacillus cepacia and Pseudomonas fluorescens) effectively reduce lesions on and damage to common bean plants caused by anthracnose. Biological application of plant extracts such as Alchornea cordifolia, Azadirachta indica, Carica papaya, Cymbopogon citratus, Cymbopogon flexuosus, Lantana camara, Ocimum sanctum, Piper guineense, Piper nigrum, Tabernaemontana pachysiphon, Vernonia polyanthus and *Xylopia aethiopica* on bean leaves and stems can control anthracnose (Enyiukwu et al. 2021). Foliar application of Cymbopogon flexuosus, V. polyanthus and Carica papaya in Brazil reduced anthracnose severity by 57.2, 37.6 and 34% respectively (Silva et al. 2015). Studies in Nigeria recorded high (83%) anthracnose incidence on untreated cowpea plots compared to 20.4, 27 and 30% incidence when L. camara, Tabernaemontana pachysiphon and Alchornea cordifolia treatments were used, respectively (Enyiukwu et al. 2021). Toxic activity of some plant extracts like X. aethiopica, P. guineense and Azadirachta indica in the form of benomyl, carbendazim and thiophanate-methyl minimize anthracnose in legumes (Awurum and Uchegu 2013). Biological control of anthracnose is an economical and environmentally sound approach, but has received comparatively little attention in Africa due to lack of information available for farmers on how and when to use biological control measures. Common bean farmers in Africa should be trained on biological control methods to control the incidence and severity of anthracnose. Researchers should establish demonstration plots to promote the use of plant extracts to control anthracnose, reducing the negative impacts of synthetic pesticide use.

Chemical control: Seed treatment with Apron Star, benlate, carbendazim, difenoconazole, mancozeb, Seed Plus, Seed Care and thiram increases seed germination, controls mycelial growth of C. lindemuthianum, controls seed-borne infection and increases seed quality and grain yield (Buruchara et al. 2010; Padder et al. 2010; Boersma et al. 2020; Alkemade et al. 2022). Foliar spraying of bean plants with azoxystrobin, benomyl, carbendazim, chlorothalonil, folpan, mancozeb, mancozeb + carbendazim, pyraclostrobin or thiophanate-methyl + chlorothalonil at an early stage of disease development reduces pressure from anthracnose (Mohammed 2013; Hirpa and Selvaraj 2016). For example, economic analysis of fungicide application in Ethiopia revealed that

the highest net benefit is obtained from the Awash Melka bean variety when spraved at one- or two-week intervals (USD 953.50 ha⁻¹ and USD 889.60 ha⁻¹) followed by Chercher (USD 848.90 ha⁻¹ and USD 823.3 ha⁻¹) (Hirpa and Selvaraj 2016). In Brazil, pyraclostrobin application provided USD 86–181 ha⁻¹ return on investment due to decreased disease development (Gillard and Ranatunga 2013). Azoxystrobin application in Brazil reduced mean AUDPC by 63% and increased mean yield by 150% (Polanco et al. 2014). In Nigeria, carbendazim and benomyl reduced the development and spread of anthracnose disease on cowpea by 46 and 40% respectively (Emechebe and Florini 1997). The highest marginal rates of return of 3071 and 2568% were observed in Awash-1 without seed treatments, sprayed at flowering and pod setting respectively (Negera and Dejene 2018). Despite these chemical control options, anthracnose disease continues to destroy common bean farmers' fields in Africa. Identified challenges include a lack of information provision at appropriate times for spraying as well as the economic injury level of anthracnose control. Seed treatment fungicides are usually available only in large lots, which are difficult for smallscale farmers to access locally. Farmers' knowledge of seed treatment and foliar spraying methods, application rates, time of application and management methods after application is limited. Therefore, researchers should work with designated authorities to advise farmers accordingly. Research on application methods, rates and timings for chemical control measures of anthracnose disease in Africa should continue. Development of resistance to some fungicides by anthracnose has been reported (Gadaga et al. 2017; Kiptoo et al. 2020). Researchers should evaluate the efficacy of new fungicides that provide cost-effective management options that do not damage the environment.

Integrated disease management: Integrated disease management is the recommended option for anthracnose control since the pathogen infects the seed and all growth stages of the crop and has high diversity. Integration of soil solarization, seed treatment and foliar spray with systemic and contact fungicide effectively reduces anthracnose epidemics (Mohammed 2013). Botanicals and bio-pesticides together with synthetic fungicides have also been shown to efficiently control the disease (Fitsum et al. 2014). Management of primary inoculum (crop rotation, use of resistant varieties) and seed treatment with contact or systemic fungicides effectively controls the disease. For instance, seed treatment with mancozeb followed by carbendazim foliar spray and both seed treatment and foliar spraying with carbendazim significantly reduce bean anthracnose severity (Amin et al. 2013). Ethiopia's Awash-1 variety, without seed treatment and without foliar spray, showed the highest decrease in foliage (-86.0%) and 71.32% pod severity with AUDPC of 2771.19 days for leaves and 1150.25 days for pods compared to Awash-1 variety, with seed treatment and with foliar spray. However, many bean growers in Africa use a single method to control anthracnose and as a result the disease continues to threaten bean fields and reduce grain yield. Common bean farmers lack information on efficient integration, application methods, and timing and rates of application. Training on and promotion of integrated bean anthracnose management is required in Africa.

Conclusion

This review was aimed to assemble information on the mechanism of anthracnose infection in common bean, its pathogenicity and management approaches in Africa. Reviewing the mechanism of anthracnose infection and pathogenicity provides knowledge of host-pathogen interactions between bean plants and C. lindemuthianum. Many details related to successful fungal infection and subsequent disease development have yet to be elucidated. Topics that would benefit from further research include quantification of protein and glycoprotein production by C. lindemuthianum, identification of factors determining whether host penetration results in successful colonization, and exploration of the mechanism by which temperature affects pathogen adhesion to the host. Understanding interactions between pathogen virulence, resistance and host susceptibility is essential. Identification of plantderived signals and parts of signal transduction chains involved in cellulase, cutinase, lignase and pectinase induction and appressorium formation in С. lindemuthianum is important. Opportunities for research on anthracnose management in Africa include exploring why most African farmers use local rather than commercial varieties, and why farm-saved seed is preferred.

Planting resistant varieties is recommended because the seed is the major source and survival structure for anthracnose disease. Integrated anthracnose disease management is also recommended due to the fact that the pathogen occurs from seed throughout all growth stages of common bean and due to high pathogen diversity. Further research integrating the use of resistant varieties, testing the efficacy of cultural, biological and chemical controls is of great importance to design consolidated integrated common bean anthracnose management approaches in Africa. Tricot is an important approach to control anthracnose and evaluate anthracnose-resistant varieties, but is not widely used in Africa. Future studies in other African countries can complement tricot research already underway on common bean anthracnose in Tanzania, for increased productivity, nutrition and income.

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Conceptualization ELK, ERM, validation PV, TMA, JCN, investigation and resources ELK, ERM, PV, JCN, TMA, CMM, JCR, writing review and editing all authors' visualization ELK and funding JCR. All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability

Information presented in this study will be available uppon request to the corresponding author

Ethics Approval

Not applicable

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