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Chapter 3

# Cassava Superelongation Disease in the Caribbean

Angela T. Alleyne

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**Abstract**

An important economic constraint to the growing cassava industry in the Caribbean islands is the disease caused by the fungal pathogen *Sphaceloma manihoticola*, synonym *Elsinoë brasilensis* (Bitancourt & Jenk). One hundred percent incidence has been recently observed on some farms in the Caribbean islands. The fluctuation in individual farming practices such as lack of fertilizing and irrigation schemes may play a role in the level of health and disease resistance of the plants, which in turn may affect the severity of the disease and levels of incidence. Severe elongation may be seen of the internodes in mature plants but primary symptoms include small yellow leaf spots, leaf curling, stem and petiole scab-like lesions and defoliation. The use of disease-free planting material, fungicide pre-treatment of nodal stem cuttings and germplasm maintenance of *in-vitro* stocks of high performing varieties is suggested. However, new molecular tools for disease diagnosis and analysis of the pathogen population dynamics are required to adequately manage the disease in the region.

**Keywords:** cassava, super-elongation, gibberellin A4

## 1. Introduction

*Manihot esculenta* Crantz (cassava)—a woody shrub of the family *Euphorbiaceae* native to South America, is extensively cultivated as an annual crop in tropical and subtropical regions such as West Africa, Southern and Central America, and South East Asia [1, 2], for its edible, starchy, tuberous root. While production showed increased growth in Africa and Asia in the 1990s, production levels of cassava in Latin America and the Caribbean were relatively stable [2]. Since 2005 however, Latin America and the Caribbean has seen a resurgence in cassava production as the implication of threats to food security have emerged and new markets for secondary cassava products continue to grow and develop [2]. Consequently, cassava has been recognized as a diverse crop for development of primary and secondary agricultural products

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in the Caribbean region. From 2004 to 2014, cassava production has increased from 20 to 400% from Trinidad and Tobago in the south to Bahamas in the north (**Table 1**) [3]. However, with increased production of cassava, Superelongation disease (SED) has re-emerged as a significant threat to its cultivation in the region [4, 5].

Superelongation disease of cassava is caused by the fungal pathogen *Sphaceloma manihoticola* (Bitancourt & Jenk) synonym *Elsinoë brasilensis* and has been responsible for crop losses of up to 80% in several Latin American countries, such as Columbia, Brazil, Venezuela [4, 6, 7]. It is therefore considered to be of major of economic importance due to its potential to severely lower yields [8, 9].

The genus *Sphaceloma* de Bary (Melanconiales) is composed of over fifty fungal species [6, 10]. The majority of the species thrive in tropical or subtropical regions. Conidia are small, unicellular and hyaline, formed either in an acervulus-like structure or on continuous fertile layers of densely packed phialidic conidiophores [6, 11–13]. Different species have the ability to form large spindle-shaped, septate spores which may be pigmented with a thick cell wall. This characteristic growth has been referred to as the “fawcetti” conidia and allows it to be carried long-distances by the wind, aiding in dissemination of the pathogen [7, 14, 15].

Normally cassava is planted from stem or nodal cuttings known as stakes, which are at least 10 months old [16–19]. The cuttings should be healthy; otherwise the plants produced will bear diseases that were infecting the stake. The cultivation of cassava through the planting of disease-free stakes is therefore designed to reduce the effect of potential diseases such as SED [20].

In the English speaking Caribbean islands, there are several local varieties of cassava grown; with growers in each island having their particular preference; these include local names and descriptions such as: Sugarloaf, Butterstick, Redstick, Maracas Blue Stick, Maracas Black Stick, Green stem, and Guyana Sweet among others [19]. However, improved cassava varieties obtained from the International Centre for Tropical Agriculture (CIAT) are generally higher yielding and have been bred and maintained *in vitro* for tolerance to particular pests and diseases such as bacterial blight, anthracnose, SED, and thrips [6, 19]. Despite the introduction of improved cassava varieties, it was observed that in Brazil many of these resistant

| Country | Productivity per year in tonnes[a] | | |
|---|---|---|---|
| | 2004 | 2008 | 2014 |
| Bahamas | 155 | 413 | 938 |
| Barbados | 317 | 466 | 553 |
| Trinidad | 575 | 2746 | 3194 |
| Jamaica | 16,758 | 14,991 | 16,549 |

[a]Data taken from FAOSTAT 2014. FAO Database, Food and Agriculture Organization of the United Nations. Rome, Italy. http://www.fao.org/faostat/en/

**Table 1.** Growth in cassava production from 2004 to 2014 among three Caribbean islands.

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varieties are not adopted by the farmers, so these benefits may not be transferred to farmers who may have their own criteria for choosing cassava planting material [21]. A similar response to varieties exists in some Caribbean islands. Interestingly, farmers may also lack knowledge of the scientific names and nomenclature of a specific variety being planted, being only familiar with a common description which remains in the local discourse.

This preferential planting of cassava varieties by the farmer therefore narrows the genetic base, resulting in genetic erosion [22] and increases exposure to endemic diseases such as SED, which is prevalent in the region. Tracking of cassava varieties for cultivar identification through the use of genetic markers [22, 23] is therefore currently a necessary exercise for the cassava industry in the Caribbean islands.

## 2. Disease description

Epidemics of SED result in reduced root size and poor quality tubers, besides dramatic yield losses [7, 19]. An outbreak of the disease was reported for the first time in the Tolima Valley of Colombia in 1972 and 1976 [24]. In 1994, SED was reported in Brazil in cassava crops near Manaus, Maues in the Amazon region [7]. Superelongation disease was also reported in Brazil again in 1994 in Sao Paulo where the disease was observed in cassava crops in the Valle de Paranapanema [25]. In 2007, the disease was observed on cassava in fields of north central and southern Trinidad and Tobago [4] and is also widespread in Barbados [5], the Dominican Republic and Panama [4]. In an island wide survey of 2015–2016 in Barbados, an overall incidence of SED of 72–88% and severity levels of approximately 49% was seen on the island.

The disease affects the leaves, petioles and stems of the cassava plant. Early symptoms of the disease appear on expanding leaves, juvenile stems and floral tissues. Leaf spots are chlorotic and present as small, circular to irregular discolorations, approximately 0.5–5 mm in diameter, lightly colored and sometimes necrotic with a yellow halo [4, 7, 26]. Abundant spots may eventually deform the leaf causing sharp curvatures of one or more of the leaf lobes. This leaf curl causes the lower leaf surface to face upwards, resulting in severe defoliation [19]. Infrequently, chlorotic spots with necrotic centers are seen on the leaf lamina which when dried, produce a "shot hole" appearance. In the later stages of the disease, raised corky cankers appear as lesions on the petioles, leaf veins, and stems [5, 7, 13]. The stem cankers are usually hypertrophic which may coalesce to produce large elliptical to fusiform lesions [26] (**Figure 1**).

Secondary and advanced symptoms from which the disease takes its name, comprise of exaggerated internode elongation in severely infected young stalks, in susceptible cassava cultivars [4, 7, 12, 25]. Rapid elongation caused by SED results in weak plants because the stem is unable to support mature healthy growth, and is frequently followed by die-back and extensive apical defoliation. Internode elongation is prevalent in seasons during which cassava is actively growing. Therefore, during the dry season internodes do not usually elongate, even if numerous stem lesions are present [12].

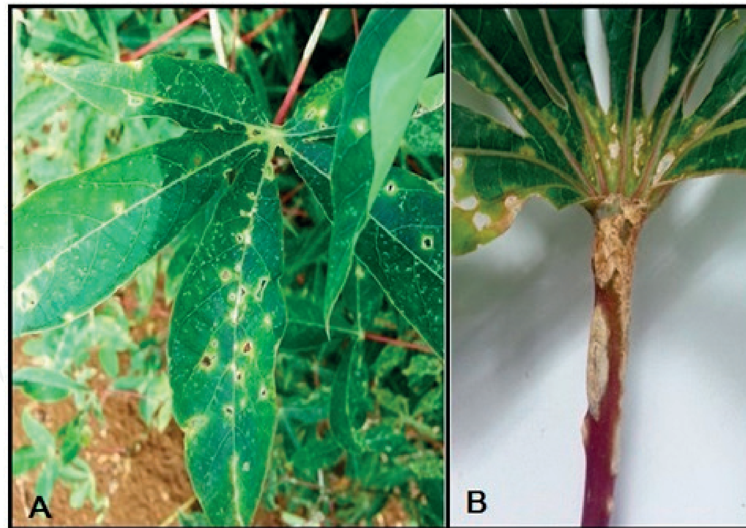


**Figure 1.** Symptoms of superelongation disease of cassava caused by *S. manihoticola*. (A) Yellow discolorations and spots necrotic lesions surrounded by a yellow halo and (B) fusiform scab-like lesions on cassava petiole.

Thus SED is considered a disease with economic impact because it not only affects the crop but the planting material [7, 13]. The stem elongation observed is a result of the over production of Gibberellins, specifically gibberellin A4 ($GA_4$), produced by the fungus *S. manihoticola* [7, 15, 26, 27].

## 3. *Sphaceloma manihoticola*: the pathogen

*Sphaceloma manihoticola* when cultured exhibits varied morphological characteristics depending on the media it is grown on [6, 7]. *S. manihoticola* was first observed and documented by Bitancourt and Jenkins [14], when they observed the pathogen on cassava and considered it a new species based solely on its symptomology and host species. Fungal colonies are usually observed as a yellow mucoid mass but there may be other colors such as black and orange, all dependent on the strain and growth conditions [6, 7]. *Sphaceloma manihoticola* has such variable colony morphology that Zeigler et al. [26] considered this feature to be limited in its usefulness to distinguish species.

The morphology of young colonies ranges from a yeast-like growth of budding, unicellular propagules and short hyphal strands to a distinctly mycelial form [10, 28]. It was noted that as colonies matured, they became raised, convoluted and bound in a gelatinous matrix. The fungus is known to synthesize large amounts of exopolysaccharides which account for the difficulty of removing the mycelium from the culture media [29]. The colonies studied by Zeigler and Lozano [13] were pulvinate or raised and deeply fissured, gummy to occasionally mucoid on agar media. Colony color ranged from orange to yellow or orange to bright red, rust and brown on Potato Dextrose agar (PDA) [6]. Yellow or orange colonies frequently formed small red sectors [6] and colony color changed based on the growth medium used. Cassava leaf agar supplemented with glucose generally produced a mixture of bright red, orange and black

colonies, while the same colonies on Czapek Dox agar (CZA) were uniformly orange in color and produced no aerial mycelium [13]. Another study on PDA showed colony color from nearly purely white through yellow to deep reddish purple and black [26].

Reeder et al. [4] consistently isolated *S. manihoticola* from infected planting material by plating on potato carrot agar. The colonies that formed were slow growing, pulvinate, fissured and bright red to tomentose in color. Conidiophores were phialidic, conidia were hyaline, non-septate and ellipsoid forming a continuous layer [4]. Morphology of the pathogen as previously observed by Zeigler [10] and others [7, 25] is therefore highly diverse.

Four different morphologies were observed when isolates were grown on CZA in Barbados (**Figure 2**). The range in colors is due to the presence of elsinochromes which are red/orange pigments that are produced by *Elsinoë* spp. and *Sphaceloma* spp. [30]. These pigments have been shown to vary based on the available nutrients in the growth medium such as glucose [28]. Elsinochromes contain perelenequinone which is a non-host virulent factor and causes lesions during fungal infections such as in citrus scab [30]. These pigments cause lipid peroxidation and electrolyte leakage into the infected leaves [31]. The presence of this phytotoxin containing pigment in *S. manihoticola* and its interaction with reactive oxygenic species may account for the lesions and necrosis observed in SED. In addition, given that these pigments were expressed on CZA, in which sucrose is the carbon source, suggests that pigment production might have been stimulated by that carbon source. Further studies are therefore necessary on isolates in the Caribbean islands to explain their yellow pigment.

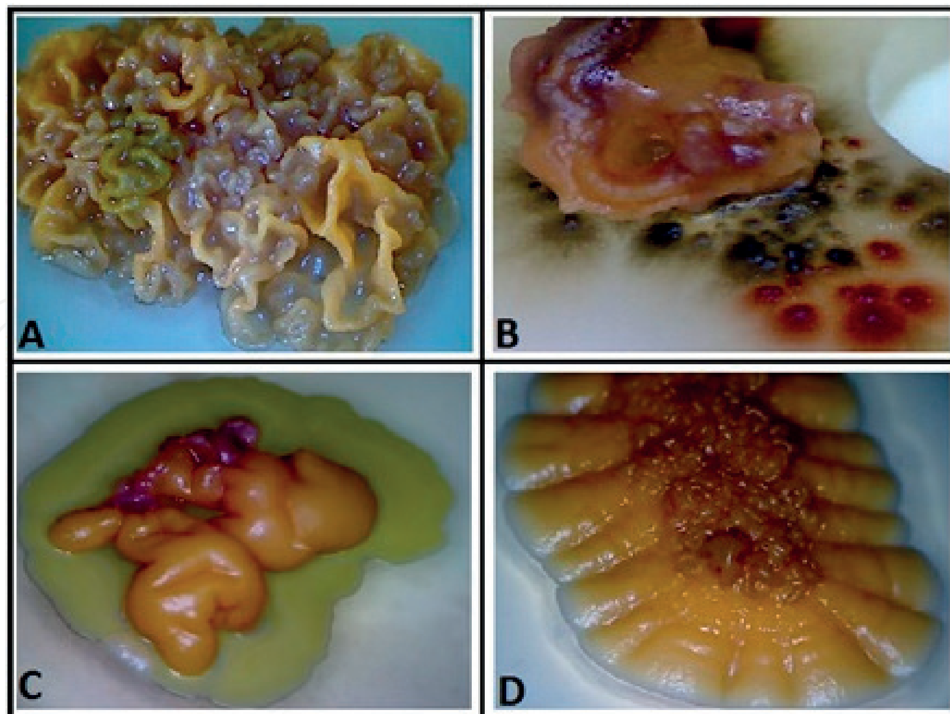


**Figure 2.** Gross morphology of *S. manihoticola* on various agar media: (A) Czapek Dox agar, (B) Cassava Leaf agar supplemented with Glucose, (C) twenty-eight day colony on Potato Dextrose agar, (D) Potato Dextrose agar.

## 4. Gibberellin A4

Gibberellins are a group of at least 136 different diterpenoid compounds of plant or fungal origin [32, 33]. These molecules are synthesized from acetyl CoA via the mevalonic acid pathway.

The production of gibberellin $GA_4$ by *S. manihoticola* promotes the growth and elongation of cells, stimulates rapid stem and root growth, induces mitotic division in leaves and increases seed germination rate [27, 29, 34]. Thus, the symptoms of SED are a result of the production of $GA_4$ by *S. manihoticola* [7, 26, 29]. In standard incubations it was determined that the wild-type strain of *S. manihoticola* (DSM1638 from the Leibniz Institute DSMZ-German Collection of Microorganisms and Cell Cultures) produced $GA_4$ in concentrations of up to 7 mg per liter of culture filtrate. *Sphaceloma manihoticola* does not produce the commercial gibberellins of $GA_3$, $GA_1$ or $GA_7$ which are also synthesized by *F. fujikuroi*, thus with $GA_4$ being the main gibberellin produced by *S. manihoticola* it can be isolated in a pure form from culture filtrates of the pathogen [26, 27, 35].

Although plants and fungi produce structurally identical gibberellins, the biosynthetic steps in the pathway for the formation of gibberellins differs significantly [36]. A major contrast in the biosynthetic pathway is the stage at which the hydroxyl groups are introduced. Fungal gibberellic acid biosynthesis requires only cytochrome P450 monooxygenases, while the formation of plant gibberellic acid requires both membrane bound cytochrome P450 monooxygenases and soluble 2-oxyglutarate dependent dioxygenases [32]. In *S. manihoticola* the production of gibberellic acid is controlled by the GA biosynthetic gene cluster.

The gene cluster has been characterized and consists of the genes *SmP450-2* (AM886290.1), *SmP450-1* (AM 886288.1) and (AM 886289.1) *SmP450-4*. These three genes are surrounded by two open reading frames; the bi-functional ent-copalyl diphosphate synthase/ent-kaurene synthase and geranylgeranyl diphosphate synthase [5, 29] (**Figure 3**).

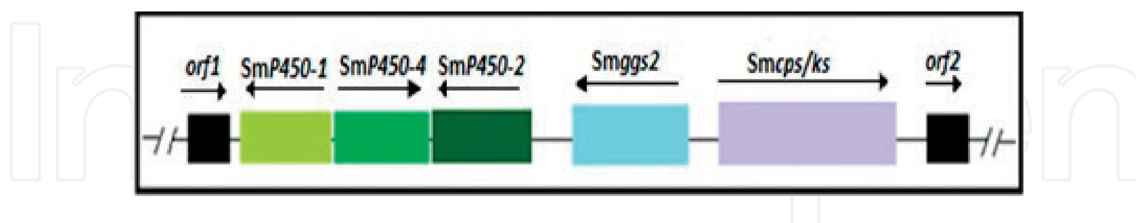


**Figure 3.** The gibberellin biosynthesis gene cluster of *S. manihoticola* (adapted from Bömke et al. [29]).

## 5. Detection of Superelongation disease in cassava

Classic methods used in the identification of pathogen infections depend on the observations of seasonally variable elongation and subsequent laboratory confirmation by fungal growth of cultures. However, this can take weeks due to the slow growing nature of the pathogen [5, 13]. With SED, because symptoms are variable and may depend on environmental conditions, visual

observations of these symptoms alone are not a reliable indicator of SED infection [12]. For example, although in Trinidad a high incidence was recorded in 2008, elongation of the internodes was not observed [4]. This was also apparent in Barbados in 2014, but hyper-elongation was evident in several cassava growing fields severely infected with *S. manihoticola* in 2015 [28]. Krausz [12], stated it is not peculiar for plants affected with SED not to exhibit elongation of internodes especially in the dry season. Therefore, variability in the consistency and severity of secondary symptoms of the disease decreases the reliability of visually confirming the disease by field observations alone.

Currently there is a disease severity scale based on symptom appearance in infected cassava. The scale was introduced by the Cassava Pathology Program at CIAT [24]. Infected cassava plants are assigned a numerical value for disease severity. As signs and symptoms of SED increased in plants, they are assigned a higher numerical value correlating with the symptoms presented, e.g., a value of 1 is assigned to plants that have no sign of SED, 2 is used for the development of spots or cankers on leaves or petioles, 3 used for signs of cankers on leaves, petioles and stems with severe leaf distortion and 4 is assigned to plants displaying elongation, cankers on leaves, petioles and stems, severe leaf distortion and scorching [20]. Additionally, the use of molecular techniques such as polymerase chain reaction (PCR) has the ability to surpass many of the shortcomings of measuring the disease severity using a disease rating scale. These methods present advantages of being specific, accurate and are faster than traditional techniques.

For analysis of the gibberellic acid gene cluster organization in *S. manihoticola* Bömke et al. [29] synthesized SMP primers. The SMP primers developed were specific for particular SMP transposons responsible for the regulation of the gibberellic acid gene cluster organization. Bömke et al. [29] utilized the SMP primers in order to characterize the gibberellin biosynthetic cluster in *S. manihoticola*. However, the SMP primers were not used to characterize the fungus, *S. manihoticola*. Since then, primers have been developed for amplification of the *SmP450-2* gene (Gen Bank Accession AM 886290) which serves for detection of SED in asymptomatic field and artificially inoculated cassava leaves, with SED [5]. They have also been used to quantify varying disease severity levels in cassava leaves and stem lesions [37].

In 2000, Alvarez and Molina characterized *S. manihoticola* by targeting the internal transcribed spacer (ITS) region of ribosomal DNA using the PCR primers: ITS4 and ITS5 [25]. Brazilian isolates of *S. manihoticola* could also be distinguished from those infecting milkweed and RAPD molecular markers designed for SED were able to distinguish variation in the pathogen population from South central Brazil and Colombia [38]. They suggested that pathogen variation may be determined by geographic location and even smaller locales such as a municipality, indicating centers of pathogen diversity [38].

Despite these established techniques to estimate disease severity and identify the disease, pathogen quantification remains one of the main challenges in the disease management of crops and moreso in SED in cassava.

High quality cassava planting material has a key role in the maintenance of genetic purity and plants free from pathogens and disease. Cassava farmers are constantly faced with the issue of generational build-up of diseases and pathogens through the use and reuse of infected planting material.

The differences in morphological diversity among isolates of the pathogen when cultured on varying media, and the use of molecular markers to identify the pathogen might also suggest that molecular differentiation is present in the pathogen population in the Caribbean islands, as suggested for Colombia and Brazil [6, 38].

## 6. Disease resistance

Both the SPM-1 and SPM-9 primer pairs designed from the gene *SmP450-2* [5] demonstrated the capability for early detection of the *SmP450-2* in the local varieties Butterstick, White stick, Red stick and the CIAT cultivar CM6604, 24 hours post-inoculation [28]. Contrastingly, the SPM-1 and SPM-9 primers did not detect the *SmP450-2* gene until 14 days' post-inoculation in the MCOL-22 and BRA-383 CIAT cassava varieties [28]. This variability in detection of the *SmP450-2* gene by the SPM primers could be applicable to investigate susceptibility and resistance among local varieties. Thus, the cassava varieties in which the SPM primers detected the expected amplicon 24 hours post-inoculation were possibly more susceptible to SED infection.

The CIAT library of cassava cultivars describes the BRA-383 cultivar as a landrace derived from Brazil and commonly referred to as, "Vassourao." The BRA-383 variety grows to a height of 200 cm and has linear-pandurate leaf lobes with white roots. Titus et al. [19] in their report on commercial cassava production described BRA-383 as being susceptible to SED infection. Additionally, the MCOL-22 variety, also known as "Uvita" originated from Colombia and was described to have white roots and grows to a height of 150 cm with the leaf lobes being straight or linear. Interestingly, the MCOL-22 variety has also been recognized as susceptible to SED infection [6], but is widely cultivated in some islands.

Although both the BRA-383 and the MCOL-22 varieties have been previously described as susceptible to SED in the region, investigations using the *SmP450-2* gene as a determinant of SED susceptibility demonstrated both varieties expressed the GA4 gene much later compared to the other local varieties examined. This may suggest differential susceptibility in local varieties when compared to well-described CIAT varieties. Zeigler et al. [6] examined several morphological features of cassava that could correlate with SED resistance and suggested that stem and leaf cuticle could account for resistance, because the water-borne inoculum cannot adhere to susceptible leaf tissues with thick cuticles. Physiological characteristics such as plant architecture, apical maturity and growth rate were also suggested to affect the resistance of cassava varieties to SED infection [6]. Thus, it is possible that the cassava varieties demonstrating susceptibility to SED infection in the Caribbean islands have physiological characteristics which aid or support in *S. manihoticola* infection.

It is also possible that the local varieties in the Caribbean islands are no longer the same varieties as described by the CIAT database. This raises the question for further investigation into the origins and characteristics of currently disseminated cassava stakes. Locally it is known that mixing and exchange of cassava stakes among farmers are common practices in most farming communities and could result in mis-identification of cassava varieties. Traditionally, tolerant varieties against SED such as Mex 55 and Mex 23 which were imported from CIAT are no longer tolerant to SED in Barbados, and are no longer distinguishable from locally grown

varieties [19, 20], this could further account for the consistency observed in early detection of the *SmP450-2* gene in susceptible local varieties. Studies are ongoing for the resistance performance of current CIAT varieties in the island.

The presence of cassava varieties showing such a high level of susceptibility to SED within the region could be the result of weak pathogenic specialization due to crossing two highly resistant cassava varieties with high physiological resistance. The result is a loss of modifying factors ultimately causing the progeny to have traits which are likely to be overcome by the highly variable pathogen [6].

## 7. Disease management

In cassava production, and farming in general, healthy planting material is the first step to having a disease-free crop. Farmers sometimes plant more than one variety of cassava per plot and the different varieties also have differences in disease resistance. Over 20 cassava varieties have been shown to express a level of resistance to Superelongation [39].

The most effective means of preventing infection and the spread of SED in cassava is by the planting of disease-free stakes in areas where weeds are scarce [19]. As a precautionary measure, stakes can be treated with a broad spectrum fungicide [19, 20]. In areas where SED is endemic, plants affected by the disease should be removed from the field and burnt. In the Caribbean islands, research by Chandler [20] produced a fact sheet indicating that to eradicate SED, Captafol® ($C_{10}H_9Cl_4NO_2S$) (Chevron™) at 400 ppm could be used as a dip for pre-treating planting stakes. Recommended weed treatment included: pendimethalin (Herbadox®, BSAF, Chile) at 4.5 liters per hectare (ha) or pendimethalin (Herbadox® 45 CS) at 4 liters per ha together with diuron (Karmex®DF (DuPont™)) at 1.5 kg per ha, or Karmex®DF at 1.5 kg per ha together with alachlor (Lasso™) at 3 liters per ha. The recommended practice was to inter-row directed sprays with Gramoxone® (Syngenta™).

Titus et al. [19] conducted research on hot water treatment of cassava as a method of disease management. The treatments used in the study included: thermotherapy, stem cuttings placed in water bath at 49°C for 49 minutes; stem cuttings immersed for 5 minutes in the commercial fungicide Kocide® 3000 ($Cu(OH)_2$) (DuPont™) and stem cuttings immersed for 5 minutes in the commercial fungicide Score® ($C_{19}H_{17}Cl_2N_3O_3$) (Syngenta™). The research determined that the treatment using Kocide® recorded the lowest average AUDPC score of 6.3.

In addition to SED being spread via infected stake cuttings, there has been increasing emphasis on the role of weeds as hosts of *S. manihoticola* infection. Chandler [20] described weed control as being critical during the first few months before the lead canopy closed over. The most common weeds occurring in cassava fields are grasses such as: *Imperata cylindrical* (Spear grass), *Cynodon dactylon* (Bermuda grass); *Panicum maximum* (Guinea grass), and *Pennisetum polystachion* (feathery pennisetum) [40]. *Sphaceloma manihoticola* and *Elsinoë* species are also common pathogens on weedy and ornamental plants related to cassava in Central and South America. These species of plants affected by *S. manihoticola* include *Jatropha curcas* (physic nut), *Jatropha aconitifolia* Muell., *Manihot glaziovii* (ceara rubber), *Euphorbia pulcherrima* Willd., and weeds such as *Euphorbia brasiliensis*, *Euphorbia heterophylla* L., *Euphorbia hypericifolia* L., and *Euphorbia prunifolia* [6, 41–43].

There are multiple challenges to integrating effective and economical weed control. Rapidly growing weeds in cassava farms will cover the ground almost completely and utilize the nutrients and water from the soil, limiting these materials for cassava plant growth [42]. Weed growth results in shading of the cassava plant and therefore decreases sunlight exposure to young cassava plants. Therefore, it is almost impossible to remove the weeds without affecting the growing cassava.

Moreover, observations of SED infections in Costa Rica occurring immediately after planting in grassy fields may further suggest the possibility of grass species being alternate hosts of *Sphaceloma* species (Alvarez, E. personal communication).

Bermuda grass is one of the most common weeds found in cassava production [44] and this weed among others harbors pests that later infect cassava. Bermuda grass has been recently found to be a reservoir host of *S. manihoticola* in Barbados by using the *Spm1* molecular marker [45]. *Cynodon dactylon* may therefore be a potential reservoir host of *S. manihoticola* in the Caribbean.

One mechanism by which fungi spread, even globally, is via wind so at a local level since tomentose spores are observed in *S. manihoticola* they may easily be carried by the wind from the Bermuda grass to cassava found nearby.

A combination of these factors satisfies the parameters of a disease triangle; cassava fields throughout the island—the host, a weed that harbors the pathogen and others that weaken the plants' robustness and suitable environmental conditions favorable to pathogen growth. This finding of alternate hosts is therefore important in improving disease management of SED in local communities in the Caribbean islands.

## 8. Conclusion

As cassava production has increased in the region the impact of SED has also risen. The long-term consequences for productivity and success of the cassava industry are yet to be measured. Disease mitigation strategies are use of clean stakes as planting material, use of SED or tolerant resistant varieties, weed control, crop rotation, and fungicide treatments. Disease awareness in cassava cropping area is also required so continuous famer training in disease recognition is important in mitigating the devastation of this disease in cassava planting areas, especially in the Caribbean islands.

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