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Development and screening of Fusarium wilt resistant lines in Sweet potato [*Ipomoea batatas* (L.) Lam]

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Abstract Fusarium wilt is among the major soil borne fungal diseases hindering sweet potato production in temperate regions of the world. Resistant cultivars are important for commercial production as the impact of Fusarium wilt is aggravating due to intensifying climate variability, thus the need for quick screening methods. The objectives of this study were to test a quick screening method at the early growth stage, to identify resistant parents, and to screen resultant progenies for resistance under glasshouse and field conditions. Virulent isolates of *Fusarium oxysporum* f. sp. *batatas* (PPRI15907) and *F. oxysporum* f. sp. *vanillae* (PPRI9458) were used. The soil was artificially inoculated and planted with month-old sweet potato plantlets. Visual leaf and stem disease assessments were recorded at 6 weeks after planting using an ordinal rating scale (0–3). Both leaf and stem disease assessment results indicated significant variation ($P \leq 0.05$) in the disease reaction of the genotypes. Based on stem disease severity index (DSI) as determined from the quick screening, Bonita (4.2%) and Monate (8.3%) were classified as resistant, and Blesbok (66%) and Lethlabula (81%) as highly susceptible parents. Furthermore, DSI categorized the 92 progenies into 44 resistant, 21 moderately resistant, 13 moderately susceptible, seven susceptible and seven highly susceptible genotypes. The field experiment confirmed that 84% of genotypes that were identified to be resistant to Fusarium wilt under glasshouse conditions, were also resistant under field conditions. Thus the glasshouse method is reliable, efficient, and can screen large numbers of sweet potato genotypes for resistance against virulent *Fusarium oxysporum* isolates.

Keywords Disease screening · *Fusarium oxysporum* · Sweet potato · Wilt disease

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Introduction

A greater challenge facing agricultural production in many parts of the world is to produce high quality foods in high enough quantities that are rich in vital micronutrients, and that are able to meet the daily requirements and sustain a healthy diet (Bouis et al. 2013). Sweet potato (*Ipomoea batatas* L. Lam) is a climate resilient world staple crop that fits into many farming systems (Mwanga et al. 2021). Increased attention has been placed on sweet potato in the past

four decades due to its ability to secure food and nutrition in the developing countries, especially in Africa and Asia, where malnutrition and food insecurity is prevalent. Sweet potato is the 7th most important food crops in the world and the 5th as a food crop in developing countries (FAOSTAT 2018). In South Africa (SA), sweet potato is known as a household, traditional and indigenised commercial crop (Laurie et al. 2015).

Fusarium wilt disease is widespread in temperate sweet potato production regions of the world and is particularly severe under warm (28–30 °C) and dry conditions (Clark and Moyer 1988; Ames et al. 1996). *Fusarium oxysporum* f.sp *batatas* (Wr.) Snyd. and Hans] is the major causal agent and the chlamydospores, the main survival structures, can persist in the soil for many years (Clark and Moyer 1988; Clark et al. 2013). Symptoms include stunting, yellowing and necrosis of older leaves, and stems showing a characteristic reddish-brown discolouration of tissues when cut open, and wilting or death of the whole plant (Brayford 1992; Michielse and Rep 2009). Epidemiological studies of the causal pathogens suggested that the fungi invade the plant through root tips and wounds of the roots, which can cause wilting through colonization of xylem tissue in the soil (Agrios 1988; Koyyappurath et al. 2015). Knowledge regarding biological and chemical control strategies of *Fusarium* wilt of sweet potato is limited.

High occurrence of Fusarium wilt can compromise up to 50% of the yield in sweet potato (Lebot 2009). Wilt disease of sweet potato is of economic importance in South and Central China, Korea and South eastern USA (Lin et al. 2017; Paul et al. 2020). The use of resistant cultivars has been reported as effective in controlling this fungal disease in the USA (LaBonte et al. 2008, 2011; Jackson et al. 2010; Lin et al. 2020). Studies confirmed that Fusarium wilt resistance in sweet potato is of quantitative inheritance and the crop has a high number of chromosomes, therefore, it is essential to evaluate a large and diverse number of genotypes (Collins 1977; Chang et al. 2009). It has been shown in crops such as pea (Bani et al. 2012), lentil (Stoilova and Chavdrov 2006), chickpea (Mirzapour et al. 2014), banana (Nel et al. 2006), melons (Burger et al. 2003) and cotton (Lopez-Lavellea et al. 2012) that glasshouse methods can be effective for resistance screening in diverse germplasm and breeding populations. However, in sweet potato use of glasshouse screening methods are not common.

In South Africa, the gross value of sweet potato production is estimated at approximately USD19.3 million (DAFF 2019). Fusarium wilt disease is a significant fungal disease locally and natural infection of *F. oxysporum* f. sp. *batatas* occurs in major parts of sweet potato production areas when environmental conditions favour disease development (Thompson 2011). Dau (2017) found that *F. oxysporum* f.sp *vanillae* also causes wilting in sweet potato. *F. oxysporum* f.sp *vanillae*, also known as *Fusarium batatatis* var. *vanillae* Tucker, is the causal agent of root and stem rot disease in vanilla (*Vanilla planifolia*) plants (Koyyappurath et al. 2015; Pinaria et al. 2015). Laurie et al. (2016) have indicated that in South Africa, since some of the major sweet potato cultivars grown commercially lack resistance to wilt disease, a disease-indexed scheme is operated by the Agricultural Research Council. A significant variable interaction between *F. oxysporum* spp. isolates and the South African sweet potato cultivars were reported under field conditions (Dau 2017). This variability allows the possibility to identify Fusarium wilt resistant sweet potato genotypes by artificial inoculation techniques in the glasshouse.

The objectives of this study were to test a quick screening method at the early growth stage, to identify resistant genotypes against the prevalent virulent *F. oxysporum* isolates, and to screen resultant progenies for resistance under glasshouse and field conditions.

Materials and methods

Culture and inoculum preparation

Two virulent isolates of *F. oxysporum* f. sp. *batatas* (PPRI15907) and *F. oxysporum* f. sp. *vanillae* (PPRI9458) were obtained from the Crop Protection Division, ARC-VIMP. The isolates were collected from a Fusarium wilt infected field and identified by Dau (2017). The initial screening of parental cultivars was done with *F. oxysporum* f. sp. *batatas* (PPRI15907) during the 2014 production season in repeated experiments. However, in 2015 the parents were tested with a mixture of *F. oxysporum* f. sp. *batatas* (PPRI15907) and *F. oxysporum* f. sp. *vanillae*

(PPRI9458) isolates. The addition of PPRI9458 was based on pathogenicity testing by Dau (2017) indicating that isolate PPRI9458 was highly pathogenic. Due to the co-existence of more than one isolate in field collections (Dau 2017), it was advisable to use a combination of pathogenic isolates. Both isolates came from the infected field and therefor had an additive pathogenicity effect when mixed.

The isolates were grown in separate petri-dishes on half-strength Potato Dextrose Agar (PDA) in order to obtain fresh fungal cultures for the preparation of the inoculum. Cultures were then placed under cool-white and near-ultraviolet fluorescent light at 24 °C until fully grown. When the mycelium was ready, inoculum was prepared according to the method described by Nel et al. (2006) using millet seeds, with single or a mixture of *F. oxysporum* f. sp. *batatas* and *F. oxysporum* f. sp. *vanillae* isolates. Polyethylene bags containing 150 g of millet seeds (*Panicum miliaceum* L.) with 200 ml distilled water were autoclaved for 15 min, and inoculated with a 0.5 cm diameter mycelial plug with a mixture of *F. oxysporum* f. sp. *batatas* and *F. oxysporum* f. sp. *vanillae* isolates. Uninfected same-sized pure PDA media were cut and used for the control treatments. Infected and control seeds were placed under cool-white and near-ultraviolet fluorescent light for fungal colonization for 10–14 days.

Screening of parental cultivars

Two cultivars reported to be resistant to Fusarium wilt in the USA, namely, Bonita and Murasaki (LaBonte et al. 2008, 2011), were imported from Louisiana State University (LSU) AgCenter and integrated into the ARC-VIMP sweet potato disease-indexed collection during 2010–2011. In addition to the two imported cultivars, local cultivars, such as Blesbok (susceptible), Bosbok, Lethlabula, Mvuvhelo, Ribbok, Mvuvhelo, Monate and Ndou were used in this study. Cuttings of the cultivars were multiplied in seedling trays at a day/night temperature regime of 28/20 °C for 6 weeks in a controlled glasshouse at the ARC-VIMP in Roodeplaat (25°59′ S 28°35′ E), Pretoria, South Africa. Plantlets were transplanted to steam-pasteurized (Mini Electrode Steam Generator, Model: M60, Marshal and Fawler) red soil with vermiculite in ratio 8:5 mixed with 10 kg of compost. Before planting, 1 kg soil was artificially inoculated by thoroughly mixing 30 g of millet seeds colonized by *F. oxysporum f. sp. batatas and F. oxysporum f. sp. vanillae*. Six weeks old plantlets were removed from the seedling medium and the roots washed with tap water. Then the plantlets were transplanted directly into the 7 cm pots containing the *F. oxysporum* infected soil. The experiment was arranged in a randomized complete block design (RCBD) with four pots (four replicates) for both infected and control treatments. The pots were spaced 10 cm apart in rows and the plants were kept in a glasshouse at a day/night temperature regime of 28/20 °C for 6 weeks.

Disease assessment

Disease development was recorded based on leaf and stem visual observations (Bani et al. 2012). Plants were monitored daily for typical *Fusarium* wilt disease symptoms on the leaves starting from the second day after planting. Disease incidence was recorded at the 6th week by observing both leaf and stem symptoms arising from artificial inoculation. An ordinal rating scale (0–3) was used, where: 0 = stem healthy/no disease symptoms; 1 = slight browning of stem tissues; 2 = distinct reddish-brown discolouration of stem tissues and wilting/death of plants; 3 = stem blight/stem dieback (Pers. Comm. Alistair Thompson, ARC-VIMP). To observe the dark discolouration of vascular tissue, the base and middle part of the stem was cut sideways into the vascular tissue above the soil surface. Disease reaction was calculated based on stem symptoms to distinguish resistant and susceptible cultivars. The disease severity index (DSI) was calculated according to Hossain et al. (2010).

$$\text{Disease severity index (DSI)} = \left(\frac{\textit{Sum of all diseased rating''}}{\text{Total number of rating} \times \text{Maximum disease grade}} \right) \times 100$$

(where total number of rating = 4; Maximum disease grade = 3).

Hybridization

During the 2013/2014 and 2014/2015 seasons, crosses were made between the imported resistant cultivars (Bonita and Murasaki) and six local cultivars (Bosbok, Lethlabula, Mvuvhelo, Ribbok, Mvuvhelo, Monate and Ndou) in a line × tester crossing scheme in a glasshouse. Parents were cleft grafted onto 6 weeks old *Ipomoea setosa* plantlets to induce flowering (Rossel et al. 2008). Flower buds were clipped with paperclips in the afternoon to avoid cross contamination by unwanted pollen grains and emasculation and pollinated the next morning. The stigma was covered with a 4–5 mm piece of plastic straw, stapled on one end, to prevent unwanted pollination. The number of crosses made per combination was dependant on the availability of flowers and varied from 11 to 50. Bonita was compatible with all the local cultivars, while Murasaki was only compatible with Monate. Ripe seed capsules were harvested manually 4–5 weeks after pollination. Seeds were stored in manila envelopes in a cold room at 7 °C. The seeds were scarified with concentrated sulphuric acid.

Glasshouse screening of progenies

A total of 92 progenies derived from controlled crosses, as well as the susceptible control cultivar Blesbok were subjected to the glasshouse screening (Fig. 1) using a mixture of *F. oxysporum* f. sp. *batatas* (PPRI15907) and *F. oxysporum* f. sp. *vanillae* (PPRI9458). The experiments were conducted across two seasons (2015 and 2016). The same screening method and disease assessment described for screening of parental cultivars were used.

Field screening of progenies

Seventy-four promising progenies selected from the glasshouse screening, together with the parental cultivars and the susceptible cultivar Blesbok were screened in a field known for natural Fusarium wilt occurrence. The experiment was conducted at Roodeplaat (25.604° S, 28.345° E), Pretoria, South Africa during the summer of 2016/17 at an average maximum/minimum temperature of 28/10 °C. Nitrogen (N) at a rate of 120 kg ha^{-1} and phosphorus (P) at a rate of 20 kg ha^{-1} was applied as per fertilizer recommendation. The area received 278 mm of rainfall during the trial period and supplementary irrigation was also applied. Herbicides, Eptam® and Avalon® were applied before planting at a rate of 3.5 L per hectare and 1.8 L per hectare, respectively. The experiment was laid out in a latinized design with three replications consisting of 45 columns/plots and three rows per replicate. An inter-row and intra-row spacing of 1.2 m and 0.5 m, respectively, were used. No pesticides and fungicides were applied during the growth period.

Disease assessments were made at 20 weeks after planting using the same rating scale as described under the disease assessment section. Lower stem tissue samples were collected from the diseased vines, transferred to sterile glass vials and labeled. The stem tissues were surface sterilized with 1% NaOCl (Sodium hypochlorite, commonly known as bleach) for 5 min, followed by rinsing twice in sterile water.

Fig. 1 Wilt disease resistance screening of sweet potato progenies derived from crosses of nine parental cultivars in the glasshouse

From each stem, five thin transverse sections were cut and plated out separately on PDA *Fusarium* selective medium (FSM). The plates were then incubated at 25 °C for 6–7 days. Fusarium-like cultures were visually detected and pure cultures were obtained by plating onto half-strength PDA in Petri™ dish plates.

Fourteen soil samples from different parts of the field were collected after the removal of plants and storage roots to further confirm the presence of *F. oxysporum*. The confirmation of the presence of *F. oxysporum* was done using the soil dilution plate method. After incubation, each individual colony that developed on the Petri™ dish plate was considered as one colony forming unit (CFU) (Nazir et al. 2007; Jasuja et al. 2013). A colony forming unit was determined by the population density expressed per gram of soil with dilution factors using the following equation:

$$CFU = \left(\frac{\text{Total number of CFU of an individual sps}}{\text{Total number of CFU of all sps}} \right) \times 100$$

Statistical analysis

Data of leaf and stem disease ratings of each experiment were analyzed by two-way analysis of variance (ANOVA) using Genstat 64-bit Release 20.1 software (VSN International (2021) and Microsoft Excel 2010 was used for graphs. Means separation was performed using the Bonferroni test based on the results at $P = 0.05$, when significant treatment was observed. The data obtained from non-infected pots were not included in statistical analyses as these were used as control treatments only.

Results

Resistance of parental cultivars to Fusarium wilt PPRI15907 and PPRI9458

Figure 2 presents the disease scores for leaf and stem symptom expression for the glasshouse experiments using artificial inoculation of a single *F. oxysporum* f. sp. *batatas* (PPRI15907), mean of two experiments during 2014, and with a mixture of *F. oxysporum* f. sp. *batatas* (PPRI15907) and *F. oxysporum* f. sp. *vanillae* (PPRI9458) during 2015. Very similar reactions were observed in the three experiments. The parental sweet potato cultivars reacted differently to the artificial inoculation with the Fusarium wilt causal pathogens. Significant ($P \leq 0.05$) differences were observed for leaf and stem disease scores in all experiments. Not all cultivars revealed wilt symptoms on both leaves and stems. In the disease assessments with a single isolate *F. oxysporum* f.sp. *batatas,* cultivars Bonita and Murasaki showed no leaf symptoms, significantly different from Lethlabula, with a mean of 1.5. High stem disease incidences were observed in Lethlabula and Blesbok, while minor symptoms occurred in Bonita and Monate, and the rest of the

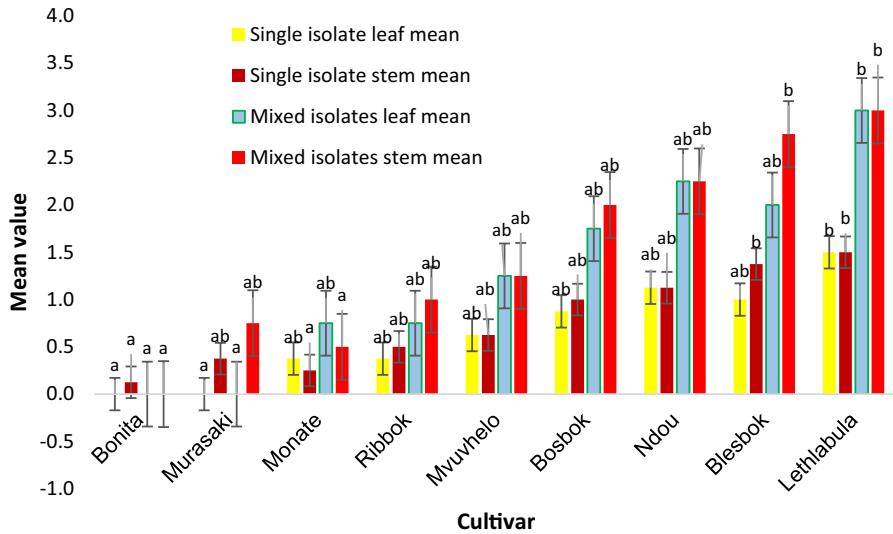

Fig. 2 Mean disease index for leaf and stem symptom expression across three glasshouse experiments for nine sweet potato parental cultivars. Note that in experiments 1 and 2, cultivars were tested against a single *F. oxysporum* f. sp. *batatas* (PPRI15907) isolate; while in experiment 3 cultivars were tested against *F. oxysporum* f. sp. *batatas* (PPRI15907) and *F. oxysporum* f. sp. *vanillae* (PPRI9458)

cultivars was not significantly different in stem symptom expression. In co-infection of *F. oxysporum* virulent isolates, higher disease incidences were observed in all cultivars, except Bonita, which did not show any disease incidence. The same trends for resistance and susceptibility were observed among the cultivars to the co-infection and single isolate inoculum. From the results, the glasshouse experiments were effective in discriminating cultivars for disease severity to single and mixed virulent isolates of *F. oxysporum.*

It is evident from Fig. 3 that the DSI clearly distinguished susceptible and resistant sweet potato cultivars. Based on a single infection, DSI values ranged from 4.2 to 79.2%, with a mean value of 33%. Whereas, with co-infections, DSI values ranged from 4.2 to 81%, with a mean of 35.3%. Some cultivars revealed varying degrees of responses to a single and co-infection. For example, Murasaki changed its classification from moderately susceptible with a single infection to moderately resistant with the co-infection. Mvuvhelo and Ribbok, on the other hand, changed from moderately resistant and resistant to moderately susceptible and susceptible with a single and with the co-infection, respectively. Based on a single and co-infection, Bonita was identified as resistant to Fusarium wilt, Monate as moderately resistant, Ndou and Bosbok as susceptible, and Blesbok (the most popular South African cultivar) and Lethlabula identified as highly susceptible.

Resistance of progenies screened in the glasshouse

The progenies derived from crosses among the parental cultivars showed significant ($P<0.05$) variation against the infection of the mixture of isolates, *F. oxysporum* f. sp. *batatas* (PPRI15907) and *F. oxysporum* f. sp. *vanillae* (PPRI9458). The first symptoms were observed on Lethlabula, susceptible cultivar, 7 days after planting, followed by five progenies (FS9-11, FS9-4, FS10-27, FS10-30 and FS12-5). As time progressed, the wilting symptoms on infected plants increased. Some progenies were identified as resistant and others were highly susceptible to the Fusarium wilt artificial infection (Fig. 4). The results indicated the importance of screening genotypes for disease resistance during the early stages of the breeding phases.

Table 1 presents the mean values for the leaf disease assessments of sweet potato genotypes assessed in the glasshouse. The Bonferroni test ($P=0.05$) clearly clustered the 92 progenies and the nine parental cultivars into nine groups. Mean leaf assessment ratings revealed that Lethlabula was the most susceptible, with a leaf disease score of 2.29, followed by three progenies, namely, FS10-27 (score = 2.00), FS9-11 (score = 1.88), and FS9-4 (Score = 1.86), in which the majority of them were Ribbok × Bonita crosses. Blesbok, Ndou, Bosbok and Mvuvhelo and five progenies did not differ significantly from Lethlabula. with mean disease scores ranging from 0.75 to 1.50, and classified as highly susceptible. Thirteen

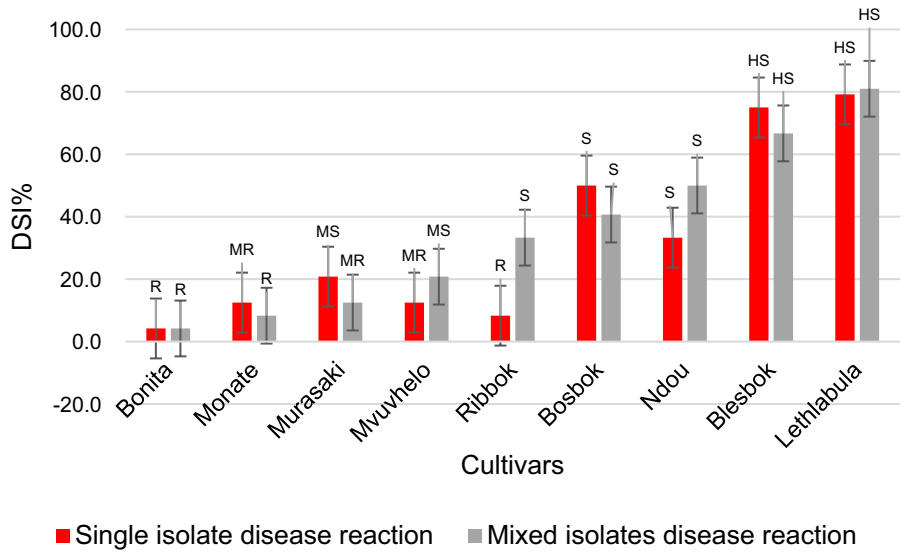

Fig. 3 Mean disease severity index (DSI) for stem wilt in nine sweet potato cultivars across three experiments. In experiments 1 and 2, cultivars were tested against a single *F. oxysporum* f. sp. *batatas* (PPRI15907) isolate; while in experiment 3 cultivars were tested against *F. oxysporum* f. sp. *batatas* (PPRI15907) and *F. oxysporum* f. sp. *vanillae* (PPRI9458). R = resistant, MR = moderately resistant, MS = moderately susceptible, S = susceptible, HS = highly susceptible)

Fig. 4 Infected sweet potato plants with different Fusarium wilt symptoms in the glasshouse

progenies had a mean disease score between 0.71 and 0.50, which were classified as susceptible. Cultivars Ribbok and Monate were among the nine genotypes with means ranging from 0.43 to 0.38 (moderately susceptible). Twenty-seven genotypes had a mean disease score ranging from 0.29 to 0.13 (moderately resistant). However, 37 genotypes, including the two imported cultivars Bonita and Murasaki, were found to be highly resistant with no symptoms of leaf wilting, and with a mean score of 0.00.

Fusarium wilt stem symptoms were more severe in most of the genotypes as compared to leaf symptoms (Table 1). All of the parent cultivars presented stem disease symptoms to the Fusarium wilt isolates used. The highest mean score for stem disease was observed in cultivar Lethlabula (2.43). However, considerable variation was observed among the genotypes. Progenies FS9-11, FS9-14, and FS10-27 and Blesbok had a mean disease score of 2.00, followed by genotypes FS6-2 (1.88) and FS9-4 (1.71). Cultivar Bosbok was among the nine genotypes with a mean score ranging from 1.00 to 1.43, followed by a group containing Mvuvhelo among the 20 genotypes with mean scores ranging from 0.57 to 0.88. Cultivar Ribbok was among the nine genotypes with means ranging from 0.50 to 0.40. Cultivars Monate and Murasaki were among 17 genotypes with means ranging from 0.25 to 0.38. The last group consisted of 39 genotypes, including cultivar Bonita, with mean scores ranging from 0.00 to 0.20, indicating the most resistant group. Genotypes such as FS10-16, FS4-6 and FS11-7 that had moderate leaf disease scores showed a low stem disease score.

The DSI values classified the 101 sweet potato genotypes into five categories according to their wilt response (Fig. 5). Forty-eight genotypes, including Bonita and Monate, were classified as resistant, while 23 genotypes were moderately resistant. Fourteen entries, including cultivars Bosbok and Ndou, were found to be moderately susceptible, while nine were classified as susceptible and seven as highly susceptible. The latter included commercial cultivars Blesbok and Lethlabula.

Field screening of progenies

The response of genotypes to *F. oxysporum* significantly varied in the field experiment. Among the 83 genotypes tested, yellowing of leaves associated with Fusarium wilt were observed in 15 progenies (20%) and four cultivars. Leaf assessments indicated that genotype FS11-1 was the most susceptible, with a mean score of 1.7, followed by FS4-2, with a mean of 0.7 (Fig. 6). Other genotypes that showed wilting symptoms include FS10-18, FS11-7, FS4-3, FS5-1, FS10-10, FS10-15, FS10-25, FS10-32, FS11-18, FS11-4, FS6-1, FS7-3, FS9-7, Mvuvhelo, Bosbok, Lethlabula and Ndou. The remaining progenies, four parental cultivars, such as Bonita, Monate, Murasaki, Ribbok, and the susceptible control cultivar Blesbok did not show any Fusarium wilt symptoms.

In terms of stem assessments, 10 (13.5%) of the 74 progenies (FS4-2, FS11-1, FS6-3, FS10-10, FS10-15, FS7-3, FS11-18, FS10-25, FS10-32 and FS 9-1) (Fig. 6) showed typical symptom characteristics

Table 1 Mean leaf and stem infection ratings of sweet potato genotypes and cultivars, with co-infection of *F. oxysporum* f.sp. *batatas* and *F. oxysporum* f.sp. *vanillae* assessed in the glasshouse

Genotype	Pedigree	Leaf mean	Stem mean	Genotype	Pedigree	Leaf mean	Stem mean
Lethlabula		2.29a	2.43a	FS5-5	Mvuvhelo × Bonita	0.17de	0.00e
FS10-27	Monate × Murasaki	2.00ab	2.00ab	FS11-14	Mvuvhelo × Bonita	0.14de	0.00e
FS9-11	Ribbok × Bonita	1.88abc	2.00ab	FS5-2	Mvuvhelo × Bonita	0.14de	0.14e
FS9-4	Ribbok × Bonita	1.86abc	1.71abcd	FS11-13	Mvuvhelo × Bonita	0.14de	0.14e
FS6-2	Monate × Bonita	1.63abcd	1.88abc	FS11-2	Mvuvhelo × Bonita	0.14de	0.86bcde
FS9-14	Ribbok × Bonita	1.5abcde	2.00ab	FS5-8	Mvuvhelo × Bonita	0.14de	0.00e
Blesbok		1.38abcde	2.00ab	FS10-2	Monate × Murasaki	0.14de	0.57bcde
FS10-6	Monate × Murasaki	1.25abcde	1.38abcde	FS4-7	Bosbok × Bonita	0.13de	0.63bcde
Ndou		1.13abcde	1.13abcde	FS10-1	Monate × Murasaki	0.13de	0.38de
FS10-11	Monate × Murasaki	1.00abcde	1.00abcde	FS9-5	Ribbok × Bonita	0.13de	0.38de
Bosbok		0.88abcde	1.00abcde	FS10-18	Monate × Murasaki	0.13de	0.00e
FS10-12	Monate × Murasaki	0.88abcde	1.13abcde	FS9-15	Ribbok × Bonita	0.13de	0.25de
FS6-4	Monate × Bonita	0.86abcde	0.86bcde	FS10-15	Monate × Murasaki	0.13de	0.25de
FS4-4	Bosbok × Bonita	0.86abcde	1.43abcde	Bonita		0.00e	0.13e
Mvuvhelo		0.75abcde	0.63bcde	FS11-1	Mvuvhelo × Bonita	0.00e	0.00e
FS7-11	Ndou × Bonita	0.71bcde	0.71bcde	FS11-8	Mvuvhelo × Bonita	0.00e	0.00e
FS7-5	Ndou × Bonita	0.67bcde	1.00abcde	FS4-3	Bosbok × Bonita	0.00e	0.17e
FS11-12	Mvuvhelo × Bonita	0.67bcde	0.83bcde	FS10-20	Monate × Murasaki	0.00e	0.14e
FS10-9	Monate × Murasaki	0.63bcde	0.88bcde	FS5-6	Mvuvhelo × Bonita	0.00e	0.5cde
FS11-16	Mvuvhelo × Bonita	0.60bcde	0.60bcde	FS4-1	Bosbok × Bonita	0.00e	0.4cde
FS11-10	Mvuvhelo × Bonita	0.60bcde	0.60bcde	FS6-3	Monate × Bonita	0.00e	0.00e
FS10-16	Monate × Murasaki	0.57bcde	0.14e	FS7-1	Ndou × Bonita	0.00e	0.17e
FS4-6	Bosbok × Bonita	0.50bcde	0.00e	FS10-14	Monate × Murasaki	0.00e	0.14e
FS10-22	Monate × Murasaki	0.50bcde	0.63bcde	FS4-2	Bosbok × Bonita	0.00e	0.25de
FS9-9	Ribbok × Bonita	0.50bcde	0.67bcde	FS1-1	Lethlabula × Bonita	0.00e	0.00e
FS5-4	Mvuvhelo × Bonita	0.50bcde	0.67bcde	FS5-1	Mvuvhelo × Bonita	0.00e	0.33de
FS10-28	Monate × Murasaki	0.50bcde	0.63bcde	FS10-19	Monate × Murasaki	0.00e	0.14e
FS4-8	Bosbok × Bonita	0.50bcde	0.50cde	FS7-6	Ndou × Bonita	0.00e	0.29de
FS7-2	Ndou × Bonita	0.43cde	1.14abcde	FS11-6	Mvuvhelo × Bonita	0.00e	0.20e
FS10-3	Monate × Murasaki	0.43cde	0.57bcde	FS11-18	Mvuvhelo × Bonita	0.00e	0.00e
FS11-7	Mvuvhelo × Bonita	0.43cde	0.00e	FS7-9	Ndou × Bonita	0.00e	0.00e
FS11-5	Mvuvhelo × Bonita	0.43cde	0.71bcde	FS7-12	Ndou × Bonita	0.00e	0.17e
FS9-13	Ribbok × Bonita	0.43cde	0.43cde	FS9-10	Ribbok × Bonita	0.00e	0.33de
FS11-19	Mvuvhelo × Bonita	0.38cde	0.63bcde	FS10-26	Monate × Murasaki	0.00e	0.13e
Ribbok		0.38cde	0.50cde	FS9-12	Ribbok × Bonita	0.00e	0.43cde
Monate		0.38cde	0.25de	FS10-10	Monate × Murasaki	0.00e	0.38de
FS9-1	Ribbok × Bonita	0.38cde	1.13abcde	FS4-5	Bosbok × Bonita	0.00e	0.00e
FS10-25	Monate × Murasaki	0.29de	0.14e	FS7-10	Ndou × Bonita	0.00e	0.20e
FS11-3	Mvuvhelo × Bonita	0.29de	0.43cde	FS10-32	Monate × Murasaki	0.00e	0.00e
FS11-15	Mvuvhelo × Bonita	0.29de	0.33de	FS10-33	Monate × Murasaki	0.00e	0.25de
FS10-21	Monate × Murasaki	0.25de	0.25de	FS10-4	Monate × Murasaki	0.00e	0.33de
FS10-23	Monate × Murasaki	0.25de	0.38de	FS10-5	Monate × Murasaki	0.00e	0.00e
FS9-2	Ribbok × Bonita	0.2de	0.60bcde	FS9-8	Ribbok × Bonita	0.00e	0.00e
FS7-3	Ndou × Bonita	0.2de	0.20e	FS5-3	Mvuvhelo × Bonita	0.00e	0.25de
FS10-31	Monate × Murasaki	0.2de	0.60bcde	FS7-7	Ndou × Bonita	0.00e	0.00e

Table 1 (continued)

Genotype	Pedigree	Leaf mean	Stem mean	Genotype	Pedigree	Leaf mean	Stem mean
FS10-7	Monate × Murasaki	0.2de	0.40cde	FS7-8	Ndou × Bonita	0.00e	0.14e
FS11-4	Mvuvhelo × Bonita	0.17de	0.17e	Murasaki		0.00e	0.38de
FS4-9	Bosbok × Bonita	0.17de	0.00e	FS11-9	Mvuvhelo × Bonita	0.00e	0.50cde
FS11-21	Mvuvhelo × Bonita	0.17de	0.00e	FS5-9	Mvuvhelo × Bonita	0.00e	0.00e
FS10-8	Monate × Murasaki	0.17de	0.17e	FS1-2	Lethlabula × Bonita	0.00e	0.00e
FS9-6	Ribbok × Bonita	0.17de	0.83bcde				
Error mean square		0.40	0.37				
Least significant difference		1.55	1.48				

Means followed by different letters are significant different at $P \leq 0.05$

associated with Fusarium wilt, namely, discolouration of the vascular tissues. Genotype FS11-1 showed disease symptoms in two replications, whereas the rest of the genotypes only showed disease symptoms in one replication, therefore, although the field was known for its natural infection, the distribution of the pathogen in the soil was not equally spread. The mean stem disease index ranged from 0.3 to 2.0, being most severe for FS11-1, followed by FS10-18 and FS4-2.

Furthermore, the soil analyses detected the presence of *Fusarium* sp. in nine of the 14 soil samples tested. The soil analysis indicated significant variations in Fusarium populations in the soil. Both CFU and growth media culture results confirmed that *Fusarium* sp. were present in the soil and responsible for pathogenic reactions of sweet potato genotypes in the field.

Discussion

Due to climate fluctuations and frequent prevalence of drought, Fusarium wilt is gaining importance. This emphasizes the prominence of breeding for resistance to Fusarium wilt. Development of sweet potato resistant cultivars were reported to reduce the rate of disease infection and spread in fields (Clark and Moyer 1988; Egel and Martyn 2013). An assessment of the levels of Fusarium wilt resistance in sweet potato parental cultivars was critical for identification of diverse parental lines to develop segregating progenies with maximum resistance levels for further selection in this study. In the present study, stem disease incidence from single and co-infections was used to calculate DSI percentage. DSI classified parental cultivars as resistant (Bonita and Monate), moderately resistant (Musaraki), susceptible (Ribbok, Bosbok and Ndou), moderately susceptible (Mvuvhelo) and highly susceptible (Lethlabula and Blesbok). However, Dau (2017) reported based on DSI, that Ribbok and Ndou were tolerant to *F. oxysporum* under natural field infestation conditions. The difference in the disease reactions observed in the current study and that of Dau (2017) could be attributed to the difference in the forma speciales that caused wilting. Paul et al. (2020) also employed a rapid assay method and used the DSI percentage based on stem disease incidence to identify five resistant varieties out of 21 sweet potato cultivars tested.

An interesting finding is that the South African isolates used in the present study caused wilt symptoms on Murasaki, which was reported to be resistant to Fusarium wilt in the USA. Previous studies reported that among other USA commercial varieties, Murasaki was resistant, while Bonita was classified as moderately resistant to Fusarium wilt disease isolates in the USA (LaBonte et al. 2008, 2011). Thus, it indicated that different strains might be present in South Africa. In South Africa, *F. oxysporum* f.sp *batata*s and *F. oxysporum* f.sp. *vanillae* (Thompson et al. 2011; Dau 2017) were reported to cause wilt disease and recently Nkosi (2020) found other formae speciales, such as *F. oxysporum* f.sp. *tuberosi* as additional causal agents for the disease. Clark and Moyer (1988) reported in the past that only *F. oxysporum* f.sp. *batatas* was the causal agent of Fusarium wilt in the USA. The two other local isolates may pose a threat to sweet potato production in South Africa and

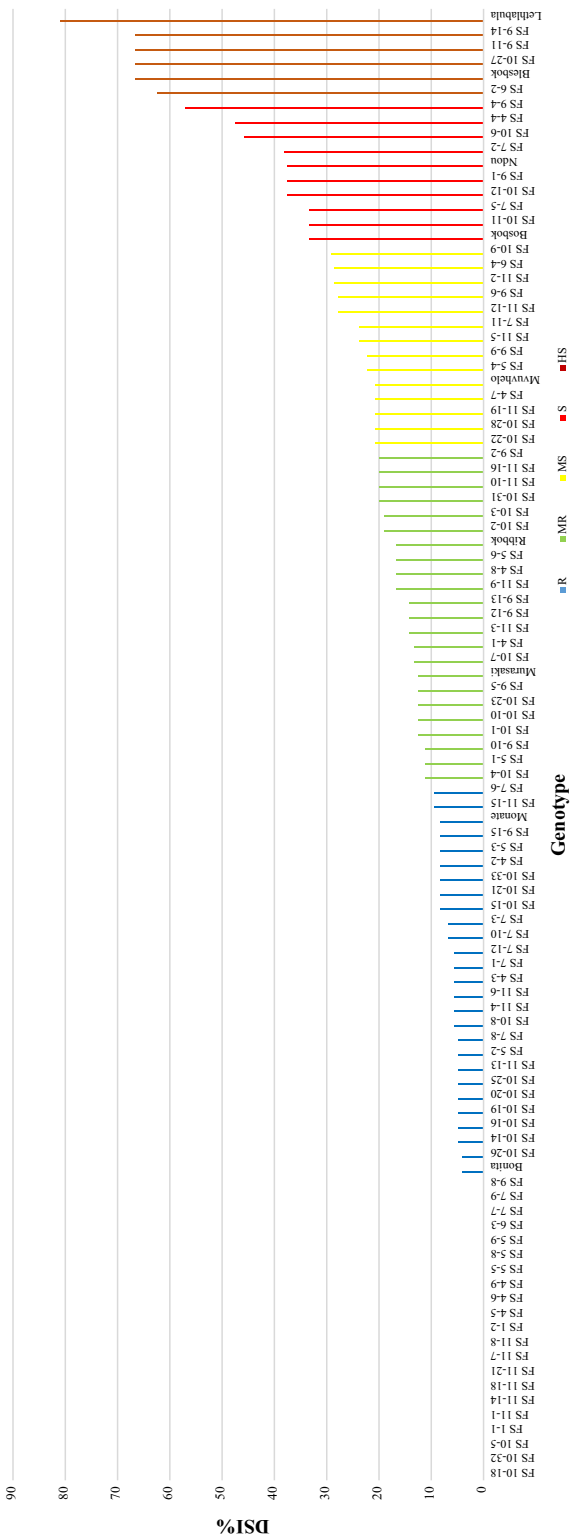

Fig. 5 Mean DSI for stem wilt on 101 sweet potato genotypes in a glasshouse artificially infected with *F. oxysporum* f.sp. *batatas* and *F.oxysporum* f.sp. *vanillae* (R = Resistant, MR = Moderately resistant, MS = Moderately susceptible, S = Susceptible, HS = Highly susceptible)

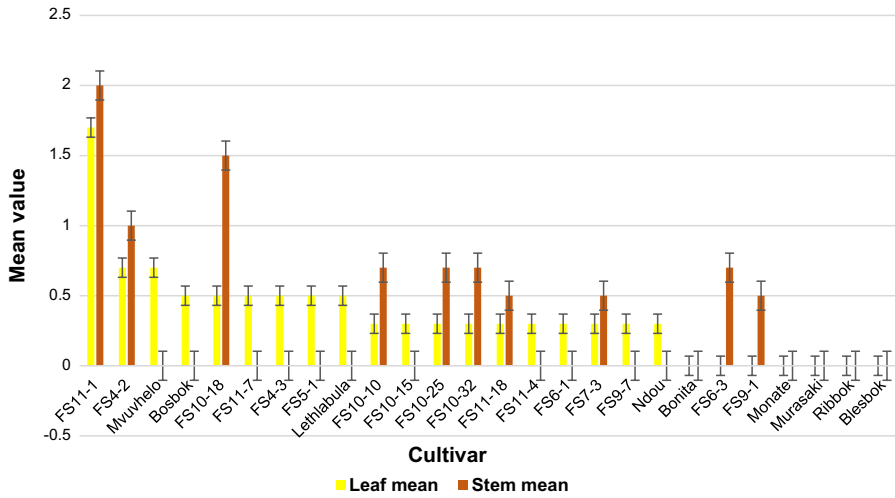

Fig. 6 Mean disease index for leaf and stem symptom expression in 17 sweet potato genotypes, eight cultivars, and the susceptible cultivar Blesbok (FS4 = Bosbok × Bonita; FS5 = Mvuvhelo × Bonita; FS6 = Monate × Bonita; FS7 = Ndou × Bonita; FS9 = Ribbok × Bonita; FS10 = Monate × Murasaki; FS11 = Mvuvhelo × Bonita)

neighbouring countries. The results confirmed that the degree of plant damage differs from cultivar to cultivar and with the different *Fusarium* species due to host specificity of the pathogen (LaBonte et al. 2008; Jackson et al. 2010; Pérez-Vicente et al. 2014; Paul et al. 2020).

Controlled crosses between South African and the USA sweet potato cultivars were successful to breed for resistant genotypes against South African virulent isolates of *F. oxysporum* f.sp. *batatas* and *F. oxysporum* f.sp. *vanillae*. Forty-six of the progenies revealed significant levels of resistance with DSI values ranging from 0 to 9.5%, while 23 genotypes were found to be moderately resistant. A number of researchers have reported variations in the disease reaction levels of different sweet potato genotypes obtained from crossing schemes for Fusarium wilt resistance (Clark et al. 1998; Stoilova and Chavdrov 2006; Yencho et al. 2008; Lopez-Lavallea et al 2012). Genotypes have different genetic defence mechanisms against diseases; however, susceptible genotypes lack important genes or mechanisms to rapidly supress the development of the pathogen (Agrawal and Karban 1999; Kover and Schaal 2002; Voigt 2014).

In general, all the glasshouse experiments presented in this study showed that the disease incidence and severity were higher in the stems than on the leaves. Higher disease reactions on the stem is explained by the fact that the pathogen invades the plant through the root tips and it spreads to the whole plant through the stem as the disease progresses over time (McClure 1949). The variation in resistance levels found among progenies, also confirms the existence of variability among parental cultivars. Fusarium wilt of sweet potato is quantitatively inherited (Collins 1977; Ribeiro et al. 2001; Jackson et al. 2010). Based on both leaf and stem disease assessment results (Table 1), it clearly shows significant variation among progenies of the same pedigree. For instance, out of the 28 progenies of Monate × Murasaki, nine were found to be resistant and 10 moderately resistant, whereas the rest were susceptible. Therefore, resistance and susceptibility of sweet potato genotypes to Fusarium wilt could be directly associated with the frequency of favourable alleles for the resistance gene. Although the inheritance of resistance genes in sweet potato has not been studied, recent reports on proteomics and genomics indicated the presence of differentially expression of genes and defence-related proteins during exposure to *F. oxysporum* f.sp *batatas* (Lin et al. 2017, 2020). Lin et al. (2017) identified various genes that were expressed during exposure to Fusarium wilt, as well as pathogenesis related genes, and genes involved in salicylic acid (SA) and jasmonic acid (JA) signalling pathways. Similarly, Lin et al. (2020) reported that sweet potato resistance responses to *F. oxysporum* f.sp *batatas* infection includes many proteins associated with signal transduction, plant resistance, chitinase and subtilisin-like protease.

Stand reduction in the field that eventually leads to yield loss exhibits the major negative impact of Fusarium wilt in sweet potato production (Michielse and Rep 2009). In recent studies, Paul et al. (2020)

and Yang et al. (2018) conducted a rapid assay (10–12 days) screening method on sweet potato breeding lines for selection of resistance. In their experiments, cuttings were placed in a solution containing 1×105 conidia m/L. The optimum temperature reported by Yang et al. (2018) was 28 °C, which was equivalent to the daily glasshouse temperature in the present study. In the present study, in highly susceptible genotypes, disease symptoms were noted as early as 7 days after planting, and it is therefore recommended that disease recording can be done 14 days (14 days) after planting. The importance of the development of rapid disease screening methods is emphasized by the variation among progenies of the same pedigree, demanding screening of all progenies.

In the present study, progenies such as FS10-32, FS10-25, FS4-2, FS6-3, FS7-3, FS10-15, FS11-18 and FS11-1, that were found to be resistant in the glasshouse screening, were susceptible in the field screening. The variation observed in disease reaction among the genotypes and susceptible control cultivar Blesbok under glasshouse and field conditions could be attributed to disease escape due to uneven distribution of the pathogen in the soil or other soil and environmental factors. Many environmental factors affects Fusarium disease development under field conditions that may influence the susceptibility of the plants and the virulence of Fusarium strains in the field (Smith and Snyder 1971; Fravel et al. 2003; Yang et al. 2018). Detection of *Fusarium sp*. using PDA and soil dilution techniques confirmed that *F. oxysporum* was present in the soil and responsible for the pathogenic reactions of the genotypes in the field. All the experiments conducted in this study showed that although screening of genotypes under controlled conditions reduces environmental influences, variation in the expression of disease symptoms is precisely of genetic nature. However, it is important to confirm resistance under field conditions because there are other soil and climatic factors that influence disease expression and development.

Conclusions

The glasshouse screening used in the present study enabled screening of large numbers of clones within a short period time, which minimises the number of genotypes to be evaluated under field conditions. Murasaki, which was reported as a resistant cultivar, was found to be moderately resistant to Fusarium wilt in both glasshouse and field conditions in this study. In the current study, parental cultivars such as Bonita and Monate were identified as the best parental cultivars for downstream resistance breeding. It is important to note that selection of sweet potato genotypes should be done under conditions of naturally infected soils because Fusarium pathogens occur naturally in various combinations. Observations from this study confirmed that 62 genotypes were found to be resistant to Fusarium wilt under both controlled and field environments. Genotypes classified as resistant and moderately resistant to Fusarium wilt in the current study can be further evaluated for agronomic traits to identify potential lines for release for the informal and commercial production, as well as used as potential parents for future hybridization.

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Declarations

Conflict of interest The authors have no conflict of interest to declare.

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