EFFECT OF SOIL PH ON POSTHARVEST PATHOLOGICAL DETERIORATION OF SWEET POTATO STORAGE ROOTS

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ABSTRACT

Sweet potato (Ipomoea batatas (L.) Lam.) is the world's seventh most important food crop but its potential to contribute to food security and income generation is limited in tropical developing countries by its short shelf-life. Environmental and cultural stresses during growth are known to directly or indirectly predispose sweet potato storage roots to postharvest microbial infection. Research was conducted to determine the effect of soil pH on postharvest deterioration of sweet potato roots using two sweet potato cultivars, Yanshu 1 (CIP 440024) and KSP 20 (CIP 440170), and three soil pH levels, 4.6, 5.8 and 6.1 arranged in randomized complete block design with four replications. Preharvest experiments were conducted in a greenhouse followed by postharvest evaluations in the laboratory at the National Agricultural Research Laboratories (NARL), Nairobi. Nine-mm circular agar plugs, removed from the edge of actively growing two-day old culture of two postharvest pathogens of sweet potato, Rhizopus orvzae and Botryodiplodia theobromae, were used to inoculate the sweet potato roots. Pathological deterioration (PD) was estimated by measuring the diameter and depth of the developing internal lesion (extent of tissue degradation) on the storage roots, 24 hours after inoculation. Results showed that postharvest PD of the storage roots was not significantly (P>0.05) influenced by growing sweet potato in soil at the different pH levels. Growing sweet potato in soil at pH levels within the range for normal plant growth is unlikely to affect postharvest deterioration of the storage roots.

Key words: Ipomoea batatas, Botryodiplodia theobromae, Rhizopus oryzae, Disease, Kenya

INTRODUCTION

Sweet potato (*Ipomoea batatas* (L.) Lam.) is the world's seventh most important food crop after wheat, rice, maize, potato, barley and cassava (FAO, 1992). More than 95% of the production is in the developing countries, with approximately 92% in Asia, 5% in Africa and 3% in the rest

of the world (FAO, 1992; CIP, 1999). In Kenya, sweet potato is the third most important root and tuber crop after Irish potato and cassava (MOA, 1991). It is grown in different agro-ecological zones, largely by small-scale farmers, for home consumption and surplus is sold in local markets (Mutuura *et al.*, 1992). Sweet potato is an important secondary crop that plays an important role in household food security in Kenya (Mutuura *et al.*, 1992). It combines many advantageous attributes that give it great potential as food (Woolfe, 1992). The yellow-orange cultivars contain variable, but sometimes large, quantities of carotenoids which act as precursors of vitamin A (FAO, 1992; Woolfe, 1992), and their consumption is considered an important food-based approach to combat vitamin A deficiency (Hagenimana *et al.*, 1999; Low *et al.*, 1997).

Despite its many good attributes, the harvested root of sweet potato has a short shelf life of less than four weeks in the tropics (UNIFEM, 1993). The root is covered by a thin and delicate skin which is easily damaged during harvesting and postharvest handling (UNIFEM, 1993). The resulting injuries become easy pathways for entry of spoilage microorganisms and moisture loss (Clark, 1992; Bashaasha *et al.*, 1995; NRI/NARO, 1996). Consequently, postharvest pathological deterioration is a principal limiting factor in the marketing and the wider utilization of sweet potato in the tropics (Jenkins, 1982; George, 1988; Abubaker, 1990). During a national social-economic survey of sweet potato farmers in the main sweet potato production areas of Kenya, rotting of roots was rated as the seventh most important production constraint of the 17 cited constraints (Mutuura *et al.*, 1992).

Microorganisms may infect sweet potato roots at different stages, including field, harvest and storage stages (Clark and Moyer, 1988). Infection is mainly facilitated by mechanical injuries of the roots and environmental conditions, but the physiological condition of the root may influence infection (Wills *et al.*, 1998). In addition, environmental and cultural stresses during growth also directly or indirectly predispose the roots to postharvest microbial infection (Clark and Moyer, 1988).

Sweet potato is frequently cultivated in areas with moderate to high acid soils (O'Sullivan *et al.* 1997; Ila'ava *et al.*, 2000). Controlled experiments have demonstrated that it can grow normally at pH values as low as 4 (Ila'ava *et al.* 1996; Ila'ava *et al.*, 2000), but is sensitive to alkaline soil (Rasco Jr. *et al.*, 1986; Woolfe, 1992). Good growth and yields may be obtained in slightly to moderately acidic soils (Steinbauer and Beattie, 1938) with the optimum pH being 5.6 to 6.6 (Rasco Jr. *et al.*, 1986; Woolfe, 1992).

3

This reseach was conducted to study the effect of soil pH on postharvest pathological deterioration of sweet potato roots.

MATERIALS AND METHODS

The research was carried out in a screenhouse at the National Agricultural Research Laboratories (NARL), Kabete, Nairobi in two consecutive years. The temperature and relative humidity inside the screenhouse were not regulated. The sweet potato planting material comprised 25-cm long healthy apical-end vine cuttings obtained from the International Potato Center (CIP) germplasm conservation plot located at the Field Station, University of Nairobi, Kabete Campus, Kenya. Vine cuttings of two sweet potato cultivars: Yanshu 1 (CIP 440024) and KSP 20 (CIP 440170) were planted in 20-litre plastic pots (34 cm diameter and 30 cm depth) in soils obtained from three sweet potato growing areas with different soil pH levels: 4.6, 5.8 and 6.1. Ballast and sand were mixed with each soil in the ratio of 2:2:6 (ballast: sand: soil, v/v) to improve water uptake. The cultivars were selected based on their relative importance in the sub-Saharan region (Carey et al., 1999), their relative easy availability and their reported resistance/susceptibility to postharvest deterioration (Kihurani, 1997). Two apical-end vine cuttings of the cultivars were planted in each pot and the soil mixtures maintained at field capacity moisture content throughout the growing period by canwatering. Water was placed in a small basin in which the pot stood to allow water to diffuse upwards to the plants and prevent leaching of soil nutrients. The two cultivars and the three pH levels were arranged in randomized complete block design with four replications of 8 to 10 roots per treatment.

The roots were harvested at 20 weeks after planting. The pot was inverted gently to remove both the soil and plant roots. The roots were then detached from the plants and placed in small gunny bags, previously labeled with cultivar name, treatment and replicate number, and moved to the laboratory for postharvest analysis.

The harvested roots were inoculated with virulent isolates of the test sweet potato postharvest pathogens, *Rhizopus oryzae* and *Botryodiplodia theobromae*, previously preserved in sterile soil. The pathogen was retrieved from storage by sprinkling a few grains of the soil carrier onto PDA in a petri dish. The petri dish was then incubated for 48 hours at 28°C to allow the pathogen to grow. Using a sterile nine-mm-cork borer, circular agar plugs were removed from the edge of the actively growing culture and used to inoculate the freshly harvested sweet potato roots.

The roots were first washed in running tap water to remove adhering soil and allowed to drip dry in air, then surface-sterilized by briefly dipping them in 96% ethanol to remove surface contaminants. Each root was wounded at the median as follows: A sterile nine-mm diameter cork borer was driven into the flesh of the root to a depth of 5 to 7 mm. The cork borer was then withdrawn in a manner that ensured that the bored tissue was removed, thereby creating a circular 9-mm diameter wound. Each root was inoculated by introducing the inoculum (agar plug) with mycelia side facing down. Sterile agar plugs were used to inoculate the control roots.

Inoculated sweet potato roots were placed in incubation bags which comprised sterile sun-transparent bags (autoclavable, Sigma cell culture (440 x 205 mm) with 24 mm 0.02 micron filter disc). The bags was closed (using staples) to allow creation of high relative humidity around the roots to facilitate infection. Electronic data loggers (Onset® Computer Corp. 1998, US) were also kept besides the roots to monitor temperature and relative humidity during incubation. The bags containing inoculated roots were placed in ordinary stackable plastic crates (57 cm long, 39.5 cm wide and 22 cm deep) and kept at room temperature (20° C to 24° C) for 48 hours.

Disease development was measured on the roots 48 hours after inoculation. Each inoculated root was removed from the incubation bag and cut-open longitudinally with a knife through the inoculation point. The diameter and depth of the internal lesion, shown by the extent of root tissue degradation, was measured in millimeters. The mean diameter and depth were calculated to give the internal lesion dimension as in Duarte and Clark (1993).

Data values for mean internal lesion dimensions (MILD) recorded on the inoculated roots were subjected to analysis of variance using Statgraphics Plus 3.1 statistical software. Where significant differences occurred between treatments, comparisons were performed by Fisher's least significant difference test at P = 0.05 significant level.

RESULTS

In both years of the study, MILD did not differ significantly (P < 0.05) amongst soil pH levels. However, the two-way interactions of soil pH x pathogen, and pathogen x cultivar were significant (P < 0.05) in year 1, but not in year 2. There were significant (P < 0.05) differences between the cultivars and between the pathogens in year 1, but not in year 2. MILD did not differ significantly (P < 0.05) amongst soil pH levels in both cultivars with *Botryodiplodia theobromae*. However, with *Rhizopus oryzae*, MILD in both cultivars was significantly larger at soil pH 6.1, compared to soil pH levels

4.6 and 5.8. In Yanshu 1, MILD differed significantly (P < 0.05) between the pathogens at pH levels 5.8 and 6.1, but not at pH level 4.6. In KSP 20, MILD differed significantly (P < 0.05) only at pH level 6.1. MILD differed significantly (P < 0.05) between the cultivars, with larger MILD developing in Yanshu 1 compared to KSP 20 (Table 1). In year 2, there were no significant interactions in MILD among treatments. In addition, MILD did not differ among the soil pH levels and between the cultivar and the pathogens.

| | inoculate | ed with <i>Rhiz</i> o | opus oryza | <i>te</i> and <i>B</i> | otryodiplodia |
|--|-----------|-----------------------|------------|------------------------|---------------|
| theobromae in year 1 | | | | | |
| Soil | Yanshu 1 | | ŀ | KSP 20 | |
| pН | | | | | means |
| | Rhizopus | Botryodiplodia | Rhizopus | Botryodiplo | dia |
| | oryzae | theobromae | oryzae | theobromae | |
| 4.6 | 16.40 | 12.89 | 7.00 | 10.37 | 11.67 |
| 5.8 | 21.09 | 14.38 | 9.41 | 11.41 | 14.07 |
| 6.1 | 24.36 | 10.26 | 15.94 | 10.08 | 15.16 |
| Mean | 20.62 | 12.51 | 10.78 | 10.62 | |
| LSD ($P=0.05$) for comparing soil pH means = 3.65 | | | | | |
| LSD ($P=0.05$) for pathogen X cultivar interaction = 4.06 | | | | | |
| LSD ($P=0.05$) for comparing cultivar means = 2.98 | | | | | |
| LSD ($P=0.05$) for soil pH level X pathogen interaction = 4.98 | | | | | |
| LSD ($P=0.05$) for comparing pathogen means = 2.98 | | | | | |
| Standard Error $= 3.44$ | | | | | |

Table 1. Effect of soil pH on mean internal lesion dimension (MILD)
(mm) in two sweet potato cultivars Yanshu 1 and KSP 20
inoculated with *Rhizopus oryzae* and *Botryodiplodia*
theobromae in year 1

DISCUSSION

As per the results of this study, different soil pH levels (4.6, 5.8, and 6.1) did not significantly (P<0.05) influence postharvest infection of sweet potato roots by the fungal pathogens, *Rhizopus oryzae* and *Botryodiplodia theobromae*. There was no difference in the extent of deterioration amongst roots harvested from the soils at the different pH levels. The significant interaction between soil pH and pathogen indicated that soil pH influenced the way the different pathogens infected the roots. The magnitude of the influence was, however, not large enough to significantly affect overall root deterioration. Similarly, the interaction between the pathogens and cultivars was significant, but only in the first year. This showed that the pathogen infected roots of the cultivars differently, although the magnitude of the effect was not large enough to significantly influence the level of root deterioration. The inability of the different soil pH levels to significantly influence infection level may be attributed to the tolerance of the sweet potato plant to variations in soil conditions (O'Sullivan *et al.*, 1997; Abruna *et al.*, 1979; Ila'ava *et al.*, 2000).

These results suggest that it is possible to grow sweet potatoes in soils at varying pH levels without significantly influencing postharvest pathological deterioration of the storage roots. This is important because sweet potatoes are frequently cultivated on previously marginal areas with wide variations in soil conditions.

CONCLUSIONS AND RECOMMNDATIONS

The magnitude of the effect of soil pH on sweet potato root susceptibility to infection was not pronounced therefore suggesting that growing sweet potatoes would be a prudent way of utilizing moderately or strongly acidic soils without enhancing postharvest infection of the storage roots. The sweet potato crop does not appear to be strongly influenced by soil pH levels within the range for normal plant growth.

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Afr. J. Hort. Sci. (2008) 1:1-8

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