

FIELD EVALUATION OF ROOT ROT DISEASE AND RELATIONSHIP BETWEEN DISEASE SEVERITY AND YIELD IN CASSAVA

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(Accepted 26 November 2004)

SUMMARY

Reports of cassava root rot disease from different African countries have increased in recent times. Field studies were conducted from July 1998 to October 1999 to determine a reproducible disease assessment method that would allow the comparison of results from different locations and an evaluation of the relationship between disease severity and root yield. Single point disease assessments at 6, 9, 12 and 15 months after planting (MAP) were compared to multiple points assessment based on the area under a disease progress curve (AUDPC). Single point assessments at 12 and 15 MAP, and the AUDPC identified continuous variation ($p \leq 0.01$) among the genotypes. However, a consistent result across trials was obtained only with the assessment based on AUDPC. Root dry yield (DYLD) at 15 MAP showed a strong negative correlation with AUDPC ($r = -0.74$). Regression analysis also confirmed the negative relationship between yield and root rot severity. The five genotypes compared were separated into resistant (91/02324, 30572 and 92/0427) and susceptible (92/0057 and TME-1) groups. It was concluded that root rot disease may cause significant yield loss; however, the magnitude of the yield loss will depend on the susceptibility of the cassava genotype.

INTRODUCTION

Cassava (*Manihot esculenta*) is the most important food crop in sub-Saharan Africa. More than 600 million people depend on the crop in Africa, Asia and Latin America where it serves as an important household food security crop (FAO, 2002). Cassava tuberous roots deteriorate within two to three days after harvest, and the produce become unmarketable after a short time because of this rapid post-harvest deterioration. Consequently, cassava growers commonly leave cassava roots in the soil after maturity until needed (Knoth, 1993). With this method, the rhythm of harvest can be adapted to the needs of the farmer, thereby making the crop a major source of food between the planting and harvesting seasons of other staple crops.

Root rot disease is one major constraint to in-ground storage of cassava. Although previously considered not to be an economic problem, there have been increasing reports of root rot from various African countries such as Bénin, Congo, Ghana,

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Nigeria and Togo (Makambila *et al.*, 1994; Boher *et al.*, 1997; Msikita *et al.*, 1998; IITA, 2000). The disease is caused by different soil borne fungi with varying regional importance. Commonly reported pathogens include *Botryodiplodia theobromae*, *Nattrassia mangiferae* and *Fusarium* spp. (Boher *et al.*, 1997; Msikita *et al.*, 1997; Onyeka, 2002).

Although yield losses as high as 100 % have been reported to occur in farmers' fields (Moses *et al.*, 2003), information is scanty on the effects of root rot on cassava yields from experimental fields and the responses of different varieties to the disease. One major limitation to the availability of information on disease–yield relationships is the lack of a standardized disease assessment method that can be used to compare results. Therefore, the objectives of this study were to determine a disease assessment method which would allow the comparison of results from different locations and to evaluate the relationships between tuberous root yield and root rot disease for different cassava genotypes.

MATERIALS AND METHODS

Field site, experimental layout and crop establishment

Six cassava genotypes, which included a popular African landrace (TME-1) and five elite genotypes (30001, 30572, 92/02324, 92/0427 and 92/0057) from the International Institute of Tropical Agriculture (IITA) improved germplasm collection, were evaluated in the field over a period of 15 months. The genotypes 30572 (resistant) and TME-1 (susceptible) were used as controls; they are both widely grown by cassava farmers in Nigeria.

The study was conducted between July 1998 and October 1999 at the IITA experimental field, Ibadan, Nigeria, located in the transitional forest agro-ecological zone (lat. 7°43' N, long. 3°90' E). The experiments were established in a randomized complete block design with six replications. Cassava genotypes were randomly allocated to plots; each plot consisted of nine rows. Each row was 5 m long and spaced 1 m from neighbouring rows. Two experiments were conducted simultaneously in nearby experimental fields previously under cassava cultivation. All six cassava genotypes were planted in the first field, and five (excluding 30001) were planted in the second field. Weeding was carried out manually when needed throughout the duration of the study.

Data collection

Disease and yield assessments were carried out at 6, 9, 12 and 15 months after planting (MAP) using a destructive sampling procedure. At each sampling, five plants on alternate rows were sampled per plot (second row at 6 MAP, fourth row at 9 MAP, sixth row at 12 MAP and eighth row at 15 MAP). At each sampling, the total number of roots and root fresh weight were recorded for each plot and used to calculate the fresh yield (FYLD). Root samples were collected and the root dry matter content was determined by the oven dry method. Root dry yield (DYLD) in t ha^{-1} was derived by multiplying the fresh root weight by the percentage dry matter content. Root rot disease was assessed by visual observation and by counting the number of rot-infected

roots; the percentage of rot-infected roots was then calculated. Disease severity was therefore evaluated as the percentage of the roots infected (%Rot) at 6, 9, 12 and 15 MAP. Also, disease severity was evaluated as the cumulative area under the disease progress curve (AUDPC). This is obtained by plotting mean disease severity (%Rot) against time. It represents the magnitude of disease for the entire growing period and was calculated according to the formula proposed by Shaner and Finney (1977):

$$A = \sum_{i=1}^n [(x_i + x_{i+1})/2]t_i$$

where x_i = the transformed percentage rot on date i ,

n = the number of assessments

t_i = the time in months between disease assessment x_i and x_{i+1} .

Statistical analyses

Only data for the five genotypes planted in both trials were included in the analyses. A square root transformation of the disease data (percentage rot-infected roots per plot) was carried out before analyses. Analyses of variance (ANOVA) were performed using the general linear model (GLM) procedure of SAS statistical package version 8, for the percentage root rot (%Rot) at various assessment periods and the cumulative AUDPC at 12 and 15 MAP. Correlation and regression analyses were used to determine the relationship between yield and disease severity.

RESULTS

Assessment of disease severity

With percentage rot (%Rot) as the disease severity index, no differences were observed among the genotypes at 6 and 9 MAP; also there was no difference between trials. At 12 MAP, differential responses were observed among genotypes, and there was also a difference in disease levels between the two experimental locations (trials) ($p = 0.01$). The performance of individual genotypes was consistent across trials as indicated by the non-significant genotype \times trial interaction. At 15 MAP, strong variations ($p < 0.01$) among genotypes and between trials were observed. Also, the performance of genotypes across trials differed (Table 1). With the AUDPC as the disease index, a differential response among genotypes was established, but there was no variation between trials at either 12 or 15 MAP.

Relationship between yield and disease severity:

The data for the two trials were combined and disease parameters were correlated with yield at 12 and 15 MAP. The DYLD at 12 MAP showed a negative correlation with %Rot ($r = -0.55$) and AUDPC ($r = -0.52$). A stronger correlation was established between yield and disease at 15 MAP with correlation coefficients of -0.73 for %Rot and -0.74 for AUDPC. A similar trend was observed between root fresh yield and disease severity. Yield was also related to disease severity by simple linear regression analysis. Again, a negative relationship was observed. The estimated regression line

Table 1. Analyses of variance for the response of five cassava genotypes to root rot disease in the field evaluated as percentage root rot (%Rot) at different growth stages and the area under the disease progress curve (AUDPC).

| MAP† | Source | d.f. | %Rot | | AUDPC | |
|------|---------------------|------|-------------|------------------|-------------|------------------|
| | | | <i>m.s.</i> | <i>F</i> -values | <i>m.s.</i> | <i>F</i> -values |
| 6 | Genotype | 4 | 2.06 | 1.13 | | |
| | Trial | 1 | 0.20 | 0.11 | | |
| | Genotype × Trial | 4 | 1.28 | 0.70 | | |
| | Rep within Genotype | 20 | 1.29 | 0.71 | | |
| | Error | 25 | 1.82 | | | |
| 9 | Genotype | 4 | 10.18 | 2.22 | | |
| | Trial | 1 | 0.01 | 0.01 | | |
| | Genotype × Trial | 4 | 0.76 | 0.17 | | |
| | Rep within Genotype | 20 | 3.04 | 0.66 | | |
| | Error | 25 | 4.59 | | | |
| 12 | Genotype | 4 | 101.18 | 3.02* | 724.64 | 3.10* |
| | Trial | 1 | 251.37 | 7.50* | 485.98 | 2.08 |
| | Genotype × Trial | 4 | 21.13 | 0.63 | 150.10 | 0.64 |
| | Rep within Genotype | 20 | 22.37 | 0.67 | 146.40 | 0.68 |
| | Error | 25 | 33.52 | | 233.93 | |
| 15 | Genotype | 4 | 53 20.51 | 356.11** | 27 939.58 | 54.31** |
| | Trial | 1 | 1415.04 | 94.71** | 2099.59 | 4.08 |
| | Genotype × Trial | 4 | 239.31 | 16.02** | 305.59 | 0.68 |
| | Rep within Genotype | 20 | 166.72 | 11.16** | 994.38 | 1.93 |
| | Error | 25 | 14.94 | | 514.43 | |

† MAP, months after planting.

* Significant at $p \leq 0.05$.** Significant at $p \leq 0.01$.

between root yield and root rot disease (%Rot) is given by $\text{yield} = a + b (\text{disease})$, where $a = 33.56$ (*s.e.* 1.097) and $b = -0.62$ (*s.e.* 0.137) at 12 MAP. Although a weak relationship was established between yield and disease at 12 MAP, a stronger negative effect was obtained at 15 MAP (Table 2).

Comparison of the various genotypes for reaction to root rot disease and yield performance showed that two of the elite genotypes (91/02324 and 30572) performed consistently better than the susceptible control (TME-1). Genotype 30572 had the lowest percentage root rot, 1.2 % at 12 MAP and 7.8 % at 15 MAP, compared to 8.4 % and 56.4 % obtained at the corresponding times for the susceptible control. For all the genotypes, a greater than 100 % increase in root rot occurred between 12 and 15 MAP; however, this caused a corresponding reduction in yield only with the susceptible genotypes (Table 3). All the elite genotypes yielded more than TME-1.

DISCUSSION

Field results for disease evaluation can be influenced by the timing and method of assessment (Dowley *et al.*, 1991); therefore, identifying the best parameter for disease assessment is critical to relating disease severity to yield loss. Our results showed that a significant relationship between the root rot pathogen and the cassava host was

Table 2. Coefficients of correlation (r^2) and F -statistics between root rot disease severity and cassava root yield (dry yield) at 12 and 15 months after planting (MAP).

| Rot index† | r^2 | F |
|-----------------|-------|---------|
| Yield at 12 MAP | | |
| 6 MAP | 0.07 | 4.48 |
| 9 MAP | 0.04 | 2.45 |
| 12 MAP | 0.31 | 25.75** |
| AUDPC | 0.27 | 22.49** |
| Yield at 15 MAP | | |
| 6 MAP | 0.00 | 0.01 |
| 9 MAP | 0.03 | 1.75 |
| 12 MAP | 0.15 | 9.82** |
| 15 MAP | 0.54 | 68.11** |
| AUDPC | 0.54 | 69.83** |

† Disease severity was assessed as the percentage root rot at 6, 9, 12, and 15 MAP; and as the cumulative area under disease progress curve at 12 and 15 MAP.

** F -values are significant at $p \leq 0.01$.

Table 3. Mean groupings for field assessment of percentage root rot (%Rot)†, area under disease progress curve (AUDPC)‡, and dry root yield (DYLD) in $t\ ha^{-1}$ of five cassava genotypes at 12 and 15 MAP.

| Genotypes | 12 MAP | | | 15 MAP | | |
|-------------|--------|-------|------|--------|-------|-------|
| | %Rot | AUDPC | DYLD | %Rot | AUDPC | DYLD |
| 91/02324 | 1.7 | 4.4 | 12.3 | 9.6 | 24.3 | 10.8 |
| 30572 | 1.2 | 4.9 | 11.0 | 7.8 | 20.9 | 12.9 |
| 92/0427 | 4.3 | 17.2 | 11.1 | 9.5 | 41.0 | 9.3 |
| 92/0057 | 5.1 | 9.6 | 10.2 | 31.2 | 73.9 | 7.0 |
| TME-1 | 8.4 | 22.1 | 8.8 | 56.4 | 137.2 | 3.7 |
| Mean | 4.1 | 11.6 | 10.7 | 22.9 | 59.5 | 8.7 |
| <i>s.e.</i> | 1.67 | 4.42 | 0.58 | 1.12 | 6.55 | 0.253 |

† %Rot is the percentage of rot-infected roots at 12 and 15 MAP.

‡ AUDPC curve up to the point of harvest.

obtained only at 12 and 15 months but not before or at nine months after planting. Fagbola *et al.* (1998) in a study with vesicular-arbuscular mycorrhiza obtained a higher rate of infection of cassava roots by the mycorrhizal fungi after nine months. However, we also discovered that assessment of root rot severity based on the percentage of infected roots at a single stage was subject to variation between fields. Also, the difference in replication within a genotype obtained at 15 MAP is an indication that results based on the single point assessment of the percentage of rotted roots are likely to become more unreliable after 12 months due to within-field variation.

The multiple points evaluation as represented by the cumulative AUDPC was able to identify a differential response between genotypes, and produced a consistent result across trials. Jeger (2004) noted that the use of AUDPC as a measure of disease intensity will help to average out the undoubted variation often associated with field

assessment. Therefore, for a particular pathosystem such as cassava root rot disease where a strong variation in the spatial distribution of the inoculum exists, the use of AUDPC will help to reduce the variation in results, and consequently provide a uniform basis for the comparison of results from different locations. Reproducibility of results with minimal variation is critical to any assessment method that relates the effect of disease to crop yield loss (Lipps and Madden, 1989). Jeger and Viljanen-Rollinson (2001) observed that the AUDPC method integrates all aspects of disease progress in relation to host development and growth. Consequently, it provides a better basis for relating the disease effect to yield. This is consistent with the results of Edema and Adipala (1995) in a study on brown rust of cowpea, in which they obtained a better coefficient of correlation between AUDPC assessment and yield than with a critical point assessment.

Different factors could be responsible for the high levels of variation observed with the single point assessment method in this study. Roots that were infected at the very early stage of plant development might have completely deteriorated before assessment at a critical point, and consequently would not contribute to the analyses. Also, roots infected close to the time of assessment might not have developed enough external symptoms at the critical point. The above factors, in addition to the non-uniform distribution of inoculum in the soil, will continue to affect field evaluation of root rot disease based on a single point assessment. The use of AUDPC involves extra costs for sequential assessments, but it provides results that are comparable under different conditions. Jeger and Viljanen-Rollinson (2001) showed that with as few as two assessments, as much information could be generated from AUDPC as with many sequential assessments. There is a need therefore for further studies to determine the number of assessments that will enable optimization of the value of the information obtained in relation to the time, costs, and calculation effort required to use AUDPC.

Analyses of the relationships between yield and disease severity by both correlation and simple linear regression indicated that root rot disease can reduce the expected yield from a cassava field. Also, our results showed that leaving cassava in the field beyond 12 MAP, a common practice among farmers (Knoth, 1993; Fagbola *et al.*, 1998) will lead to an increased reduction in yield for susceptible genotypes. This is in agreement with the results of Ambe (1994) who reported a reduced yield on a white-skinned cassava variety after 12 months in the field. However, it has been observed that different cassava genotypes respond differently to root rot disease (Boher *et al.*, 1997; Onyeka, 2002). Therefore the effect of increasing severity on yield will depend on the susceptibility of the genotypes, as was confirmed in this study. High levels of resistance were identified in some of the improved genotypes with less than 2 % of total root rot at 12 MAP. The genotypes were clearly separated into two groups: resistant (91/02324, 30572, and 92/0427) and susceptible (92/0057 and TME-1). Although a greater than 100 % increase in percentage root rot was observed for all the genotypes between 12 and 15 MAP, this increase resulted in a corresponding significant reduction in yield from susceptible genotypes (92/0057 and TME-1) only. There was no effect on 30572 which is known to be resistant to other pest and diseases of cassava and has been extensively used as a source of resistance in breeding programmes (IITA, 2000).

CONCLUSIONS

This study showed that the effect of root rot disease on cassava is best identified from 12 months after planting. To enable different genotypes and results from different locations to be compared, root rot evaluation should be based on multiple point AUDPC assessment. It is concluded that root rot disease epidemics can lead to significant yield losses. However the extent of yield loss will depend on the susceptibility of the genotypes. High levels of resistance were identified in two of the genotypes studied (30572 and 91/02324). This shows that the use of resistant genotypes is a potential approach to effective management of cassava root rot disease.

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