Quantitative nature of downy mildew resistance in Nigerian elite pearl millet lines

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Abstract

Downy mildew disease caused by the oomycete, Sclerospora graminicola, causes yield losses across the millet growing zones of Nigeria. Identification of the source of resistance and nature of inheritance controlling resistance is useful in developing appropriate breeding strategies. Therefore there was need to study downy mildew resistance in Nigerian elite pearl millet (Pennisetum glaucum) lines. Five pearl millet parental lines, BUDUMA, SOSAT-C88, LCICDMR36-4, 20B-2 and 25B-4, were obtained from Lake Chad Research Institute, Maiduguri, Nigeria. Using factorial mating scheme of North Carolina Design II, 6F1s, 6F2s, 6BC1P1s and 6BC 1P2s were generated from these five parents during November 2004 to May 2005 off-season under irrigation at Lake Chad Research Institute. The five parents, twenty-four crosses and one check (7042S) were evaluated using a randomized complete block design (RCBD) with three replications in the downy mildew field nursery of the Lake Chad Research Institute and the Experimental Farm of the Department of Crop Production and Horticulture, Federal University of Technology, Yola, Nigeria during 2005 and 2006 cropping seasons. The evaluations were done to estimate the number of genes involved in the inheritance of downy mildew resistance. Results from this study indicated that several loci were involved. Average number of loci controlling resistance to downy mildew incidence was 10.15 and 5.14 for downy mildew severity, which made the inheritance quantitative in nature. Narrow-sense heritability was moderately high (66% for downy mildew incidence and 53% for downy mildew severity) indicating improvement perhaps can be achieved by using this specific breeding stock.

Introduction

In Nigeria, pearl millet (Pennisetum glaucum) is second only to sorghum (Sorghum bicolor) in the areas where it is cultivated (Werder and Manzo 1992). The crop can be used for various purposes. Millet grain is used for human consumption in which the whole grain is cooked and served like rice. The flour is processed into thick porridge called tuwo served with traditional vegetable soup; also fried snack called masa and non-alcoholic beverage called fura are prepared in Mali, Niger and Nigeria. According to Hash et al. (1997), downy mildew, caused by the oomycete Sclerospora graminicola, is the most serious disease of pearl millet in Nigeria, where the epiphytotics occur annually and it is common to find systematic infection up to 50% in farmers’ fields. Breeding for resistance to diseases of economic importance, especially downy mildew, will contribute to increased productivity and stability of pearl millet grain, stover and forage yields. Several published reports on the inheritance of downy mildew resistance summarized by Hash et al. (1997), demonstrated it to be controlled by one or two dominant genes, while in others it was reported to be controlled polygenically and by additive and non-additive gene effects. In the few cases where clear Mendelian segregations have been observed, the number of dominant genes governing resistance has been one or two, suggesting that non-additive gene action is responsible for much of the heritable variability. To increase the durability of resistance several gene pyramiding and deployment strategies have been proposed by Hash et al. (1997). This study is therefore designed to determine the number of loci governing the nature of inheritance of downy mildew resistance in Nigerian elite pearl millet lines.

Methodology

Five pearl millet parental lines (BUDUMA, SOSAT-C88, LCICDMR36-4, 20B-2 and 25B-4) were obtained from Lake Chad Research Institute, Maiduguri, Nigeria. From the five parents, 6F1s, 6F2s, 6BC1P1s and 6BC 1P2s were generated using a factorial mating design (North Carolina Design II) during November 2004 to May 2005.
off-season at the Lake Chad Research Institute under irrigation. The five parents, twenty-four crosses and one check (7042S) were evaluated using a randomized complete block design (RCBD) with three replications in the downy mildew field nursery of Lake Chad Research Institute and the Experimental Farm of Federal University of Technology, Yola, Nigeria during 2005 and 2006 cropping seasons. Downy mildew incidence (DMI) was computed as the percentage of diseased plants expressed as:

\[
\text{DMI} = \frac{\text{Number of diseased plants}}{\text{Total number of plants}} \times 100
\]

Disease severity was scored on a 1–5 scale as described by Williams et al. (1981), where 1 = no disease; 2 = symptoms on aerial tillers only; 3 = symptoms on less than 50% basal tillers; 4 = symptoms on more than 50% basal tillers; and 5 = total destruction of stand or no production of normal head. Downy mildew severity (DMS) index (%) was calculated using a formula described by Williams et al. (1981), as follows:

\[
\text{DMS} = \frac{n_1(1-1) + n_2(2-1) + n_3(3-1) + n_4(4-1) + n_5(5-1)}{N(5-1)} \times 100
\]

where \(n_1...n_5\) = number of plants with different disease grades described in the 1–5 scale above; and \(N\) = total number of plants assessed.

Minimum number of loci involved in genetic control of the inheritance of the character was calculated using the least-square means and variances by a formula developed by Mather and Jinks (1971):

\[
n = \frac{0.25 \times (0.75 - h + h^2)D^2}{\delta^2F_2 - \delta^2F_1}
\]

where \(n\) = minimum number of loci involved in the inheritance of the character being considered;

\(h = F_1 - P_1/P_2 - P_1\) and \(F_1, P_1\) and \(P_2\) are means of \(F_1\), \(P_1\) and \(P_2\) respectively; and

\(D^2 = \text{squared deviation of either parents from the mid-parent value (P}_2 - \text{P}_1)\) based on the following assumptions: (i) no linkage between relevant loci; (ii) each loci contributes only positively or alleles effects of all loci are equal; (iii) the degree of dominance is the same; and (iv) no epistatic interactions exist between loci.

Estimates of broad-sense and narrow-sense heritability were calculated for DMI and DMS in pearl millet by using the variance of the parent, \(F_1\), \(F_2\) and backcross generations to estimate phenotypic (\(V_p\)), environmental (\(V_e\)), total genetic (\(V_g\)), additive genetic (\(V_a\)) and dominance genetic (\(V_d\)) variances:

\[
\begin{align*}
V_p &= V_{F_2} \\
V_e &= 0.25(V_{P_1}) + 0.25(V_{F_2}) + 0.5(V_{F_1}) \\
V_g &= V_{F_2} - V_e \\
V_a &= 2(V_{F_2}) - V_{BCP1} - V_{BCP2} \\
V_d &= V_{BCP1} - V_{BCP2} - V_{F_2} - V_e
\end{align*}
\]

Broad-sense heritability:

\[
h_b^2 = \frac{(V_A + V_D)VF_2}{\delta^2F_2 - \delta^2F_1} = \frac{\delta^2g}{\delta^2F_2}
\]

where \(V_A + V_D\) represent the genetic variance of \(F_2\) according to Allard (1960).

Narrow-sense heritability = \(h^2 = \frac{V_A/V_{F_2}}{\delta^2F_2 - (\delta^2B_1 + \delta^2B_2)/\delta^2F_2}\) as described by Warner (1952).

Results

Estimates of number of loci controlling resistance to DMI and DMS on pearl millet are presented in Table 1. Estimated number of loci controlling resistance to DMI varied from 0.3 to 20.0, where the crosses between 20B-2 × LCICDMR36-4 and 25B-4 × LCICDMR36-4 had the lowest (0.3) and highest (20.0) number of loci respectively, with a mean value of 10.2 for incidence (Table 1). Estimated number of loci controlling resistance to DMS varied from 0.3 to 10.0 where the crosses 20B-2 × LCICDMR36-4 and 25B-4 × SOSAT-C88 recorded the lowest (0.3) and highest (10.0) loci respectively, with a mean value of 5.1 (Table 1).

Results from this study indicated that several loci were involved. Therefore, the inheritance of resistance to downy mildew is a quantitative character in the set of parents studied and breeding for downy mildew resistance should be possible. However, the high number of loci obtained for crosses involving 25B-4 × SOSAT-C88 means that these parents have broad genetic base for downy mildew resistance. It also ensures stability against breakdown of resistance as commonly observed in single-cross hybrids. However, this does not mean that such resistance will be easy to manipulate in a pedigree or backcross-based breeding program. To increase the durability of resistance, several gene pyramiding and deployment strategies have been proposed (Hash et al. 1997), all of which require the availability of a pool of QTLs (quantitative trait loci) with different pathogen specificities.
The quantitative nature of inheritance was demonstrated by the estimation of several loci controlling resistance to DMI and DMS among the materials studied. Estimates for broad-sense heritability for resistance to DMI ranged from 38 to 89% with overall mean value of 64%. Narrow-sense heritability ranged from 44 to 88% with mean value of 66%. Crosses involving SOSAT-C88 as a donor parent had both the highest broad-sense and narrow-sense heritability for incidence (Table 1). Broad-sense heritability estimates for resistance to DMS ranged from −14 to 93% with a mean of 40% and narrow-sense heritability estimates ranged from 8 to 97% with overall mean of 53% (Table 1). This shows that resistance to downy mildew is highly heritable and would respond to selection, which could be facilitated by modern biotechnological tools such as marker-assisted breeding techniques. In this study the high heritability estimates for DMI and DMS indicate that transfer of resistance to recipient parents from donor parents was highly possible.

### References


