INHERITANCE OF RESISTANCE TO YELLOW SPOT IN SEGREGATING POPULATIONS OF SUGARCANE

By
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Abstract

The inheritance of resistance to yellow spot in eleven families (parent varieties and progeny) was studied over three disease evaluation dates over two years. The study showed that the control and parent varieties could be consistently classified in the disease reaction classes assigned from previous field trials. The absolute infection level of a control or parent clone and families fluctuated owing to seasonal variation in infection pressure. There was a clear tendency for the mean infection level of the progeny to increase as the combined susceptibility of the parents increased. Narrow-sense heritability derived from the partitioning of variances between and within families varied from 0.68–0.71 when infection was relatively low, increased to 0.83 under severe infection pressure and was very high, 0.98, when it was derived from data pooled over all evaluation dates. Frequency distributions of infection pooled over evaluation dates showed a fairly wide distribution of progenies with transgressive segregants occurring in the lower and higher susceptibility classes in all categories of crosses. The distribution was skewed towards the lower infection classes for crosses among resistant and slightly susceptible parents, whereas it tended towards a binomial distribution in crosses which involved a susceptible or highly susceptible parent. The mode of segregation indicated an oligogenic system with often a quantitative expression for the trait. Parent clones and families differed widely with respect to infection level but parent x date or year and family x date or year interactions were not important within the same year of evaluation or between years, which indicated very high broad-sense heritability of 0.89–0.95. The significance of these findings in designing a sound breeding strategy for yellow spot resistance is discussed.

Introduction

Yellow spot, a fungal disease caused by Mycovellosiella koepkei (Krüger) Deighton is considered to be the most important of the leaf spots of sugarcane (Saccharum spp.) in the tropics (Ricaud and Autrey, 1989). The disease causes yellow spotting of the leaf surface which may coalesce in susceptible varieties under suitable weather conditions to cover large areas which assume a brick red or reddish brown colour. The disease is particularly severe under high relative humidity and prolonged rainfall (Ricaud, 1974; Roach, 1975), conditions which are characteristic of the wet uplands of Mauritius covering around 30% of the area under cane. In these areas, the disease has caused economic losses of 10–15% in commercial varieties which were grown on a large scale (MSIRI, 1978). The threshold of foliar area infection at which losses usually occur is about 15% (Autrey et al., 1983) whereas the extent of cover of top-most 8–10 leaves by the spots may be as much as 35–50% (Ricaud, 1974). Disease build-up and losses are more important in flowered than in vegetative stalks (Ricaud et al., 1983). In Mauritius, infection usually starts towards the end of January and ends in July (Ricaud, 1974) when the cane stalk has nearly completed its elongation and is actively accumulating sugar.

In routine resistance trials, promising varieties at final phase of the selection program are assessed twice during a year, in April and after flowering towards the end of June to ensure that the peak of infection in different varieties is not missed (Ricaud et al., 1983). Disease evaluations are conducted over a plant crop and two successive ratoon crops to make allowance for yearly variation of epiphytotics and disease build-up in ratoons. Disease assessment has been found to be more accurate in ratoon than in plant cane as a result of disease build up.

In Mauritius, a fairly high number of promising varieties suitable for the very humid zones (annual rainfall > 2400 mm) cannot be released for commercial cultivation owing to high susceptibility to yellow spot and this is a matter of concern. Aerial spraying of fungicides in sugarcane plantations to control the disease is economically feasible (Autrey et al., 1983) but has not been adopted. The alternative is to breed for resistant varieties. Little information is available on the genetics of resistance to the disease. At the species level, Roach (1975) reported high levels of resistance in the wild Saccharum spontaneum and lower levels in the wild S. robustum and S. officinarum varieties. The latter tend to be less susceptible than commercial-type hybrids (Hussain and Singh, 1970; Roach, 1975). A preliminary study (Ramdoyal et al., 1996) suggested a 3–4–gene inheritance model with dominance for resistance. A trial was designed to investigate further the genetics of resistance to yellow spot in commercial crosses with repeated measurements on infection levels in order to develop strategies for producing varieties resistant to the pathogen.

KEYWORDS: Sugarcane, Breeding, Heritability, Mycovellosiella koepkei, Resistance.
Materials and methods

Plant material

Eleven crosses between parents varying in their reaction to the disease and including selfing of the highly susceptible male variety, were used in the trial. Potted seedlings were transplanted in April 1993 to the experimental site in a very humid environment at Union Park Sugar Experiment Station (UPSES) (altitude 350 m, annual rainfall 3486 mm, yearly mean minimum and maximum temperatures of 18°C and 24.5°C respectively) where yellow spot resistance trials are commonly planted. Each family was split into two groups of 52 seedlings, each allocated to a replicate in a randomised complete block with two replicates. The families were planted in two rows of 26 progenies per row in each replicate spaced at 1 m within a row and 1.5 m between rows. Parents were established from one-eyed cuttings in pots which were planted at regular intervals in each family row. Each female and male parent was replicated four times within each family row. Similarly eight control varieties were established in pots from one-eyed cuttings in alternate stools along the row. The experimental site occupied a total area of around 0.42 ha. The trial was harvested in September 1994 as plant crop and in October 1995 as a first ratoon crop and subsequently after another period of twelve months in October 1996 as a second ratoon crop.

Disease evaluation

Disease evaluation was performed in first and second ratoon crops in 1995 and 1996 respectively. Each progeny, parent and control stool was rated three times: the first two evaluations were done in 1995 at the time of peak infection on 27 April and towards the end of peak infection on 29 June respectively and the third one was done on 13 May 1996, a time that coincided with the peak infection for that year. Infection was assessed visually and expressed as a percentage of the mean area covered by fungal spots on the ten uppermost leaves on a sample of one stalk per stool (Ricaud, 1970). For the stools in which a majority of stalks had flowered in June 1995, infection rate was determined on both flowered and vegetative stalks. Flowering usually occurs as from the third week of May to end July. In order to determine the peak time of infection, in 1995, the evolution of the disease was followed at regular intervals on ten stalks in the spreader rows of varieties, B3337 and S17, in both the experimental plot and in routine yellow spot disease trials at UPSES. In 1996, the evolution of the disease was followed at monthly intervals in the variety R570 in disease testing trials at Britannia (altitude 197 m, annual rainfall 1927 mm, yearly mean minimum and maximum temperatures of 18.6°C and 25.7°C respectively) which is located at about eight kilometres from UPSES.

Analysis

Population statistics were calculated for the crosses, parents, and controls at each date of evaluation and on pooled data within and across years. Frequency distributions of progenies within infection classes were compiled on data pooled over all evaluation dates. Since the infection rate of the whole population of progenies ranged from 0–50% with the majority of progenies grouped between 0–30%, an arcsine transformation was done on the data before they were analysed. Three methods were used to determine the heritability of the character: analysis of full-sibs based on the 'between families' and 'within families' components of variance, \( \sigma^2_B \) and \( \sigma^2_W \), which are then used to derive the additive variance, \( \sigma^2_A \), and the environmental variance, \( \sigma^2_E \) (Kearsey and Pooni, 1996). Then, ignoring dominance, the heritability in the narrow sense, \( h^2_n \), is derived from the relationships:

\[
\sigma^2_B = 1/2 \sigma^2_A
\]

\[
\sigma^2_W = 1/2 \sigma^2_A + \sigma^2_E
\]

\[
h^2_n = \frac{\sigma^2_A}{\sigma^2_A + \sigma^2_E}
\]

The second method is based on the regression of family means onto the mid-parent ratings, and the regression coefficient gives an estimate of \( h^2_n \). The results of the analyses of variance obtained for these two methods are given in the same table since the 'between families sum of squares' can be broken down into 'regression sum of squares' and 'residual sum of squares'. Furthermore, the value of the 'residual sum of squares' itself can be tested for significance against the 'within family mean square'. This test will indicate the presence or absence of dominance/epistasis (Kearsey and Pooni, 1996). The third method which estimates heritability in the broad-sense (\( h^2_b \)) is based on methods used by Allard (1960) and Hanson et al. (1956). The combined data of the two dates of 1995 and those of the two years 1995 and 1996 were analysed, as for a factorial experiment, to give two estimates of \( h^2_b \) as follows:

\[
h^2_b = \frac{\sigma^2_B}{\sigma^2_B + \sigma^2_E}
\]

Where \( \sigma^2_B \) and \( \sigma^2_E \) are the variance components due to differences between families or clones, families \( \times \) dates (or years) interactions and error respectively and \( r \), and \( f \) refer to the number of families, replications and dates (or years) of evaluation respectively; the denominator is the total phenotypic variance. Here, dates and years were substituted for environments.

Results

Disease evolution

The extent of flowering in the trial was low and there was not much difference in the rate of infection between flowered and vegetative stalks. All analyses
and results relate to non-flowered stalks. Figure 1 shows the evolution of yellow spot infection in 1995, in the two spreader varieties, B3337 and S17, in the experiment and in disease trials at UPSES (Figure 1a) and in 1996, in the control variety R570 in disease trials at Britannia (Figure 1b). In 1995, the evolution of the disease in both the experiment and the disease trials followed the same trend. Disease build-up started in March to reach a peak towards end April/ beginning May, and started to subside towards the end of June. The spreader variety B3337 which is also a standard in disease trials is considered as one of the most highly susceptible varieties (Ricaud, 1970; MSIRI, 1979) with peak infection nearing 48% in 1995 in the experimental trial. In 1996, the highest level of infection was reached end April/ beginning May, when the disease assessment was performed.

**Infection level in control varieties and parents**

The mean infection level for the resistant and highly susceptible control varieties in 1995 was 1.2% and 33.1% respectively and in 1996, it ranged from 0.7% to 45% respectively. The level of infection for all clones at the two dates of evaluation in 1995 was very close and the disease was more severe in 1996 compared to 1995. The assignment of clones to each of the four categories R, SS, S and HS in the Mauritius Sugar Industry Research Institute (MSIRI) disease testing program is based on the maximum rate of infection recorded for the respective clones over a three year assessment period and is as follows: 0–5%, resistant; >5–15%, slightly susceptible; >15–25%, susceptible; >25%, highly susceptible. The ratings pooled over all evaluation dates in the trial showed that the controls and parents could consistently be classified in the disease reaction classes assigned previously from disease trials. However, the absolute infection rate of a variety fluctuates owing to yearly variation of disease pressure and may increase to a level which is beyond the threshold infection class or may regress to below that level if conditions are either conducive or unfavourable to disease build-up. Generally, infection rates are higher when a susceptible variety is planted in a pure stand in commercial plantations (Ricaud et al., 1980; Ricaud et al., 1983) and may take epidemic proportions.

**Infection level in families**

There was a clear tendency for the mean infection rate of the progenies to increase as the combined susceptibility levels of the parents increased (Table 1). The lowest levels of infection were recorded for crosses among R and SS parents whilst infection levels were highest for the family resulting from the selfing of the highly susceptible male parent, R570. In 1996, a large percentage of progenies in all crosses with the highly susceptible male parent exceeded the threshold level of 15% at which economic losses are expected to occur (Autrey et al., 1983). For crosses among R and SS parents, 12–32% of progenies would exceed the threshold level of 15% infection. This percentage increased to 39 and 47 for S × SS crosses and from 71 to 86 for crosses between SS or S and the HS parent (Table 1). In 1995, the

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**Fig. 1**—Evolution of yellow spot infection (a) in spreader rows of varieties B3337 and S17 in experimental (exp) and pathology (path) trials at Union Park Sugar Experiment Station in 1995 (b) in control variety R570 in disease trials at Britannia in 1996.
proportion of progenies which exceeded the threshold infection level was much lower for all crosses as compared to 1996 and emphasised the need to assess for reaction over several years in presence of adequate disease pressure. Frequency distributions for yellow spot infection pooled over the three dates of evaluation over the two years are shown in Figure 2 for crosses with the resistant (M1277/79) and slightly susceptible (M596/78, M881/80) male parents (Figure 2a) and for crosses with the highly susceptible parent, R570 (Figure 2b). Generally, there was a fairly wide distribution of progenies in all infection classes and transgressive segregants in the lower (0–6%) and higher (24–30%) infection classes occurred in all categories of crosses. Crosses among R and SS parents were generally skewed with a majority of progeny grouped towards resistant or slightly susceptible classes (Figure 2a). The distribution of progenies within the majority of crosses which involved a susceptible or highly susceptible parent tended towards a binomial one (Figure 2b). The mode of segregation suggests the involvement of several genes with quantitative effect.

The partitioning of variances between and within families showed highly significant differences between families at all dates and for the pooled dates. Similarly, the regression mean squares were highly significant. Although the major part of this variation appeared to be additive, the values for the ‘residual mean squares’ were also significant, thus showing that the non-additive component (dominance/epistasis) was present. The estimated values of $h^2$ from regressions were generally high but lower than those obtained by the first method (Table 2). The highest value of $h^2$, for individual dates was obtained in 1996 when the infection was most severe, indicating that screening of varieties for disease infection is best conducted under heavy disease pressure.

The results obtained from the analysis of variance of parent varieties and families showed that infection level did not differ between the two dates of evaluation in 1995. However, differences in infection level between the two years 1995 and 1996 were highly significant, confirming that yearly variation in epiphytotics was more pronounced than variation between the two different dates of evaluation of the same year. Both parent x date and parent x year interactions were not significant. Similarly, neither family x date nor family x year interactions were significant. These results indicate that the relative infection rates of both parent clones and families do not change from one date to another in the same year or from one year to another. Differences between parent clones and families account for the major part of the total variance such that the corresponding broad-sense heritability for combined analysis was very high and ranged from 0.89–0.95.

**Discussion**

Absence of parent/family x date interaction within the year 1995 confirmed that no differential build-up of disease in either the parent clones or in the families occurred during the infection period. Differential disease build up can occur in highly flowering clones since there is no renewal of leaves after flowering and the percentage cover of the leaf laminae by the disease may continue to increase until complete defoliation occurs (Ricaud et al., 1983). On the other hand, vegetative stalks recover from the disease with the emergence of new leaves when conditions become unfavourable for disease expression. Similarly, no parent or family x year interaction was evident. This indicates that even in a year when infection was considered to be very high (1996), parent clones and families maintained their relative resistance/susceptibility levels. This was confirmed by the very high broad-sense heritability obtained for the analyses of combined dates.

The assignment of varieties in resistance/susceptibility classes in disease trials is based on a set of standards with well established disease reaction. An infection rate of 15% which is the threshold level beyond which economic losses will occur is set as the limit above which no commercial variety would be released for cultivation in the humid to very humid zones even if they are highly productive or better.

**Table 1—Yellow spot infection (%) for families pooled over replicates in 1995 and 1996 and percentage progeny exceeding 15% threshold infection level.**

<table>
<thead>
<tr>
<th>Variety</th>
<th>Infection %</th>
<th>% progeny &gt;15% infection</th>
<th>Infection %</th>
<th>% progeny &gt;15% infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>M134/75 × M1277/79</td>
<td>R × R</td>
<td>4.3 (90)</td>
<td>9</td>
<td>7.1 (68)</td>
</tr>
<tr>
<td>M134/75 × M596/78</td>
<td>R × SS</td>
<td>4.9 (67)</td>
<td>11</td>
<td>10.0 (74)</td>
</tr>
<tr>
<td>M937/77 × M596/78</td>
<td>SS × SS</td>
<td>6.0 (87)</td>
<td>9</td>
<td>11.1 (78)</td>
</tr>
<tr>
<td>M2077/78 × M881/80</td>
<td>S × SS</td>
<td>7.7 (29)</td>
<td>14</td>
<td>11.9 (36)</td>
</tr>
<tr>
<td>M2077/78 × M596/78</td>
<td>S × SS</td>
<td>4.9 (90)</td>
<td>2</td>
<td>13.7 (84)</td>
</tr>
<tr>
<td>M134/75 × R570</td>
<td>R × HS</td>
<td>10.4 (59)</td>
<td>27</td>
<td>14.6 (57)</td>
</tr>
<tr>
<td>M937/77 × R570</td>
<td>SS × HS</td>
<td>12.6 (52)</td>
<td>34</td>
<td>18.6 (92)</td>
</tr>
<tr>
<td>M887/70 × R570</td>
<td>S × HS</td>
<td>12.0 (88)</td>
<td>24</td>
<td>21.0 (83)</td>
</tr>
<tr>
<td>S17 × R670</td>
<td>S × HS</td>
<td>14.2 (84)</td>
<td>36</td>
<td>22.2 (76)</td>
</tr>
<tr>
<td>Q96 × R570</td>
<td>S × SS</td>
<td>13.6 (88)</td>
<td>39</td>
<td>23.8 (76)</td>
</tr>
<tr>
<td>R570 × R570</td>
<td>HS × HS</td>
<td>14.5 (68)</td>
<td>43</td>
<td>25.1 (51)</td>
</tr>
<tr>
<td><strong>Seasonal mean</strong></td>
<td></td>
<td>9.6</td>
<td></td>
<td>16.4</td>
</tr>
</tbody>
</table>

1 Pooled over April and June evaluations
2 Number of progenies evaluated
3 Threshold infection level at which economic losses occur
Fig. 2.—Frequency distributions for yellow spot infection pooled over three dates of evaluation in 1995 and 1996 (a) for crosses with resistant (M1277/78) and slightly susceptible (M586/78, M81/80) male parents (b) for crosses with the highly susceptible (RS70) parent.

Table 2—Estimates of variance components and heritability for yellow spot infection derived from methods 1 (components of variance) and 2 (offspring–parent regression) for individual dates and pooled dates in 1995 and 1996.

<table>
<thead>
<tr>
<th></th>
<th>σ²y</th>
<th>σ²w</th>
<th>σ²e</th>
<th>σ²h</th>
<th>h²_m</th>
<th>h²_p2</th>
</tr>
</thead>
<tbody>
<tr>
<td>April 1995</td>
<td>33.3</td>
<td>60.0</td>
<td>26.7</td>
<td>66.4</td>
<td>0.71</td>
<td>0.68 ± 0.12</td>
</tr>
<tr>
<td>June 1995</td>
<td>26.7</td>
<td>52.3</td>
<td>25.6</td>
<td>53.4</td>
<td>0.68</td>
<td>0.71 ± 0.10</td>
</tr>
<tr>
<td>May 1996</td>
<td>44.7</td>
<td>63.1</td>
<td>18.4</td>
<td>69.5</td>
<td>0.83</td>
<td>0.75 ± 0.10</td>
</tr>
<tr>
<td>Pooling</td>
<td>28.8</td>
<td>44.8</td>
<td>15.7</td>
<td>57.2</td>
<td>0.78</td>
<td>0.68 ± 0.10</td>
</tr>
<tr>
<td>All dates</td>
<td>33.5</td>
<td>34.9</td>
<td>1.4</td>
<td>67.0</td>
<td>0.98</td>
<td>0.71 ± 0.10</td>
</tr>
</tbody>
</table>

h²_m: Method 1 (Components of variance)

h²_p2: Method 2 (Offspring–parent regression)
than the standard commercial varieties. Under heavy disease pressure as in 1996, this limit was exceeded by 12–32% of progenies in crosses involving R and SS parents and by 70–86% of progenies in crosses between SS, S, HS female parents with the highly susceptible male parent. There was a clear indication that the mean susceptibility of the families increased with increasing susceptibility of the parents. Distribution of progenies for crosses which involved R and SS parents was skewed towards resistance and slightly susceptible classes and tended towards a binomial type with increasing susceptibility of the parents. The segregation pattern indicated an oligogenic to a quantitative type of inheritance as opposed to a monogenic system with minor genes as was reported for rust resistance in sugarcane (Daugrois et al., 1996; Ramdoyal et al., 2000). A minimum of 3–4-gene system in the control of yellow spot resistance was proposed in a preliminary study (Ramdoyal et al., 1996) where the degree of resistance was assumed to be dependent upon the number of loci bearing at least one dominant allele. However, the latter study was performed under reduced disease pressure and progeny that escaped disease might have been interpreted as resistant.

According to Van der Plank (1982), segregation of a few loci contributes most to the genetic variance in the quantitative expression of inheritance. An oligogenic system of disease resistance in sugarcane was suggested for downy mildew (Chu et al., 1959) and gumming (Stevenson, 1965) whereas multiple factors governed the inheritance of resistance to mosaic (Breaux and Fanguy, 1965).

Generally, the high narrow-sense heritability derived from the partitioning of the variances between and within families or offspring-parent covariance analyses indicated that the major part of the genetic variance would be additive. However, the significance of the residual term from the regression analyses suggested that interaction due to non-additive gene effects could also be expected. The performance of the crosses can be confidently predicted from the susceptibility levels of the parents. As a consequence of this study, computer-aided crossing programs for the very humid zones (Ramdoyal et al., 1999) have been revised to exclude susceptible and highly susceptible parents from all crosses planned for these zones. In future, greater emphasis will be placed on the selection of parents taking into account their infection rate in disease trials to decide on their use for specific adaptation in yellow spot prone areas. Since a major part of the variance appears to be additive, a base population improvement program through recurrent selection to accumulate resistant alleles would be necessary to increase the level of resistance of parent varieties. Concurrently, the search for molecular markers or quantitative trait loci (QTLs) linked to resistance to yellow spot in crosses evaluated in this study, using random amplified polymorphic DNA (RAPD) technology is underway (MSIRI, 1999). These studies could open new avenues to screen parents earlier than it is possible now.

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REFERENCES


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TRANSMISSION DE LA RESISTANCE AU 'YELLOW SPOT' CHEZ LES DESCENDANCES ISSUES DE GRAINES CHEZ LA CANNE A SUCRE

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Résumé

La transmission de la résistance au 'yellow spot' chez la canne à sucre fut étudiée à partir de onze familles au cours de trois dates d'évaluation sur deux années consécutives. L'étude démontra que les témoins aussi bien que les parents pouvaient être classés systématiquement dans les mêmes catégories de sensibilité allouées au cours des essais établis précédemment. Le niveau d'infection absolue des témoins, parents et familles fluctuait en fonction de la variation saisonnière du taux d'infection. Le niveau d'infection absolue des descendances augmentait avec l'augmentation du niveau de sensibilité combinée des parents. Les valeurs de l'héritabilité au sens strict calculées à partir de la décomposition de la variance entre et à l'intérieur des familles, variaient de 0.68–0.71 quand le taux d'infection était relativement faible, et atteignaient 0.83 quand l'infection était sévère pour se situer autour de 0.98 quand elles furent estimées à partir des données combinées sur toutes les dates d'évaluation. Les distributions des fréquences combinées sur toutes les dates d'évaluation font apparaitre une large ségrégation avec des individus transgressant dans les classes de taux de sensibilité basse aussi bien qu'élévée chez toutes les familles. La distribution était biaisée vers les classes de sensibilité faible chez les familles issues des parents résistants et légèrement sensibles alors qu'elle approchait une distribution normale chez les familles comportant un parent sensible ou très sensible.

Le mode de ségrégation indiqua un modèle oligogénique avec souvent une expression quantitative pour la réaction au yellow spot. Les parents et les familles différaient largement quant au niveau d'infection alors que les interactions parent × date ou année, et famille × date ou année n'étaient pas importantes ce qui démontra une valeur d'héritabilité au sens large de 0.89–0.95. Les conclusions de cette étude dans l'élaboration d'une stratégie appropriée pour améliorer la résistance au yellow spot sont discutées.

Mots clés: Canne à sucre, héritabilité, Mycovellosiella koepkei, résistance, sélection améliorante.
HERENCIA DE LA RESISTENCIA A LA PECA AMARILLA EN POBLACIONES SEGREGANTES DE CANA DE AZUCAR

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Resumen
La herencia de la resistencia a la peca amarilla en once familias de caña de azúcar (padres y progenie) se estudió durante tres evaluaciones de la enfermedad en un período de dos años. El estudio demostró que tanto el testigo como los padres fueron clasificados de manera consistente en los niveles asignados de reacción en estudios anteriores. La tasa de infección absoluta del testigo o padres y sus familias fluctuó de acuerdo con el efecto de la estación en la presión del inoculo. Hubo una clara tendencia de aumento de la tasa media de infección de la progenie a medida que la susceptibilidad combinada de los padres aumentó. La estrecha herencia derivada de la división de la varianza entre y dentro las familias varió de 0.68–0.71 cuando la infección estuvo relativamente baja y aumentó a 0.83 bajo una presión severa de inoculo y llegó a ser muy alta, 0.98, cuando se unieron todos los datos de las diferentes evaluaciones. La distribución de frecuencia de todos los datos unidos de las diferentes evaluaciones mostró una distribución muy amplia de progenies con la presencia de segregantes transgresivos en la parte baja y alta de las clases de susceptibilidad en todas las categorías de cruzamientos. La distribución estuvo achatada hacia las clases de menor infección en el caso de cruzamientos entre padres resistentes y levemente susceptibles, mientras que tendió a una distribución binomial en el cruzamiento entre padres susceptible o altamente susceptible. El modo de segregación mostró un sistema oligogénico con tendencia a una expresión cuantitativa para la característica. Los padres y familias difirieron ampliamente con respecto al nivel de infección sin embargo las interacciones de padre × fecha o año y familia × fecha o año no fueron importantes dentro del mismo año de evaluación o entre años, lo cual indicó una muy herencia muy alta de 0.89–0.95. Se discute el significado de los resultados en el diseño de una estrategia de mejoramiento para la resistencia a la peca amarilla.

Palabras claves: Cana de azúcar, mejoramiento, herencia, Mycovelllosiella koepkei, resistencia.