

Fiji disease resistance in sugarcane: Relationship to cultivar preference in field populations of the planthopper vector *Perkinsiella saccharicida*

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Summary

Populations of the planthopper vector *Perkinsiella saccharicida* on sugarcane cultivars resistant (cvs Q110 and Q87), moderately resistant (cvs Q90 and Q124) and susceptible (cvs NCo310 and Q102) to Fiji disease with known field resistance scores were monitored on the plant (2000-2001) and ratoon (2001-2002) crops. In both crops, the vector population remained very low, reaching its peak in the autumn. The vector population was significantly higher on cultivars susceptible to Fiji disease than on cultivars moderately resistant and resistant to Fiji disease. The number of *P. saccharicida* adults, nymphs and oviposition sites per plant increased with the increase in the Fiji disease susceptibility. The results suggest that under low vector density, cultivar preference by the planthopper vector mediates Fiji disease resistance in sugarcane. To obtain resistance ratings in the glasshouse that reflect field resistance, glasshouse-screening trials should be conducted under both low and high vector densities, and the cultivar preference of the planthopper vector recorded along with Fiji disease incidence.

Key words: Fiji disease, planthopper vector, *Perkinsiella saccharicida*, sugarcane, resistance, cultivar preference

Introduction

Planthoppers of the genus *Perkinsiella* (Hemiptera: Delphacidae) transmit *Fiji disease virus* (Reoviridae) (FDV) causing Fiji disease in sugarcane (Hughes & Robinson, 1961; Hutchinson & Francki, 1973). *Perkinsiella saccharicida* Kirkaldy is the vector of FDV in Australia (Mungomery & Bell, 1933; Francki & Grivell, 1972). Sugarcane infected with FDV shows leaf galls and distortion, death of meristematic tissue and stunting, resulting in severe yield reductions (Egan & Ryan, 1986). Fiji disease is managed through the identification and exploitation of plant resistance (Egan & Fraser, 1977; Egan & Ryan, 1986; Ryan, 1988).

Studies on Fiji disease so far have focused mainly on plant resistance to the disease, with limited attention to whether the resistance is to the vector or the virus. Cultivar preference by *Perkinsiella vitiensis* Kirk. influenced the Fiji disease susceptibility ratings of sugarcane in Fiji (Husain *et al.*, 1967). But Taniguchi *et al.* (1980) reported no relationship between Fiji disease resistance ratings and survival and development of *P. saccharicida* nymphs, even though the survival and development of *P. saccharicida* differed significantly between cultivars. Studies on the feeding patterns of the vector show that the susceptibility of sugarcane cultivars

to Fiji disease is related to the proportion of time spent on phloem feeding by *P. saccharicida* (Chang & Ota, 1978). Candy *et al.* (2001) reported that resistance to Fiji disease in sugarcane is not mediated via a gene-for-gene system, and suggested that it could be mediated either via resistance to the planthopper vector or via a more general biotic/abiotic response mechanism. Recent glasshouse studies in Australia suggest that the resistance to Fiji disease in sugarcane is mediated by cultivar preference of the planthopper vector (Dhileepan & Croft, 2003).

In the field, sugarcane cultivars have a strong influence on the planthopper vector population, and cultivars highly susceptible to Fiji disease had the highest vector population (Bull, 1977, 1981). Cultivar preference and performance of *P. saccharicida* in the glasshouse differed significantly from the field conditions (Bull, 1977), and there was no relationship between the cultivar preferences by the planthopper and the Fiji disease resistance scores in the field (Bull, 1977). All previous studies on planthopper vector populations were carried out in the 1970s when the vector population was high, with several hundred planthoppers per plant (Bull, 1977, 1981). The vector population in the field has been low in the 1980s and 1990s and this may be associated with the removal the highly Fiji disease

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susceptible variety, NCo310 (Egan *et al.*, 1989). Resistance screening methods for arthropods and pathogens are usually population or inoculum density dependent (Harris, 1979). At low vector density, sugarcane cultivars resistant and moderately resistant to Fiji disease attract fewer planthoppers than the susceptible cultivars, but no distinction in the preference between cultivars of different resistant status was evident at high vector density in the glasshouse (K Dhileepan, unpublished data). This suggests that vector density has a significant influence on cultivar preference by planthoppers in the glasshouse. This has not been tested in the field. With the current low vector populations in the field, cultivar preference by the vector and its impact on Fiji disease resistance in sugarcane is unknown. Hence, in this study, the planthopper vector population was monitored on sugarcane cultivars resistant, moderately resistant and susceptible to Fiji disease with known field resistance scores for two seasons (2000-2001 & 2001-2002).

Materials and Methods

Field trial

A field trial was conducted at the Bureau of Sugar Experiment Stations (BSES), Woodford with 10 standard sugarcane cultivars with known field resistance scores for Fiji disease. Pre-germinated plants established from single-eye cuttings of sugarcane cultivars were planted in the field using a water wheel planter in a randomised complete block design with five plots of each cultivar and eight plants in each plot spaced at 25-30 cm intervals. Standard cultivars and test plants were planted in a dual-row format between rows of Fiji disease infected sugarcane cultivars (WD1 and WD2) (Egan *et al.*, 1989). Plants were fertilised at planting in late September 2000 (83 kg N ha⁻¹), in December 2000 (125 kg N ha⁻¹), and in December 2001 (125 kg N ha⁻¹). The plants were ratooned (cut at soil level) in September 2001 and allowed to regrow. Meteorological data was recorded in an automatic weather station located near the field.

Planthopper sampling

Within the field trial, planthoppers were sampled on sugarcane cultivars resistant (cvs Q110 and Q87), moderately resistant (cvs Q90 and Q124) and susceptible (cvs NCo310 and Q102) to Fiji disease. On a scale of 1 (resistant) to 9 (susceptible), the field resistance scores for cvs Q110, Q87, Q90, Q124, NCo310 and Q102 are 1, 2, 4, 6, 8 and 9, respectively. Planthopper populations were monitored at fortnightly intervals during 2000-2001 and at monthly intervals in 2001-2002. The number of adults, nymphs and oviposition sites on a randomly selected stalk in each of the eight plants

in all five plots were counted. Planthoppers are uncommon from June (late autumn) to November (late spring) (Bull, 1981) and hence the sampling was carried out from December (early summer) to May (late autumn). For each plant, on all sampling occasions, oviposition sites, including the previous oviposition sites, were recorded on all leaves, excluding dried and shed leaves.

Fiji disease scoring

All test plants were inspected for Fiji disease in February 2002, and the number of plants with Fiji disease symptoms was recorded on the basis of visible gall symptoms. Galls vary in size, shape and colour, and occur anywhere on the underside of the leaf, including the mid rib and the leaf sheath. Once gall symptoms were found on one stalk in a plant, then that plant was classified as Fiji disease infected without any further inspection of other stalks in the plant. Disease incidence was only recorded in the ratoon crop because the symptoms are difficult to detect in the plant crop.

Data analysis

Variation in the number of *P. saccharicida* nymphs and adults per stalk in relation to sugarcane cultivar, plot and year were analysed using General Linear Model Repeated Measures ANOVA and the main effects compared. Monthly sampling data on the vector population for each plant was used as the within-subjects variable, and the cultivars, plots and years were treated as between-subjects variables. Tukey's Test was employed to compare the mean values among cultivars. Variation in the number of *P. saccharicida* oviposition sites per stalk at the end of the season (May) in relation to sugarcane cultivar and year were analysed using two-way ANOVA and the main effects compared using Tukey's test. Regression analysis was employed to study the interactions between Fiji disease resistance scores, the number of *P. saccharicida* adults, nymphs and oviposition sites per plant, and percentage of plants with Fiji disease.

Results

The planthopper population remained very low in both 2000-2001 (plant crop) and 2001-2002 (ratoon crop) seasons, reaching its peak in the autumn (Fig. 1). The population of adults peaked two months earlier in 2002 than in 2001. The average number of *P. saccharicida* adults per stalk did not differ significantly ($F_{1,370} = 1.6$, $P = 0.214$) between 2000-2001 (0.45 ± 0.02) and 2001-2002 (0.42 ± 0.03) seasons. But the average number of *P. saccharicida* nymphs per stalk was significantly higher ($F_{1,370} = 47.2$, $P < 0.001$) in 2001-2002 (0.11 ± 0.01) than in 2000-2001 (0.02 ± 0.003). Oviposition by *P.*

saccharicida was evident throughout the summer and autumn (Fig. 1), and the total number of oviposition sites per stalk at the end of the season was 16% higher ($F_{1,370} = 11.8, P < 0.001$) in 2000-2001 (14.1 ± 0.39) than in 2001-2002 (12.2 ± 0.41).

The number of *P. saccharicida* adults ($F_{5,370} = 17.2, P < 0.001$), nymphs ($F_{5,370} = 4.4, P < 0.001$) and oviposition sites ($F_{5,370} = 33.1, P < 0.001$) per stalk differed significantly between cultivars. The number of adults per stalk (plant and ratoon crops combined) was significantly higher on susceptible cvs Q102 (0.61 ± 0.02) and NCo310 (0.53 ± 0.03), than on moderately resistant cvs Q90 (0.32 ± 0.03) and Q124 (0.41 ± 0.02) and resistant cvs Q110 (0.35 ± 0.02) and Q87 (0.36 ± 0.03) (Fig. 2). The number of

nymphs per stalk (plant and ratoon crops combined) was significantly higher on the susceptible cvs Q102 (0.08 ± 0.01) and NCo310 (0.06 ± 0.009) than on the resistant cv. Q110 (0.032 ± 0.008) and moderately resistant cv. Q124 (0.021 ± 0.01) (Fig. 2). The total number of oviposition sites per stalk (plant and ratoon crops combined) at the end of the season was significantly higher on susceptible cvs Q102 (17.2 ± 0.69) and NCo310 (18.9 ± 0.67) than on moderately resistant cvs Q90 (9.2 ± 0.72) and

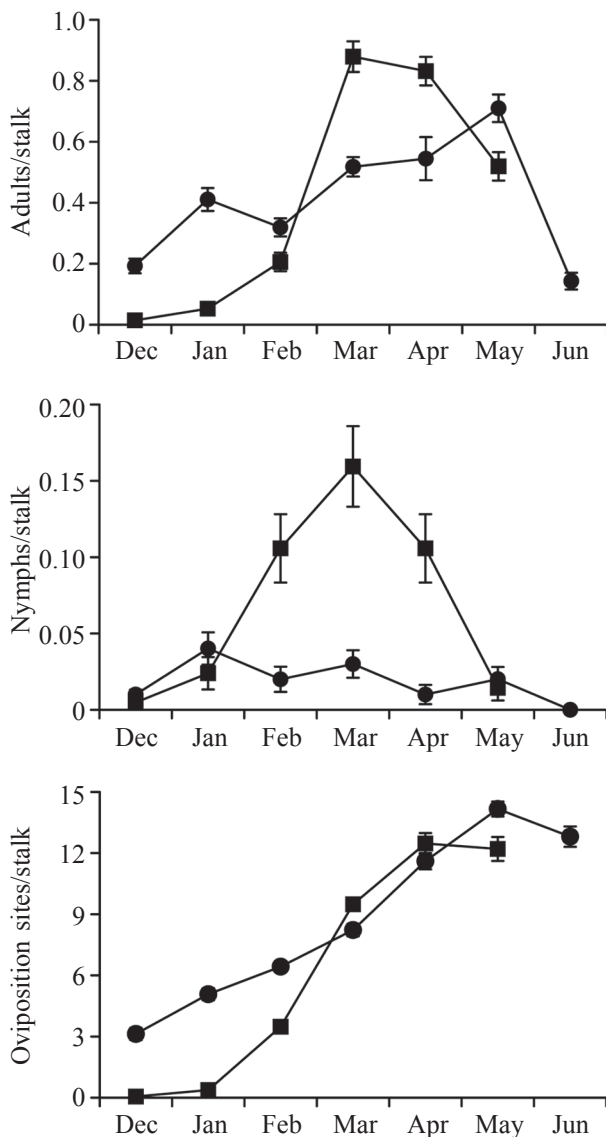


Fig. 1. Seasonal variation in the number of *P. saccharicida* adults, nymphs and oviposition sites per stalk in 2000-2001 (●) and 2001-2002 (■) seasons. The number of oviposition sites per stalk represents the cumulative value. Vertical bars represent standard error.

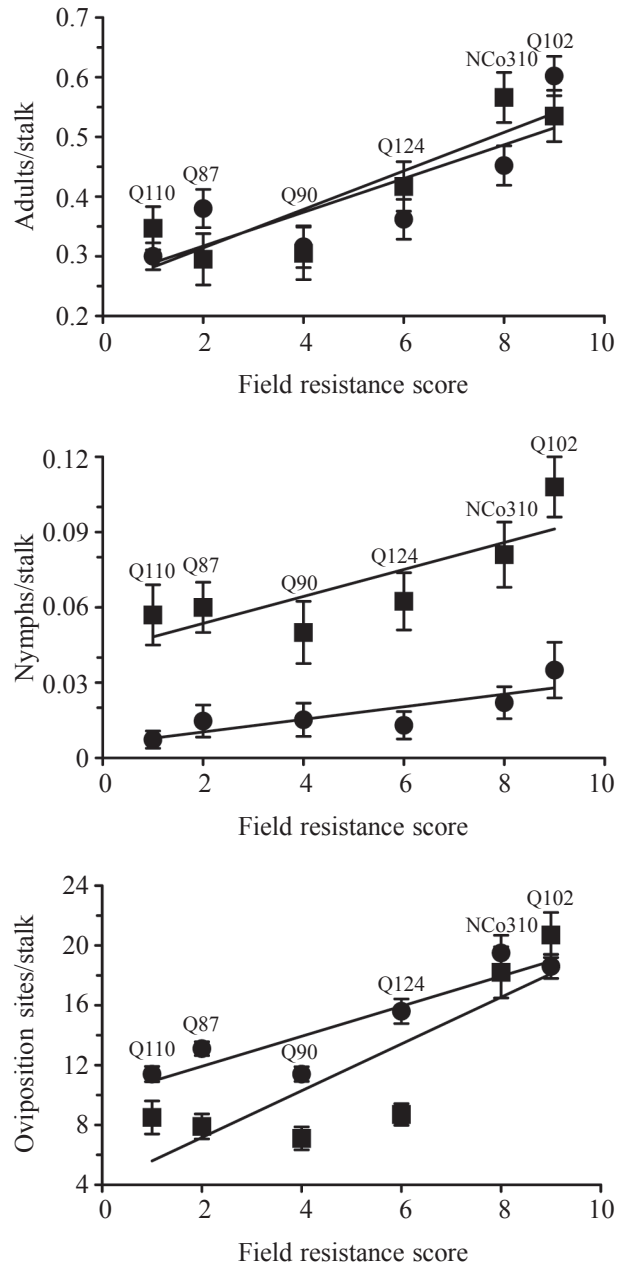


Fig. 2. Relationship between Fiji disease resistance scores and the mean number of *P. saccharicida* adults, nymphs and oviposition sites per stalk in 2000-2001 (●) and 2001-2002 (■) seasons. Fiji disease resistance scores for standard sugarcane cultivars: Q110 = 1; Q87 = 2; Q90 = 4; Q124 = 6; NCo310 = 8; and Q102 = 9. Vertical bars represent standard error.

Q124 (12.2 ± 0.67) and resistant cvs Q110 (9.9 ± 0.66) and Q87 (10.5 ± 0.68) (Fig. 2).

In both 2000-2001 and 2001-2002, the number of *P. saccharicida* adults (2000-2001: $y = 0.26 + 0.03x$, $R^2 = 0.67$, $P < 0.05$; 2001-2002: $y = 0.25 + 0.03x$, $R^2 = 0.79$, $P = 0.02$), nymphs (2000-2001: $y = 0.005 + 0.003x$, $R^2 = 0.71$, $P = 0.04$; 2001-2002: $y = 0.04 + 0.01x$, $R^2 = 0.66$, $P = 0.05$) and oviposition sites (2000-2001: $y = 9.9 + 1.01x$, $R^2 = 0.83$, $P = 0.01$; 2001-2002: $y = 4.03 + 1.56x$, $R^2 = 0.71$, $P = 0.03$) in sugarcane cultivars increased with the increase in the Fiji disease susceptibility (Fig. 2). Only six of the 4680 test plants (0.13%) in the trial contracted Fiji disease and no Fiji disease incidence was evident in any of the standard cultivars.

Discussion

The population of planthopper during this study was very low (< 1 adult per stalk) which resulted in low transmission of Fiji disease. In the 1970s when the last major Fiji disease outbreak occurred in Australia (Egan *et al.*, 1989), vector populations of several hundred planthoppers per stool were recorded (Bull, 1977, 1981). There has been no study of the reasons for the decline in planthopper populations over this period but it has been speculated that the removal of the cultivar NCo310, which was favourable to the insect (Bull, 1981), may be partly responsible (Egan *et al.*, 1989).

The results suggest that sugarcane cultivars highly susceptible to Fiji disease are also highly favourable to *P. saccharicida* and cultivars resistant to Fiji disease are the least preferred by *P. saccharicida*. Under low vector densities, both the vector population and the Fiji disease incidence increased with the increase in Fiji disease susceptibility. In an earlier glasshouse trial using sugarcane seedlings, the cultivar preference by *P. saccharicida* increased with the increase in Fiji disease susceptibility (Dhileepan & Croft, 2003). This is the first time such a preference has been reported in fully-grown field plants. Plant resistance to gall mite vector (*Cecidophyopsis ribis* Westwood) in blackcurrant also provides a high level of protection against blackcurrant reversion disease (Jones *et al.*, 1998). *P. saccharicida* adults spend significantly more time in phloem ingestion on cultivars susceptible to Fiji disease than on cultivars resistant to Fiji disease (Chang & Ota, 1978). In Fiji, cultivar preference by *P. vitiensis* influenced the Fiji disease susceptibility ratings of sugarcane (Husain *et al.*, 1967). Cultivar preference in planthoppers appears to be due to specific probing stimulants in the host plant, which facilitate more frequent phloem location and feeding on susceptible plant varieties compared to resistant ones (Cook & Denno, 1994).

In the 1970s when the *P. saccharicida* population

in the field was very high (> 150 planthoppers per stalk), the vector populations were significantly higher on the susceptible cv. NCo310 than on resistant cultivars (Bull, 1977, 1981). However, a re-analysis of Bull's data revealed no relationship between Fiji disease resistance scores and the populations of *P. saccharicida* adults (1974: $R^2 = 0.06$, $P = 0.46$; 1976: $R^2 = 0.11$, $P = 0.39$) and nymphs (1974: $R^2 = 0.28$, $P = 0.11$; 1976: $R^2 = 0.05$, $P = 0.58$). In the current study at low vector populations, the cultivar preference by the planthopper vector in the field increased with the increase in Fiji disease susceptibility. Resistant plants may become susceptible at high insect densities resulting in a narrow difference between susceptible and resistant plants (Panda & Khush, 1995). However, at both low and high vector densities in the glasshouse, susceptible cv. Q102 attracted higher number of adult planthoppers than the other cultivars (K Dhileepan, unpublished data). Susceptible plants are known to clump towards the susceptible end of the spectrum at both high and low insect densities (Panda & Khush, 1995).

In the glasshouse, the level of Fiji disease transmission was dependent on the number of planthoppers per plant (Dhileepan & Croft, 2003). As a result, cultivars with more planthoppers had a higher Fiji disease incidence, and hence were categorised as susceptible in comparison to those cultivars with fewer vectors resulting in a lower Fiji disease incidence. It appears that cultivars known to be resistant at low vector densities may end up with vector numbers similar to cultivars moderately resistant or susceptible at high vector densities, resulting in higher Fiji disease incidence and conflicting resistance rating. This could be one of the reasons why glasshouse-based resistance screening trials, which usually use high vector densities (Husain & Hutchinson, 1971; Ledger & Ryan, 1977), yield resistance ratings different to the field resistance.

The level of Fiji disease transmission in the field resistance trials in the 1980s and 1990s was very low (0.7-4.1%). In the current study, the vector population was so low that the probability of planthopper carrying the virus from the infected sugarcane plants (WD1 and WD2) to the test cultivars would be extremely low. *P. saccharicida* is an inefficient vector of Fiji disease (Baber & Robinson, 1950) and less than half the planthoppers with FDV transmit the disease (Francki *et al.*, 1985). The use of tolerant cv. WD1 as the virus source plant in the field could have also affected the Fiji disease transmission in the field. FDV incidence in *P. saccharicida* was significantly lower (9%) when the tolerant cv. WD1 was used as the virus-source plant than when the susceptible cv. NCo310 was used (60%) as the virus source plant (Dhileepan *et al.*,

personal communication).

Resistance screenings for Fiji disease in the field since the mid 1980s have been based on low vector densities, and the resistance/susceptibility of these cultivars in the field under high vector densities is unknown. A glasshouse screening method was considered unreliable, as the resistance ratings obtained in the glasshouse did not reflect the resistance in the field (Egan *et al.*, 1989), and some cultivars shown to be highly susceptible under glasshouse conditions appeared to be resistant in the field (Reimers *et al.*, 1982). The reasons for the failure of the glasshouse method are not fully understood. This study suggests that cultivar preference is important in resistance and that any screening method should allow for this character. To obtain resistance ratings in the glasshouse that reflect field resistance, it is recommended that glasshouse-screening trials should be conducted under both low and high vector densities, and the cultivar preference by the planthopper recorded along with Fiji disease incidence.

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References

- Baber E G, Robinson P E. 1950.** Feeding habits of *Perkinsiella saccharicida*. *Proceedings of the International Society of Sugar Cane Technologists* 7:155-167.
- Bull R M. 1977.** Leafhopper populations on some sugar-cane varieties. *Proceedings of the Queensland Society of Sugar Cane Technologists Conference* 44:83-87.
- Bull R M. 1981.** Population studies on the sugar cane leafhopper (*Perkinsiella saccharicida* Kirk.) in the Bundaberg District. *Proceedings of the Australian Society of Sugar Cane Technologists* 3:293-303.
- Candy J M, Croft B J, Brumley S M, Smith G R. 2001.** The identification of differentially expressed genes in sugarcane following infection with Fiji disease *fjivirus*. *Proceedings of the International Society of Sugar Cane Technologists* 24:621-623.
- Chang V C S, Ota A K. 1978.** Feeding activities of *Perkinsiella* leafhoppers and Fiji disease resistance of sugarcane. *Journal of Economic Entomology* 71:297-300.
- Cook A G, Denno R F. 1994.** Planthopper/plant interactions: Feeding behavior, plant nutrition, plant defense and host plant specialization. In *Planthoppers – Their Ecology and Management*, pp. 114-139. Eds R F Denno and T J Perfect. New York: Chapman & Hall.
- Dhileepan K, Croft B J. 2003.** Resistance to Fiji disease in sugarcane: Role of cultivar preference by planthopper vector *Perkinsiella saccharicida* (Homoptera: Delphacidae). *Journal of Economic Entomology* 96:148-155.
- Egan B T, Fraser T K. 1977.** The development of the Bundaberg Fiji disease epidemic. *Proceedings of the Queensland Society of Sugar Cane Technologists Conference* 44:43-48.
- Egan B T, Ryan C C. 1986.** Predicting Disease Incidence and Yield Losses in Sugarcane in a Fiji Disease Epidemic. In *Plant Virus Epidemics: Monitoring, Modelling and Predicting Outbreaks*, pp. 443-457. Eds G D McLean, R G Garrett and W G Ruesink. Sydney: Academic Press.
- Egan B T, Ryan C C, Francki R I B. 1989.** Fiji Disease. In *Diseases of Sugarcane - Major Diseases*, pp. 263-287. Eds C Ricaud, B T Egan, A G Gillaspie and C G Hughes. Amsterdam: Elsevier.
- Francki R I B, Grivell C J. 1972.** Occurrence of similar particles in Fiji disease virus-infected sugarcane and insect vector cells. *Virology* 48:305-307.
- Francki R I B, Ryan C C, Hatta T, Rohozinski J, Grivell C J. 1985.** The inefficiency of Fiji disease virus transmission by *Perkinsiella saccharicida* Kirk., the planthopper vector. *Plant Disease* 35:324-328.
- Harris M K. 1979.** Arthropod-plant interactions related to agriculture, emphasizing host plant resistance. In *Biology and Breeding for Resistance to Arthropods and Pathogens in Agricultural Plants*, pp. 23-51. Ed. M K Harris. Texas: A & M University Press.
- Hughes C G, Robinson P E. 1961.** Fiji Disease. In *Sugarcane Diseases of the World*, Vol 1, pp. 389-405. Eds J P Martin, E V Abbott and C G Hughes. Amsterdam: Elsevier.
- Husain A A, Hutchinson P B. 1971.** Further experience with the insectary method of testing sugarcane varieties for resistance to Fiji disease. *Proceedings of the International Society of Sugar Cane Technologists* 14:1001-1006.
- Husain A A, Brown A H D, Hutchinson P B, Wismer C A. 1967.** The testing of sugarcane varieties for resistance to Fiji disease in Fiji. *Proceedings of the International Society of Sugar Cane Technologists* 12:1154-1164.
- Hutchinson P B, Francki R I B. 1973.** Sugarcane Fiji Disease Virus. CMI/AAB Description of Plant Viruses No. 119.
- Jones A T, Brennan R M, McGavin W J, Lemmetty A. 1998.** Gall and reversion disease incidence in a range of blackcurrant genotypes, differing in resistance to the blackcurrant gall mite (*Cecidophyopsis ribis*) and blackcurrant reversion disease. *Annals of Applied Biology* 133:375-384.
- Ledger P E, Ryan C C. 1977.** Screening of sugarcane varieties for resistance to Fiji disease in Queensland: the insectary-glasshouse method. *Proceedings of the Queensland Society for Sugar Cane Technologists* 44:79-82.
- Mungomery R W, Bell A F. 1933.** Fiji disease of sugar cane and its transmission. *Bureau of Sugar Experiment Stations, Division of Pathology*, Bulletin No. 4.
- Panda N, Khush G S. 1995.** Host plant resistance to insects. UK: CAB International & Philippines: International Rice Research Institute. 431 pp.
- Reimers J F, Hall P, Hogarth D M. 1982.** The relationship between Fiji disease susceptibility and yield. *Proceedings of the Australian Society of Sugar Cane Technologists* 4:103-110.
- Ryan C C. 1988.** Epidemiology and control of Fiji disease virus of sugarcane. In *Advances in Disease Vector Research*, Vol. 5, pp. 163-176. New York: Springer-Verlag.
- Tanaguchi G V, Ota A K, Chang V C S. 1980.** Effects of Fiji disease-resistant sugarcane (*Saccharum* sp.) on the biology of the sugarcane Delphacid. *Journal of Economic Entomology* 73:660-663.