BSES Limited

FINAL REPORT – SRDC PROJECT BSS267

MAXIMISING WHOLE-OF-INDUSTRY BENEFITS FROM THE AUSTRALIAN SUGARCANE IMPROVEMENT PROGRAM THROUGH AN OPTIMAL GENETIC EVALUATION SYSTEM

by

X WEI, J STRINGER, P JACKSON and M COX

SD07009

Contact:
Xianming Wei
Senior Researcher
BSES Limited
Peak Downs Highway,
Te Kowai Qld 4740
Telephone: 07 4963 6825
Facsimile: 07 4954 5167
Email: xwei@bses.org.au

BSES Limited Publication
SRDC Final Report SD07009 **December 2007**

CONTENTS

Page No

SUMMARY

An optimal genetic evaluation system (GES) is the backbone of any breeding program because maximising genetic gains is primarily a matter of efficient selection. A GES provides information to breeders about which individuals should be selected as parents for crossing and which ones should be selected for commercial production.

At the commencement of this project, selection of both parents and clones for commercial production was principally based on the index known as net merit grade (NMG). NMG is based on the performance of a test clone (or a cross) relative to the average of a number of commercial varieties (or crosses) for the traits of commercial cane sugar (CCS), tonnes of cane per hectare (TCH), appearance grade and fibre content. NMG was used to generate a breeding code for selecting parental clones for crossing. Cross ratio, a measure of each cross's performance relative to the whole population at each selection stage, was used to determine priority of crosses. For selecting elite clones to be retained for further testing, NMG was used in all three selection stages to determine which clones would be advanced to next stage.

Some important weaknesses concerning the use of the NMG selection index were well known to sugarcane breeders at the beginning of this project. The key issues were:

- NMG was developed over 30 years ago and the weightings given to the different traits used in its calculation may not accurately reflect the economic value of traits relevant to modern sugarcane production systems.
- Selection must be practiced independently for NMG and other traits of commercial significance such as disease resistance. It has been shown that independent selection is inferior to index selection that involves simultaneous selection for multiple traits to maximize genetic gain (Cotterill and Dean 1990; Hazel *et al.* 1994).
- There was no clear definition of the association between breeding objectives and selection indices used to select parents and clones for further testing. Traits relevant to breeders (eg. TCH in small plots) were, in general, only predictors of the commercially important traits (eg TCH in pure stand). This difference has confounded the development of breeding strategies that maximise the genetic advance from selection.
- NMG did not take advantage of genetic correlations between traits and environments, nor did it take account of highly variable data quality across trials and traits.
- NMG and the prediction of breeding values did not use the complete pedigree information.

This project was developed to address these issues. The principal aim of the project was to develop and implement a new GES with the objective of maximising the economic return for the Australian sugarcane industry through genetic improvement. This system was developed through addressing its two essential components: (i) definition of breeding objectives and (ii) predicting breeding and genetic values for traits associated with those breeding objectives through their selection criteria.

Economic weights of the selection criteria were derived from a model developed for each major sugarcane producing region in Australia using information provided by a consultative group comprising industry experts specifically identified for their expertise to contribute to this project. The key objects in the model were the income and cost structures for the entire production processing and marketing chain, thereby encompassing the crop production, milling and marketing costs and revenues. These costs and incomes were linked with sugarcane traits. By changing one unit of a trait while holding other traits constant, the change in cost of producing one tonne of sugar became the economic weight of the trait. This approach assumes the principle industry objective is to minimise the cost of producing sugar. The value of different traits in a breeding program is then based on their relative contribution toward achieving this objective.

Breeding and genetic values were predicted using one of the most sophisticated statistical models currently available to any breeding program, using the computer software ASReml. For predicting breeding value, each quantitative trait took account of spatial variation at the trial level, genetic correlations among regions and all available pedigree information. For predicting genetic values, the model accounted for spatial and/or competition effects at the trial level, and genetic correlations between multiple harvests of sugarcane trials and between sites.

The new GES will produce higher genetic gains than NMG. Simulation studies were used to show that if 96 parental clones are selected from 481 candidates, an average of 19% more genetic gain can be expected from the progeny selected from crosses among those selected parents when the selection is based on GES rather than on NMG. Similarly, for 25 clones selected from a BSES-CSIRO variety improvement program final assessment trial comprising 150 clones, an average of 29% more gain would be expected when selection is practiced using GES rather than NMG.

We have implemented the GES in the BSES-CSIRO variety improvement program. GES was used to select parental clones for 2008 crossing and it guided the choice of the most desirable cross combinations in the 2007 season. It was also used in all regional selection programs in 2007 to make selection decisions, using variety data files generated from GES. The BSES-CSIRO variety improvement program database SPIDNet has been updated and improved to handle the new analyses and produce vastly improved reports.

1.0 BACKGROUND

An optimal genetic evaluation system (GES) is the backbone of any breeding program and, as (Cotterill and Dean 1990) pointed out, maximising genetic gain in advanced generation breeding is primarily a matter of efficient selection. A GES provides essential information to breeders on which individuals should be selected as parents for crossing and which genotypes should be retained for further testing or used for commercial production. Such a system firstly requires a clear definition of breeding objectives so that the economic importance of traits can be determined. It also requires genetic effects for these traits to be predicted in an optimal way based on all relevant information from field trials and molecular markers.

Until the commencement of the GES project, both the selection of clones for parental use and commercial performance relied on the index known as Net Merit Grade (NMG). NMG is a selection index that measures performance of a test clone (or a cross) relative to the average of commercial varieties (or crosses) in terms of commercial cane sugar (CCS), tonnes of cane per hectare (TCH), appearance grade and fibre content.

NMG is calculated as:

$$NMG_i = appGrade_i * \left(\frac{TSH_i}{TSH_s} + 0.03 * (CCS_i - CCS_s) \right) - 0.13 * (Fibre_i - Fibre_s) \qquad [1]$$

where:

NMG_i is the NMG of the i^{th} clone in a trial;

$appGrade_i$ is the appearance grade of clone i (a score 1-9);

TSH_i is the Tonnes of sugar per hectare of clone i;

CCS_i is the CCS of clone i;

$Fibre_i$ is the fibre content of clone i;

TSH_s is the average tonnes of sugar per hectare of the standards;

CCS_s is the average CCS of the group of standards; and

$Fibre_s$ is the average fibre content of the group of standards.

This selection system, and the way in which NMG is applied to identify elite clones for further testing and elite parents, is described below.

Crossing is primarily undertaken at Meringa, with complementary work in Bundaberg. Prior to undertaking crossing, a breeding code that is based on a clone's NMG and disease ratings was used to determine if a clone should be selected as a parent. Cross ratios (a measure of each cross's performance relative to the whole population at each selection stage) were used to prioritise crosses. This was done by denoting crosses with a higher cross ratio as proven crosses. Proven crosses were completed with higher priority than those crosses with lesser cross ratios.

Selection trials on populations of seedlings or clonal progeny are conducted in four regional selection programs. The four regional programs are aligned with production areas along the coast of Queensland and northern New South Wales. The regions are: (i) Southern based in Bundaberg (which also includes northern New South Wales), (ii) Central in Mackay, (iii) Burdekin in Ayr and (iv) Northern based in Meringa. Until 2004, the Herbert program (Ingham) was a separate regional program. In that year, the Herbert program was amalgamated with the Northern regional program.

There are three selection stages (Cox et al. 2000). The first stage of selection is called the Progeny Assessment Trials (PATs). These trials test single plants grown from true seed. Approximately 250-350 full-sib families with 80-100 seedlings per family are evaluated in either single or three row plots with 2-5 replications. TCH and CCS of each plot are measured on the plant crop. Prior to the GES project, Best Linear Unbiased Predictors (BLUPs) were derived on a single trial basis and then used to derive NMG for families. The top 40-50% of families were chosen for further selection in the first ratoon crop via clonal within-family selection. Larger numbers of clones are selected from families with higher NMG than from families with lower NMG.

The second stage of selection is called the Clonal Assessment Trials (CATs). About 2500 clones selected from PATs are tested in unreplicated or partially replicated single-row plots, 10 m long. TCH and CCS from harvesting the plant crop are measured and their BLUPs used to derive NMG. NMG was then used to select tentative clones for further testing in the first ratoon crop. The best clones based on NMG were chosen for the next stage trials. Since 2006, these selections have been also screened for smut resistance in Bundaberg.

In stage 3, known as the Final Assessment Trials (FATs), about 150 clones are tested at multiple sites. At each site, clones are planted in two replicates with 4-row, 10m plots. While TCH, CCS, and fibre content (plus appearance grade in the Northern program) are measured from field trials, resistance to important diseases was generally evaluated in trials specifically established for that purpose. For a small number of promising clones, fibre quality (shear strength, impact reading and % short fibre) is also measured. BLUPs of CCS, TCH and fibre content (plus appearance grade in North) were used to generate NMG. NMGs were averaged over crops and sites to produce a single value for selection.

In summary, the traditional genetic evaluation relied on NMG and independent evaluation of clones for disease and sugar quality. This system is inefficient with respect to the way it uses information. Some of the key issues are as follows.

1. NMG is unable to deliver maximum economic returns for the Australian sugar industry through elite varieties due to the weights on sugarcane traits being biased. It also results in lower genetic gain due to inferior selection methods. Specifically, this can be seen by considering that:
    a. NMG is highly correlated with TCH (Figure1). Thus, application of NMG as the selection index results in preferentially retaining those clones with high tonnes of cane per hectare, but it is biased against those with high CCS. Varieties with these characteristics result in higher production costs and less economic returns for the whole industry than is possible under systems that optimize the economic weights for traits. This biased weight on TCH when applying NMG has prompted sugarcane breeders to sometimes preferentially bias selection toward higher CCS clones at the expense of clones that have high tonnes of cane but lower CCS.

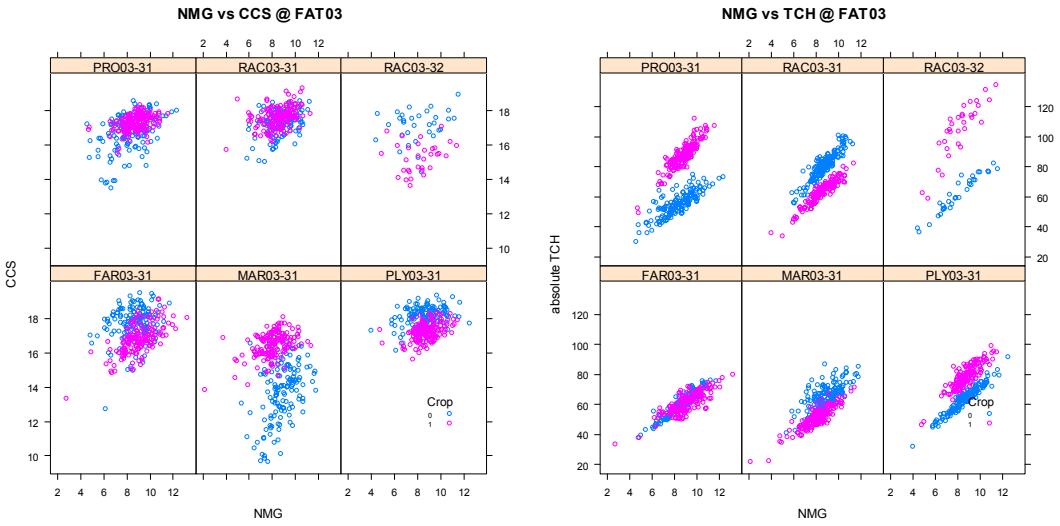


Figure 1 **Relationship between NMG and TCH/CCS at six trials (2003 FAT series) in the Central region. Blue dots represent the plant crop and red the first ratoon**

b. Prior to this project, NMG and other traits such as disease ratings were used independently of one another for selection. It has been shown that this type of independent selection is inferior to index selection that results in simultaneous selection for multiple traits with respect to maximizing genetic gains (Cotterill and Dean 1990; Hazel *et al.* 1994).

c. There was no clear definition of breeding objectives and associated selection indices. This caused the confusion between the two, and misinterpretation and misapplication of the results from the above analyses. For example, TCH in a pure commercial stand is the trait that should be selected for, whereas TCH from small plots is a selection criterion that can be used to estimate the TCH in pure stand. Therefore, TCH from a single-row plot in PATs and CATs is less informative about a clone than TCH in FATs, because TCH from single-row plots is less-well correlated to TCH in commercial stands. When evaluating a clone based on its cane yield, the increased emphasis given to TCH data from FATs rather than data from PATs and CATs needs to reflect the expected genetic correlations of the different measurements.

2. NMG did not use inherent genetic correlations between traits and environments. In spite of the fact that progeny of many clones were also tested in other regions, and that the same set of clones were trialled at different sites, analyses were generally conducted on a single crop basis. An analysis that combines trials predicts the genetic effects of these clones more accurately (Hammnod *et al.* 1992).

3. NMG did not take account of variable data quality across trials and traits. A simple example is that the calculation of NMG assumes that all traits observed in trials correlate equally well to the traits observed in commercial crops. This is doubtful. Jackson and McRae (2001) found that CCS in small plot trials was more closely correlated with CCS in pure stands than was TCH.

4. Breeding values predicted using BLUPs on data collected from PATs did not utilise the whole pedigree information to calculate a NMG score. This will reduce the accuracy of predicting breeding values, and could produce biased predictions due to selection.

To address the above issues, this project aimed to develop and implement a new genetic evaluation and selection system for sugarcane improvement that can integrate economic and genetic information from all regions and selection stages to maximise economic returns for the Australian sugarcane industry through genetic improvement. This system is built on two fundamental requirements:

- breeding objectives, or economic weights of sugarcane traits. For the purpose of this project three production systems were studies involving (a) production of sugar alone, (b) production of sugar and electricity and (c) production of sugar and ethanol; and

- predicted breeding and genetic values for traits associated with each breeding objective through their selection criteria.

2.0 OBJECTIVES

This project had five objectives, each of which was achieved as summarised below.

Objective 1: *Develop and implement a genetic evaluation and selection system for sugarcane improvement that can integrate data (economic and biological) from across all regions and selection stages, to maximise genetic gain for industry economic value.*

A GES for each of the four regional programs has been established and integrated with the BSES-CSIRO variety improvement program database (SPIDNet). In the GES, sugarcane traits are weighed by their economic importance to the whole industry and breeding or genetic values of the traits are predicted by statistical models implemented in ASReml (Gilmour *et al.* 2006). From economic weights and predicted breeding or genetic values, economic breeding values (EBVs) or economic genetic values (EGVs) are automatically generated in SPIDNet. So far, EBVs have been used in the 2007 crossing program, and also for selecting parental clones for 2008. rEGVs (Relative economic genetic values that are re-scaled from EGV, relative to a set of standard varieties) have been extensively used for selection of elite clones during the 2007 plant breeding and industry selection meetings.

Objective 2: *Establish the requirements to estimate economic merit across all sugarcane breeding objectives, including the appropriate economic and genetic weighting of traits.*

Two dual-product production systems, namely sugar and electricity, and sugar and ethanol, were studied. The economic framework for these two systems was established in a spreadsheet-based model from which economic weights for relevant selection criteria could be estimated. However, exact economic weights for electricity and ethanol related sugarcane traits were not derived because of a lack of information at the time this study was carried out.

Implementation of molecular markers into the GES has also commenced via a simulation study. The study emphasised the importance of two key factors on which cost-effective implementation of marker assisted selection will depend: 1) the true proportion of genetic variation explained by markers for economically important traits, and 2) the cost per genotype of screening clones using markers.

Objective 3: *Investigate software options for achieving these two objectives.*

ASReml was chosen as the major statistical package for predicting breeding and genetic values in the GES. This was achieved after conducting a thorough investigation of options throughout Australia and overseas. ASReml has all the necessary functions required to predict genetic effects. The benefits of ASReml will certainly become greater if it becomes an open source program so that more geneticists and statisticians from around the world can contribute to its development.

Objective 4: *Establish procedures to integrate these innovations with the BSES-CSIRO variety improvement database to enable efficient, routine application of these new methods.*

Economic breeding values (EBVs) and economic genetic values (EGVs) have become an integral part of the BSES-CSIRO variety improvement program through SPIDNet. EBVs, EGVs and rEGV have become available to, and been used by, breeders, technicians and extension officers via SPIDNet in the following areas:

1) determining the parental clones used for crossing in 2007;
2) determining the crossing combinations in 2007;

3) selecting clones in all three stage of the selection program during the 2007 plant breeding and industry selection meetings ; and
4) identifying elite clones suitable for commercial release to the Australian sugarcane industry.

Objective 5: *Build capacity in sugar industry breeding programs in the development and application of best-practice quantitative genetic theory.*

This project introduced the formal concept of GES and their application to the BSES-CSIRO variety improvement program. GES clearly defined the breeding objectives for each region and then established a selection system for discriminating among clones with the objective of maximising the genetic gains.

Researchers involved in this project, especially the principal investigators, have obtained substantial knowledge through collaborations with internationally recognized plant breeders and biometricians. The knowledge should benefit the whole of the Australian sugarcane industry into the future.

3.0 METHODOLOGY

A GES is established on the theory of selection index (Hazel 1943; Smith 1936) that equips breeders to improve multiple traits simultaneously and to select among candidate genotypes in an optimal way assuming a particular objective (eg. economic value to an industry producing one or more products). Under this theory, an individual can be expressed as an aggregate genotype that combines all traits of interest. The breeding objective is an overall goal of a genetic improvement program, which is normally expressed as an aggregate genotype and is used to determine how superior an individual is in relation to the average of a reference population. In this report, we use the terms "breeding objective" and "aggregate genotype" interchangeably. As the goal of a breeding program is generally for maximum economic returns, the breeding objective can be defined through each of its traits each weighted according to its economic value relative to all other traits. After the breeding objective is defined, a selection index can be constructed to maximise the correlation of phenotypic traits of individual genotypes with those of the aggregate genotype.

To apply the theory described above to sugarcane genetic evaluation, we assume that our breeding objective is to maximise the economic benefits to the whole of the Australian sugarcane industry arising through the genetic improvement of varieties. In this report, we will focus on just one breeding objective, namely sugar production. Under this production system, the breeding objective for selecting parental clones in one region can be expressed as follows:

$$H_i = v_{ccs} BV^i_{ccs} + v_{tch} BV^i_{tch} + v_{fbr} BV^i_{fbr} + v_{dis} BV^i_{dis} + \ldots \qquad [2]$$

where:

H_i is the economic worth of the i^{th} parental clone (denoted as EBV), or the additional economic benefit that the i^{th} clone could bring to the whole industry from producing one tonne of sugar, relative to all other clones in the breeding population;

v_{ccs} is the economic weight of CCS expressed as benefit to the industry for one tonne of sugar with one unit of improvement of CCS;

BV^i_{ccs} is the breeding value of CCS for the i^{th} individual; and

similar definitions for TCH (tch), fibre content (*fbr*), diseases (*dis*) or other traits in Eq [2].

It is important to note that economic benefits can only be delivered through commercial production, so that traits in the breeding objective should be considered in the context of commercial production; that is, pure stands.

Similarly, the breeding objective for evaluating clones can be expressed as follows:

$$H_i = v_{ccs} GV^i_{ccs} + v_{tch} GV^i_{tch} + v_{fbr} GV^i_{fbr} + v_{dis} GV^i_{dis} + \ldots \qquad [3]$$

where each variable is similar to Eq [2] except that genetic value (*GV*) is used. In this equation, H_i will be denoted as EGV.

For Eqs [2] and [3], a GES will require two fundamental components: (a) economic weights and (b) BV or GV. Economic weights can be derived from economic models as briefly described below. EVs or GVs of traits in pure stand are generally not known and will have to be predicted by measurements in small plot experiments, those measurements being the selection criteria.

Factors such as different sizes of plots at different selection stages, different accuracy of trials (for example various estimates of heritability) will complicate the prediction of EVs or GVs and their utilisation in the GES. These differences are common in sugarcane experiments where trials are normally planted in different years and sites with various configurations.

To take some of these factors into account while predicting breeding or genetic values, Restricted Maximum Likelihood (REML) and Best Linear Unbiased Prediction (BLUP) have become the methods of choice and are widely used in animal and plant genetic evaluation (Bernardo 2002; Henderson 1984; Mrode and Thompson 2005). One of its greatest advantages over traditional selection index is that it can combine all sources of information and automatically weight the information accordingly. REML/BLUP are used in our GES to predict BV or GV from trial data.

In general, predicted BVs (PBVs) or predicted GVs (PGV) by BLUP from selection trial data refer only to selection criteria. For example, PBVs from PATs refer to a 10m plot instead of the BVs for pure stand as required in Eq [2]. A further step is required to combine PBVs of selection criteria and link them to PBVs in the breeding objective. We can achieve this through a selection index (**I**) as given below (Schneeberger *et al.* 1992):

$$\mathbf{I} = \mathbf{v}'_o \mathbf{G}_{os} \mathbf{G}^{-1}_{ss} \hat{\mathbf{u}}_s \qquad [4]$$

where:

$\mathbf{v}_o$ is a vector of economic weights as defined in Eq [2];
$\mathbf{G}_{os}$ is genetic (co)variance components between breeding objective traits and selection criteria traits;
$\mathbf{G}_{ss}$ is genetic (co)variance components among traits in selection criteria; and
$\hat{\mathbf{u}}_s$ are the BLUPs of selection criteria traits.

When all traits in the selection index are the same as those in the breeding objective, the selection index in Eq [4] becomes identical to the breeding objective in Eq [2].

Sometimes it is not feasible to combine all available information into an analysis to let BLUP automatically take into account the unbalanced nature of the data set. This may be caused by the complexity of models being fitted or the poor connection between trials, which often cause the model to fail to converge. In this case, a less complicated model will have to be fitted.

However, this approach has the constraint that it produces several sets of PBV BLUPs for the same set of clones.

Despite having several possible sets of PBVs for the reasons outlined above we can combine them and predict a unique PBV for a given trait by the following equation (VanRaden 2001):

$$\widetilde{u}_{1,2} = \mathbf{c}'_{o's'} \mathbf{V}_{s's'}^{-1} \left[\hat{u}_1 \quad \hat{u}_2\right] \qquad [5]$$

where:

$\widetilde{u}_{1,2}$ is the estimated breeding value based on two sources (1 and 2);

$\mathbf{c}'_{o's'}$ is the covariance between sources and $u_{1,2}$;

$\mathbf{V}_{s's'}$ is covariance between two sources; and

$\left[\hat{u}_1 \quad \hat{u}_2\right]$ is the estimates of the two sources.

The extension to more sources is straightforward. In principle, this is basically the same as Eq [4] for predicting the effect of a trait from its selection criteria.

In summary, the new GES uses breeding objectives as the ultimate goal and selection index as the tool to discriminate between clones according to their performance. Essential information to establish the sugarcane breeding GES include:

1. Economic weights of important crop traits relevant to the breeding objectives (**v**);
2. PBV (estimated via BLUP) of the selection criteria for linking with breeding objectives (**u**);
3. Genetic (co)variances between crop traits in the breeding objectives and selection criteria ($\mathbf{G}_{os}$); and
4. Genetic (co)variance among selection criteria ($\mathbf{G}_{ss}$).

Points 2 to 4 are used to indirectly derive breeding or genetic values for traits included in the breeding objective.

3.1 Economic Weights

3.1.1 Traits excluding diseases

An economic model was built for each region to reflect its different income-cost structure. In the model, every income and cost component for the entire value chain from planting to the sugar market was modelled in an Excel spreadsheet. These costs and incomes were then linked with sugarcane traits. By changing one unit of a trait while holding other traits unchanged, the change in the total benefit for producing one tonne of sugar becomes the economic weight of the trait.

Costs and incomes for each region were provided and confirmed by an industry consultative group that included representatives from several milling companies, CANEGROWERS Limited, the Sugar Research Institute, Queensland Sugar Limited and BSES research and extension groups. Some of the milling costs were extracted from SRDC project CAT041 (Jackson *et al.* 1999). Details are presented in Wei *et al.* (2006), which is attached as Appendix 2.

3.1.2 Diseases

The economic importance of a disease was mainly determined by its risk factor or potential damage. For almost all the diseases considered in this study, damage might not have been observed in commercial production for a long period of time. For example, no damage from

Fiji leaf gall has been reported for more than thirteen years in the Central region. However, a high percentage yield loss was assumed in this study to reflect the potential damage.

Yield loss was assumed to be the only damage caused by a disease. This has been partly validated by the research (see Appendix 1) where, for three endemic diseases, CCS appeared to remain unaffected over a range of levels of disease incidence whereas TCH generally decreased with an increase in disease rating. As a result, we can simplify the economic importance of a disease to only quantify yield loss. When the yield loss of a disease is defined, its economic importance can be indirectly derived from TCH.

To quantify the yield loss, we divided diseases into three categories. This was done to avoid double-counting the impact of diseases. In Category 1, diseases are rarely present in yield assessment trials so that clones are rarely challenged by the diseases. Hence, yield and other traits in trials are unaffected. This means that the relationship between yield loss and resistance levels can be based on field experience during past or present epidemics. This category includes Fiji leaf gall, leaf scald, mosaic, chlorotic streak, red rot and smut. Smut may well change to another disease category in coming years. In Category 2, diseases are present in the yield assessment trials in such way that they could vary from plot to plot, site to site or season to season. This category includes pachymetra and yellow spot. In Category 3, diseases have a similar pattern of incidence to category 2 except that they are distributed relatively evenly with some seasonal variation and inter-plot interference effects. This category includes orange rust and brown rust. TCH losses in categories 2 and 3 were determined by FAT trials and pathology experience. Maximum yield losses for the most susceptible varieties were assumed to be lower than the observed maximum loss because of the likely impact of diseases already on yield.

Based on the above categories, the detailed research results described in Appendix 1, and experience from sugarcane pathologists and breeders, potential yield loss estimates for major diseases in each region were developed (Table 1).

Table 1 Yield loss (%) from a disease during a complete crop cycle in each region

Disease Rating	Northern	Burdekin	Central	Southern
Smut				
1-2	0	0	0	0
3	0	1	0	0
4	0	5	1	1
5	1	10	5	5
6	5	18	10	10
7	15	30	20	20
8	30	50	40	40
9	65	88	78	78
Fiji leaf gall				
1-5	0	0	0	0
6	0	0	5	5
7	0	0	10	20
8	0	0	25	40
9	0	0	50	65
Leaf scald				
1-5	0	0	0	0
6	2	2	2	0
7	5	5	5	2
8	20	20	20	10
9	40	40	40	25
Mosaic				
1-6	0	0	0	0
7	0	0	0	2
8	0	0	0	5
9	0	0	0	10
Pachymetra				
1-2	0	0	0	0
3	0.5	0	0	0
4	2	0	0.5	0.5
5	3.5	0	2	2
6	5	0	3.5	3.5
7	10	0	5	5
8	20	0	10	8
9	30	0	20	15
Yellow spot				
1-5	0	0	0	0
6	1	0	0	0
7	3	0	0	0
8	6	0	0	0
9	10	0	0	0
Orange rust				
1-6	0	0	0	0
7	5	5	5	5
8	20	20	20	20
9	40	40	40	40

A general form of yield loss (*yl%*) based on Table 1 was then constructed using equation [6].

$$yl(\%) = \text{constant} + a \cdot e^{b \cdot rating} \qquad [6]$$

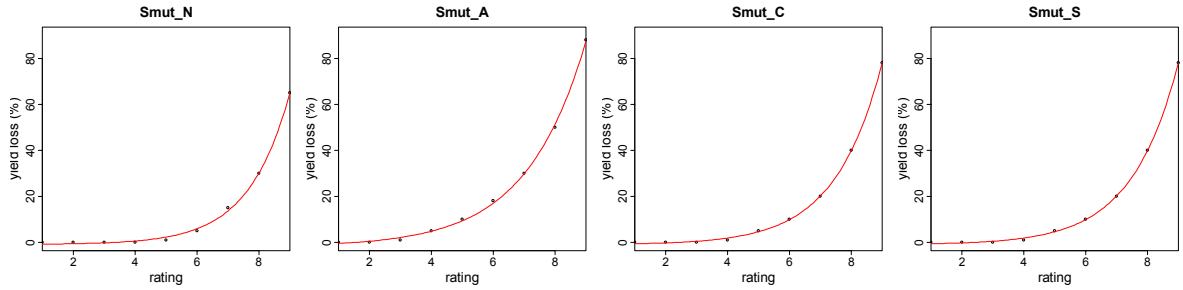
The parameters for this equation are listed in Table 2. They are also illustrated graphically (Figure 2).

Table 2 Parameters for functions (Eq.[6]) that describe the relationship between yield loss and disease rating

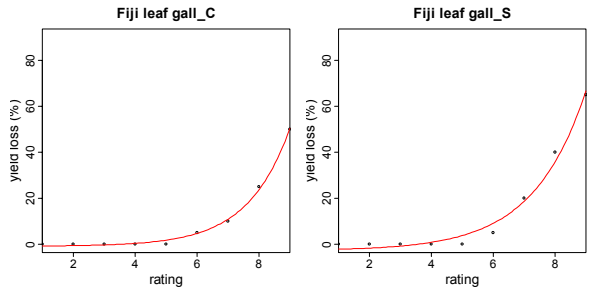
Disease	Region*	Constant	a	b
Smut	N	-0.939	0.074	0.755
	A	-1.891	0.845	0.518
	C/S	-1.033	0.209	0.66
Fiji leaf gall	C	-2.840	0.068	0.736
	S	-2.840	0.347	0.589
Leaf scald	N/A/C	-0.749	0.025	0.822
	S	-0.346	0.003	1.014
Mosaic	S	-0.244	0.009	0.778
Pachymetra	N	-0.959	0.359	0.497
	C	-0.07	0.058	0.648
	S	-0.331	0.143	0.517
Yellow spot	N	-0.407	0.051	0.592
Orange rust	N/A/C/S	-0.909	0.022	0.841

* N – Northern, A – Burdekin, C – Central, S – Southern.

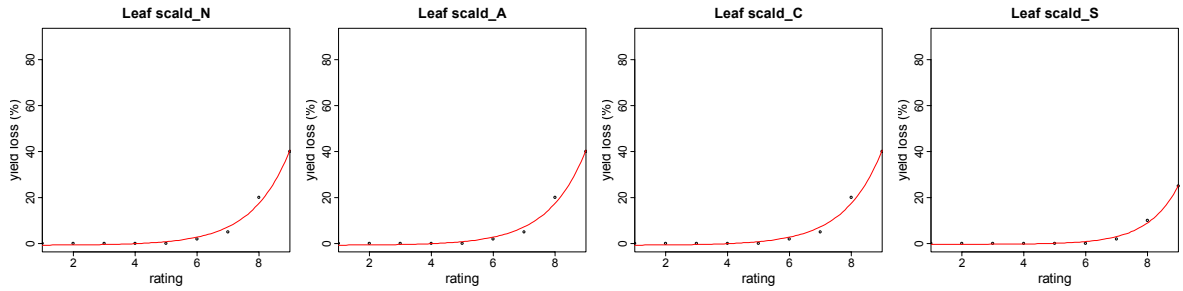
Smut



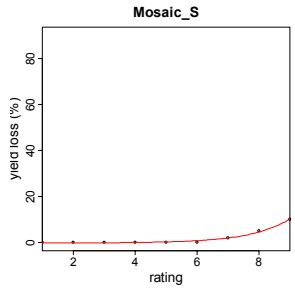
Fiji leaf gall



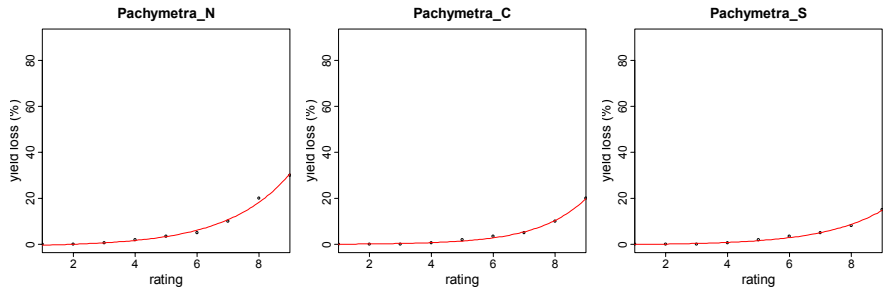
Leaf scald



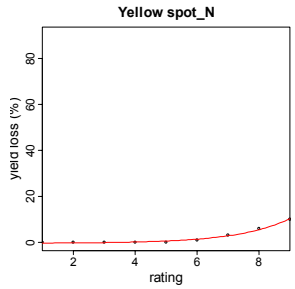
Mosaic



Pachymetra



Yellow spot



Orange rust

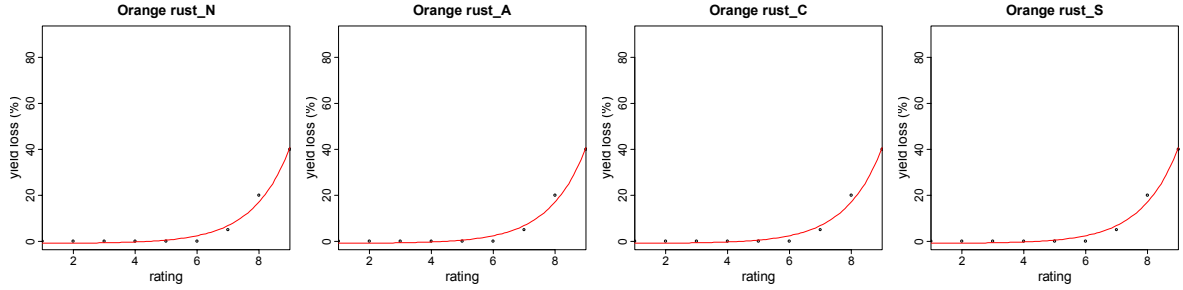


Figure 2 **Relationship between yield loss (%) and ratings for major sugarcane diseases in each sugarcane production region of Australia**

For any clone with a known disease rating in a region, the economic importance of that disease can be estimated by the amount of yield loss and the importance of TCH as a selection criterion in that region. For example, take the impact of smut on Q209$^{\phi}$ in the Central region to illustrate how the importance of smut is determined. Suppose the mean smut rating of all varieties in Central district is 6.5, therefore the average yield loss (%) due to smut in the Central district is $-1.033 + 0.209 * e^{6.5*0.660} = 14.2\%$. Similarly, the yield loss for Q209$^{\phi}$ with a smut rating of 8 is 40.0%. Therefore, relative to the average, Q209$^{\phi}$ would lose 25.8% more cane yield due to smut. Suppose the average yield in the Central region is 75.4 t/ha and the economic weight for cane yield is \$0.68, the yield loss for Q209$^{\phi}$ as a variety due to smut in the Central region would be 25.8% * 75.4 = 19.5 TCH, or \$13.23 for each tonne of sugar produced.

3.1.3 Appearance grade

In the Northern region, appearance grade is recorded in all trials to reflect the status of lodging, suckering and flowering of a clone. Berding and Hurney (2000) used appearance grade to modify the traditional NMG calculation. We estimated the economic weight of appearance grade by empirically fitting EGV with this modified NMG. This resulted in one unit of improvement of appearance grade being equivalent to an economic benefit of \$5 for each tonne of sugar produced.

3.2 Predicting genetic effects of selection criteria

To predict breeding or genetic value of a trait from field trials, we first fitted data at a trial level to develop an optimal model that accounted for correlated residual structure (eg spatial variation) in all trials for all traits and competition effects in CATs for TCH. Then we combined all the trials that had sufficient connectedness among them and accounted for their genetic correlations (eg correlation between crops and sites).

3.2.1 Breeding values

For CCS and TCH: Currently in the BSES-CSIRO variety improvement program there are two types of clones that are used as parents: 1) those that have been used previously as parents with progeny that have been tested; and 2) those that have not previously been used as parents. For the first group of clones, PBVs were derived from PATs. In each region, BLUPs for PBVs were predicted on a combined analysis for more than 10 years of PATs, using all available pedigree information.

For the second group of clones PBVs were derived from their own performance from CATs and/or FATs. We simply assumed that half of PGV is due to additive genetic effects (Rattey *et al.* 2004). It should be noted that models have recently been developed to directly partition PGV into additive and non-additive effects (Oakey *et al.* 2007), but such approaches have yet to be applied in the context of the GES developed through this project. Direct predictions of breeding values will be obtained and validated in the future.

For diseases: Breeding value for a disease (*PBV*) was estimated by the following equation:

$$PBV_i = (r_i - \bar{r})h^2 + \bar{r} \qquad [7]$$

where:

r_i is the disease rating for the i^{th} clone;

$\bar{r}$ is the average disease rating for the population of clones to which clone r_i is being compared; and

h^2 is the estimated narrow-sense heritability of the disease rating (Table 3).

Resulting PBVs for diseases were assumed to be the true BV and used directly in defining breeding objectives.

Table 3 Estimates of narrow-sense heritability (h^2) of major sugarcane diseases

Disease	h^2	Source
Smut	0.43	Average of 0.49 (Wu *et al.* 1988), and 0.41 and 0.38 (Chao *et al.* 1990)
Fiji leaf gall	0.66	Hogarth *et al.* (1993)
Leaf scald	0.55	Average of four estimates of broad-sense heritability from two trials and two crops (Offmann *et al.* 2001) adjusted with an assumption that 75% total genetic variance is additive
Mosaic	0.6	Assumed
Pachymetra	0.61	Middle value of the range of 0.57 – 0.64 (B.J. Croft and N. Berding unpublished)
Yellow spot	0.6	Assumed
Orange rust	0.6	Assumed

3.2.2 Genetic values

Genetic values for clones tested in CATs are predicted by the method developed by Stringer (2006). An outstanding feature of this method is that PGVs predicted by this method have taken into account the effects of competition and spatial variation simultaneously. Genetic values for clones tested in FATs were predicted by methods described in Appendix 3 that have taken into account of correlated residual (eg spatial variation) and genetic structures (eg correlation between crops and sites). PGV for a disease was assumed to be the rating measured from disease experiments (for smut, Fiji leaf gall, leaf scald, mosaic and pachymetra) or observed from FATs (yellow spot and orange rust).

3.3 Construction of selection index for selecting parents and clones

3.3.1 Index for selecting parents

In constructing a selection index for selecting parental clones in a given region, Eq [5] was used to derive a combined set of PBVs for a trait, and Eq [4] used to construct the selection index. In Eq [5], PBVs from its own region as well as other regions were combined, with the covariance matrix between criteria and objectives ($\mathbf{c}'_{o's'}$) drawn from the results of SDRC project CTA041 (Table 4) and covariance between criteria ($\mathbf{V}_{s's'}$) being estimated empirically from PBVs of four regions. It must be noted that the resulting PBV of a trait should be regarded in the context of single-row plot and had to be converted to PBV in pure stand to maximise the correlation with the breeding objective in Eq [2], if PBV from other stages are also used.

Table 4 **The average of genetic correlations (for CCS and TCH) between environments in each region versus (i) all independent environments (i.e. different sites) in the same region (along the diagonal) and (ii) all environments in each other region. Standard errors are not shown, but are between 0.1 and 0.2 in most cases (extracted from Chapman *et al.* 2005)**

	Northern	Burdekin	Central	Southern
CCS				
Northern (coast)	0.74	0.48	0.73	0.76
Burdekin	0.48	0.50	0.52	0.53
Central	0.73	0.52	0.82	0.87
Southern	0.76	0.53	0.87	0.88
TCH				
Northern (coast)	0.74	0.57	0.45	0.46
Burdekin	0.57	0.67	0.43	0.55
Central	0.45	0.43	0.63	0.47
Southern	0.46	0.55	0.47	0.53

To construct a selection index as in Eq [4], $\mathbf{I} = \mathbf{v}'_o \mathbf{G}_{os} \mathbf{G}_{ss}^{-1} \hat{\mathbf{u}}_s$, v was the vector of economic weights as described in 3.1; G_{os} was taken from a study by (Jackson and McRae 2001) and listed in Table 5. This study quantified the correlations between single-row and pure stand in CCS and TCH. G_{ss} was assumed to be 1. This principle applies when the index includes PBVs directly derived from FATs by the method of Oakey *et al.* (2007).

Table 5 **Mean genetic correlations of CCS and TCH from two experiments between CCS or TCH in single row plot or the middle two rows of four-row plots, and corresponding traits in pure stands (Jackson and McRae 2001)**

Trait	No of rows	Plant	1st Ratoon	Mean	Note
CCS	1	0.77	0.79	0.78	For correlating PAT and CAT to
TCH	1	0.53	0.35	0.44	pure stand
CCS	2	0.93	0.89	0.91	For correlating FAT to pure stand
TCH	2	0.65	0.45	0.55	

Because we assumed that the PBVs for diseases were the true BVs, there is no adjustment required for diseases. For each region, a different set of diseases was included in constructing the selection index appropriate for that region (see Table 6).

Table 6 **Inclusion of disease in determining economic genetic values and economic breeding values for selecting parents**

Disease	Northern	Burdekin	Central	Southern
Smut	yes	yes	yes	yes
Pachymetra	yes	no	yes	yes
Yellow spot	yes	no	no	no
Fiji leaf gall	no	no	yes	yes
Leaf scald	yes	yes	yes	yes
Mosaic	no	no	no	no
Orange rust	yes	yes	yes	yes

3.3.2 Index for selecting clones

All selection indices were built based on Eq. [3] with an assumption that each crop at every trial of each series contributed equally to the PGV for a clone, so GV in Eq [3] was predicted by the simple arithmetic mean of PGV over crops, sites and trial series. EGVs from the selection index were converted into relative economic genetic values (rEGV). We have proposed the use of rEGV as the equivalent of NMG using selection index theory and BLUPs as the basis for estimation. rEGV is calculated in a way similar to NMG in that the average rEGV for a given trial is centred on the mean of the standards, adjusted to a result of 10 (see equation [1]). Test clones with an rEGV above 10 are therefore considered superior and bring more economic benefits to the industry than the average of the standards. The formula for rEGV can be written as:

$$rEGV = \frac{EGV_t - \overline{EGV}_s}{\sigma_{EGV}} \qquad [8]$$

where:

EGV_t is the EGV of a test clone;

$\overline{EGV}_s$ is the average of EGV of standards; and

σ_{EGV} is standard deviation of the EGVs from a given trial or series.

We assumed the disease ratings from disease screening trials or observations to be the true genetic values. These values were used to obtain yield loss and then economic loss and added into the selection index. The diseases included in the selection index for each region is shown in Table 7.

Table 7 Inclusion of disease in determining economic genetic values and economic breeding values for selecting clones

Disease	Northern	Burdekin	Central	Southern
Smut	yes	yes	yes	yes
Pachymetra	yes	no	yes	yes
Yellow spot	yes	no	no	no
Fiji leaf gall	no	no	yes	yes
Leaf scald	yes	yes	yes	yes
Mosaic	no	no	no	yes
Orange rust	yes	yes	yes	yes

3.4 Implementation of GES in sugarcane improvement program

Economic weights, PBVs and PGVs were uploaded into SPIDNet manually for each region. EBVs, EGVs and rEGVs are then automatically generated by SPIDNet and are made available for breeders or other staff who have the access rights to SPIDNet. EBVs are used to assist in selecting parental clones and for determining preferred cross combinations. Report generation for these functions has been automated in SPIDNet. EGVs, and their rescaled value rEGV, are used to select clones as varieties, in particular, to generate variety data files for the annual selection meetings held in each region.

The whole GES and its associated concepts and benefits were communicated to breeders, plant improvement technicians, extension officers and industry representatives through presentations at annual selection meetings held in 2007, at ASSCT conferences, a technician training workshop to be held in December 2007, and various other meetings. BSES Newsletters and

local industry papers were also used as media to make the GES more familiar to the Australian sugarcane industry.

4.0 OUTPUTS

Detailed outputs on the GES have been already published (see Appendix 2 and 3). Further publications in scientific journals are envisaged. Here, we present the major features of the GES.

4.1 Economic merits

Economic weights for CCS, TCH, fibre content and appearance grade are presented in Table 8. In all regions CCS is the most important trait, however its relative importance varies, from 1 : 14.4 (CCS : TCH) in the Central region to 1 : 26.1 (CCS : TCH) in the North region. For sugar production alone, fibre content plays a negative role. That is, increasing fibre content leads to less economic benefits to the whole industry.

The relative importance of CCS is generally about double that of TCH, and triple that of fibre content, despite the relatively lower genetic variation for CCS. This suggests that for the same amount of effort, breeding for CCS should result in more genetic gain in economic returns than TCH and fibre content for sugar production.

Appearance grade plays an important role in evaluating the relative merit of clones in the Northern region. However, we were not able to determine its relative economic importance due to the lack of information, eg. heritability, and hence we cannot conclude how easy it is to improve this trait in relation to others. With this trait, a potential double counting must be avoided because any late measurement of CCS, TCH and appearance grade would have the impact of appearance grade already being embedded in CCS and TCH measurements. Therefore, a reward to high appearance grades, or conversely a penalty to clones with low appearance grades, would double count its effects on the economic value of a potential clone to the sugarcane industry.

Table 8 Absolute and relative economic weights of three sugarcane traits for sugar production in Australia. Absolute economic weight (= Absolute) of a trait is defined as the net benefit in dollars to the whole industry (in $/tonne sugar) resulting from one unit change of the trait while other traits are assumed to remain constant. Relative economic weight is estimated by multiplying absolute economic weight with selection response from one unit of selection intensity*

Trait	Unit	Northern	Burdekin	Central	Southern
Absolute					
TCH	tonnes/ha	0.45	0.73	0.68	0.75
CCS	%	11.76	13.23	9.81	15.88
Fibre content	%	-1.67	-2.30	-2.76	-3.08
Appearance grade	Scale	5.00			
Relative (EBV)					
TCH	tonnes/ha	2.60	4.21	3.92	4.33
CCS	%	6.70	7.54	5.59	9.05
Fibre content	%	-1.48	-2.04	-2.45	-2.73
Relative (EGV)					
TCH	tonnes/ha	4.33	7.02	6.54	7.21
CCS	%	8.05	9.05	6.71	10.86
Fibre content	%	-1.98	-2.73	-3.27	-3.65

* Assumptions: narrow-sense heritability (h^2) = 0.3 for TCH, 0.5 for CCS and 0.45 for fibre content; broad-sense heritability (H^2) = 0.5 for TCH, 0.6 for CCS and 0.6 for fibre content; phenotypic variance (σ_p^2) = 370 for TCH, 1.3 for CCS, 3.9 for fibre content. Therefore, the selection responses from one unit of selection intensity ($h^2\sigma_p$) for TCH, CCS and fibre content are 5.77, 0.57 and 0.89, respectively.

The GES has resulted in an economic value more strongly associated with CCS and less with TCH compared to the relationship between NMG and CCS/TCH. Using the same set of data set from 2003 FATs in the Central region as illustrated in Figure 1, Figure 3 clearly illustrates a more desirable relationship between the selection index with both CCS and TCH.

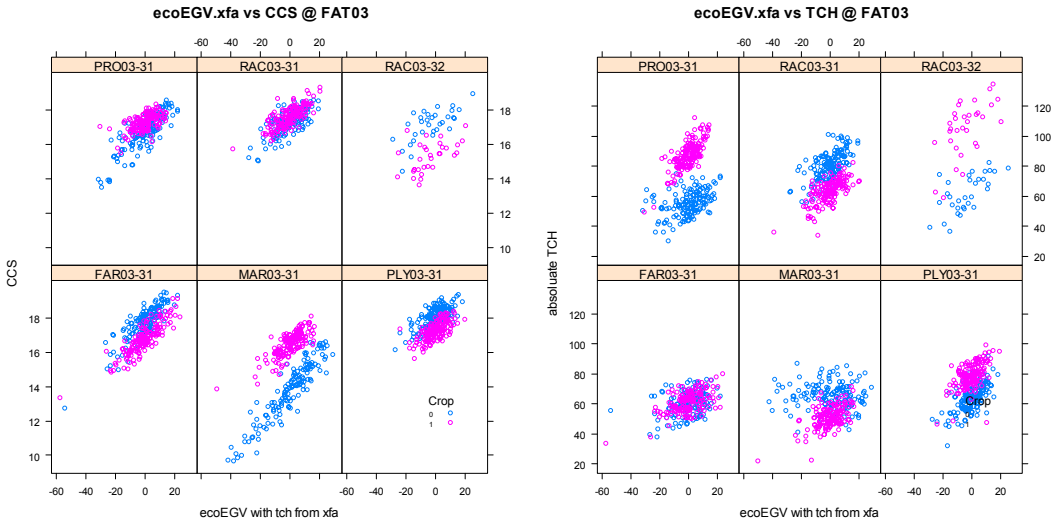


Figure 3 **Relationship between economic genetic value and TCH/CCS for six trials in the 2003 FAT series in the Central region. Blue dots representing for plant crop and red for the first ratoon**

We have considered all other traits such as GGM content, sugar colour and ash. These results are not discussed further in this report. This was due to the lack of information that would enable us to accurately derive their economic importance.

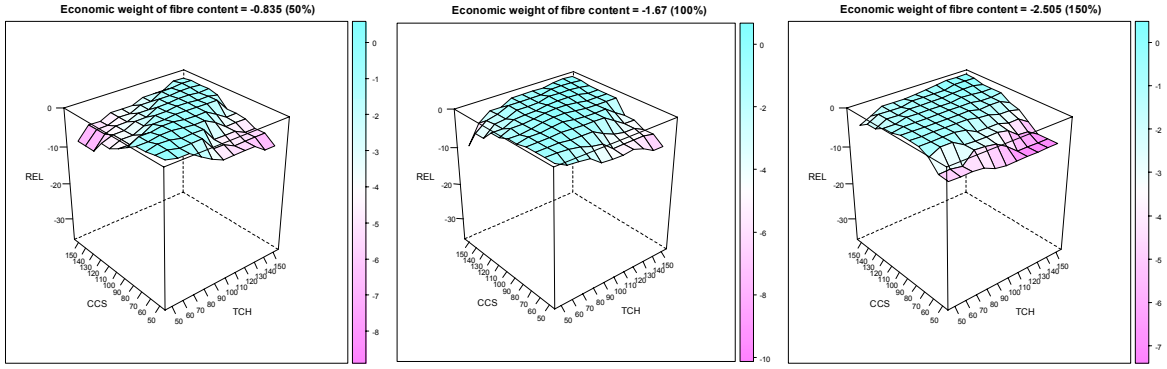
4.1.1 Sensitivity analysis of economic weights on genetic gains

The impact of economic weights for CCS, TCH and fibre content is illustrated in Figure 4. Using economic weights in the GES as base values, we changed the economic weights for CCS and TCH from 50% to 150% with increments of 10%. For fibre content we only tested three scenarios, 50%, 100% or 150% of its base value. This was carried out on the 2003 FATs and genetic gain was determined by averaging the EGVs of the top 25 clones. Relative gain (%) was used as a criterion to determine the impact of changing the economic weights. This was calculated as the ratio of the difference in the gains achieved using selection at different economic weights versus selection using the base values for the weights.

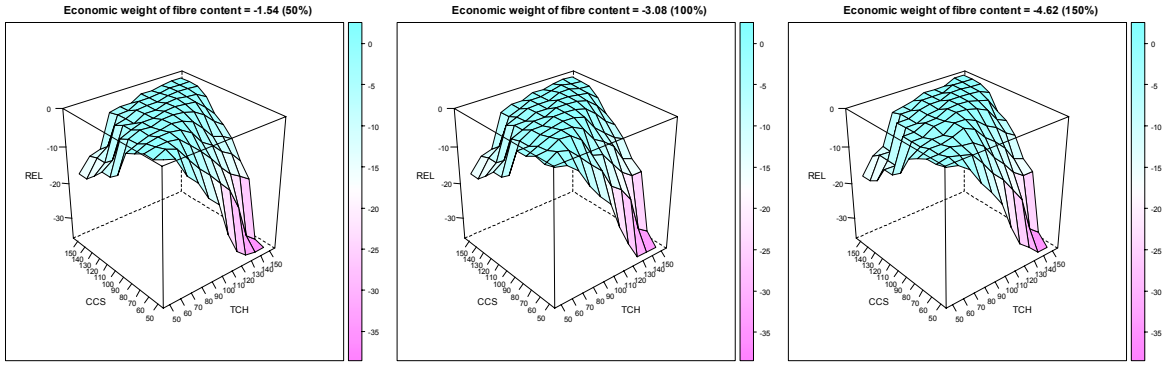
It is apparent that the impact is more profound when the weights for CCS and TCH vary at opposite directions (left and right corners in Fig 4), especially when CCS is unfavourably weighed while TCH is given more weight (right corner). When both CCS and TCH are weighed simultaneously less or more, there are relatively small variations in economic returns. For example, there are only small gains if both CCS and TCH take 50% or 150% of their base values.

The impact of varying the economic weights varies from region to region. The largest variation occurred involved the 50% of base case value for CCS and 150% of the base case value for TCH, resulting in 36% smaller gains in Burdekin, 24% smaller gains in the South, 19% smaller gains in the Central region and 9% smaller gains in the Northern region. Less sensitivity was observed in North partly due to the addition of appearance grade in the index used to derive EGV.

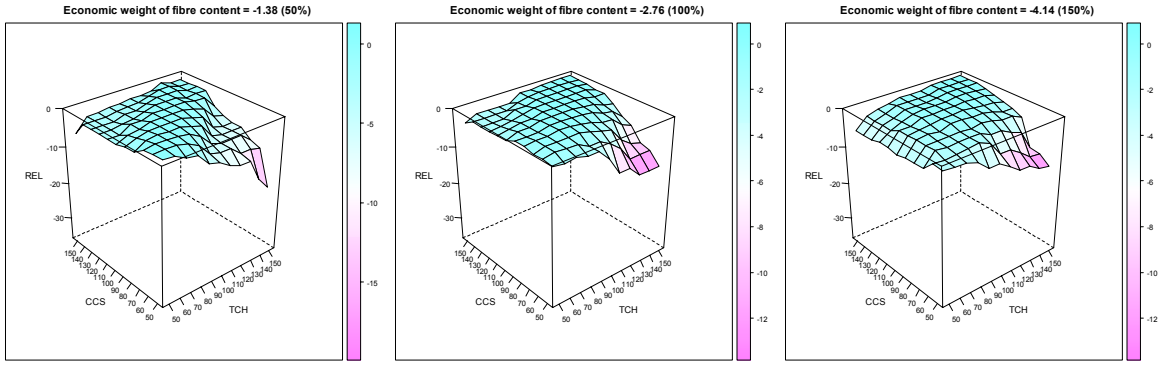
N



A



C



S

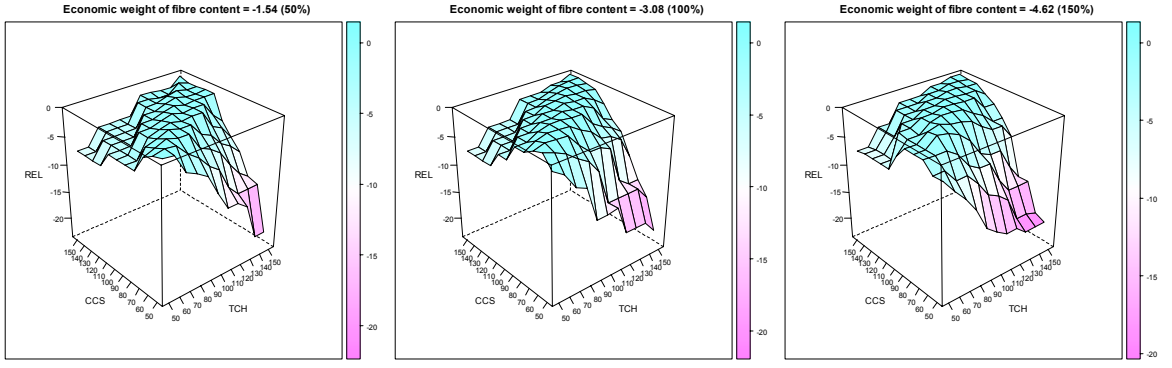


Figure 4 **Relative gains (REL in %) from selection indices with different economic weights for CCS and TCH when economic weights for fibre content was assumed to be equivalent to 50, 100 and 150% of that used in the genetic evaluation system. Analyses were based on data from the 2003 FAT series**

4.1.2 Impact of economic values of diseases on selection

It is important to investigate the impact of incorporating economic loss from diseases on other traits, because any heavier or more lenient penalty on diseases than is optimal will reduce the overall efficiency of selection in the breeding program. A simulation using smut as an example was carried out to examine (1) how many susceptible clones (rating >=7) will be selected; and (2) mean value for other traits, using smut as an example.

In the simulation, a population was generated by genetic variances estimated from FAT trials in four regions. For each trait, a normal distribution was assumed and correlations among traits were treated as zero (this is valid for constructing a selection index based on genetic values predicted from multivariate BLUP as in Equation [3]).

The results from the simulation showed that clones with higher ratings were seldom selected (Table 9) while other sugarcane traits were improved simultaneously (Table 10). For example, out of 1000 simulations, a clone with a rating of 9 was selected on only one occasion (in the Northern region program). This result is generally consistent with what would be predicted intuitively by breeders and suggests that the economic impact of the smut rating has been properly evaluated though the work done in this project.

Table 9 **Frequency of clones with smut rating ≥7, ≥8 or ≥9 being selected at 1,000 simulations. Selection is based on EGV which includes TCH, CCS, and smut ratings, fibre content and appearance grade are also included for Northern. Selection percentage is 10**

Selection threshold	Number of clones selected	Frequency*			
		Northern	Burdekin	Central	Southern
Rating ≥ 7	0	383	801	632	436
	1	391	176	283	384
	2	176	21	69	117
	3	42	2	15	51
	4	7	0	1	11
	5	1	0	0	1
Rating ≥ 8	0	863	999	992	948
	1	133	1	8	50
	2	4	0	0	2
Rating ≥ 9	0	998	1000	1000	1000
	1	2	0	0	0

* frequency indicates the number of times out of 1,000 that a given number of selected clones occurred, eg. for the Northern region, there were 383 times out of 1,000 that no clones with smut rating ≥ 7 were selected.

Table 10 Range of average values for selected clones over 1000 simulations

Trait	Unit	Northern		Burdekin		Central		Southern	
		Min	Max	Min	Max	Min	Max	Min	Max
TCH	Tonnes	6.64	38.23	7.76	40.06	1.73	17.06	0.76	15.4
CCS	%	0.02	1.17	-0.01	0.83	0.22	1.22	0.38	1.33
Fibre content	%	-2.25	1.10						
Appearance grade	Scale (1-9)	-0.01	2.62						

4.2 Predicted breeding and genetic values

Data from all recent trials (in all selection stages from each region) that are required for identifying clones to be advanced for further testing at the selection meetings to be held in March-April 2007 have been re-analysed based on the new GES methods. For PATs, BLUPs of a trait at one region were predicted using data from over 10 years of PATs (generally from the 1992 to 2005 series), along with all available pedigree information. Two sets of EBVs were then derived for a given region, one based on BLUPs of CCS and TCH predicted from its own region and another based on combined BLUPs that integrated BLUPs from all regions. Therefore, EBVs have taken accounted for the spatial variation at every trial level and genetic correlations among regions. These predictions have also utilised all the available pedigree information.

All CATs required for selection in 2006 were analysed by models that could take into account spatial variation and competition.

For FATs, series from 2000 to 2005 have been re-analysed. The BLUPs generated by these analyses have taken into account the spatial variation at each crop level and also genetic correlations between crops and sites. The impact of the analysis on predicted genetic values has been compared with the results from conventional analyses that treated single crop at a site as an independent trial (see Appendix 3 for detailed results).

Because of the optimal utilisation of all available information by the new methods, it is expected that BLUPs predicted in the new GES should have higher accuracy than the traditional methods used in the variety improvement program. This should make EBVs or EGVs more accurate in evaluating sugarcane clones and thus result in higher genetic gains. This can be seen by examining the average prediction error variance (Table 11). Because accuracy is inversely related with this variance, low variance indicates a higher accuracy. For the 2003 series of FATs, both CCS and TCH had higher accuracy from the new GES, although caution is required because this comparison is based on estimated variance components.

Table 11 Average prediction error variance for CCS and TCH based on 2003 FAT series by different models. NMG assumed independent genetic correlations among crops and trials; while new GES could take account of the correlated genetic structure between crops and trials (sites)

Trait	Method	Northern	Burdekin	Central	Southern
CCS	Old	11.73	15.93	12.54	11.73
	New	9.03	10.91	8.62	10.70
TCH	Old	0.73	0.97	1.36	0.78
	New	0.60	0.75	1.01	0.51

4.3 Impact of GES on selection

To investigate the impact of the new GES on selection, we must make the new GES and NMG comparable. NMG generally includes a part of all four traits: CCS, TCH, fibre content and appearance grade. Therefore, we used the same set of traits to derive EBV or EGV in GES when the new and old systems are being compared.

It must be noted that such a comparison could seriously underestimate the favourable impact of the new GES because of the exclusion of other traits, such as disease ratings. In the new GES, excluded traits are treated as an integral part of an index and therefore weighed according to their economic importance. In the old system, NMG and other traits such as diseases were generally selected independently. Cotterill and Dean (1990) found the expected genetic gain was only 10% from independent selection, compared with 26 – 28% from index selection.

It is important to understand that the gains expressed in this report represent the gains of a population (e.g. a group of selected parents or clones over their reference population). Therefore, they do not represent the commercial gains likely to be achieved by growers. The gains realised by growers would relate to the top 1 or 2 clones, which will be reflected in much higher gains, as will shown in the following sections of this report. More importantly, the gains are expected values based on available information. Therefore, these values could be improved if more information becomes available. For example, fibre content data generated from the use of the recently implemented NIR predictive method for cane quality measurements in the variety improvement program using SpectraCane, would result in higher gains for selecting parental clones based on the PATs.

4.3.1 Selection for parental clones

Based on the parents that are available for crossing in 2008, and also have information on EBV and NMG, 96 clones would be selected when a selection intensity of 20% was assumed. Consequently, the progeny from parents selected from the new GES would on average result in an additional gain of $1.40 in North, $0.72 in Burdekin, $0.66 in Central and $1.56 in South for each tonne of sugar compared to the gains achieved practicing selection using NMG (Table 12). These gains represent 32%, 11%, 16% and 16% more gain from the new GES over NMG in North, Burdekin, Central and South, respectively.

There are large differences in the clones selected by both methods. Out of 96 selected clones, the number of common parents varied from only 51 in North to 70 in Burdekin. Parents selected by EBV yielded more gains in CCS than those by NMG, but less in TCH. The relatively low number of common clones might indicate the importance of appearance grade in the Northern region because NMG calculated here excluded appearance grade.

Table 12 **Expected genetic gains ($/tonne of sugar) from selecting 96 parental clones by economic breeding value (EBV) and net merit grade (NMG) in four regions for 2008 crossing. The impact on CCS and TCH as well as the number of common clones selected are also listed. BLUPs of TCH and CCS used to generate EBV were combined from all regions. Appearance grade and fibre content were assumed to be constant**

Gains	Selection method	Northern	Burdekin	Central	Southern
$/tonne of sugar	EBV	5.75	6.99	4.78	11.34
	NMG	4.35	6.27	4.12	9.78
CCS	EBV	0.43	0.14	0.26	0.42
	NMG	0.22	0.11	0.14	0.33
TCH	EBV	1.60	7.09	3.22	6.16
	NMG	3.80	6.59	4.10	6.10
Number of common clones selected (out of 96)		51	70	62	62

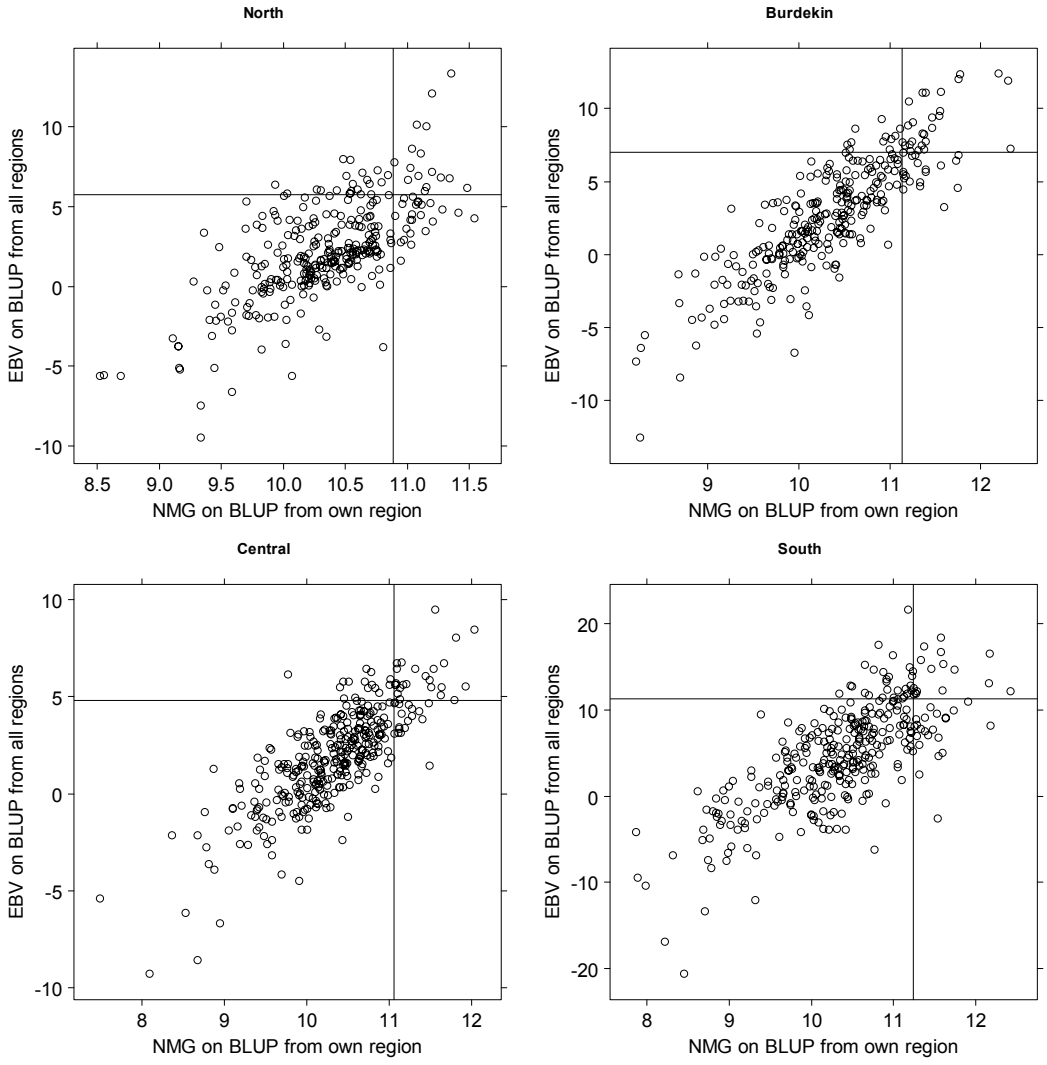


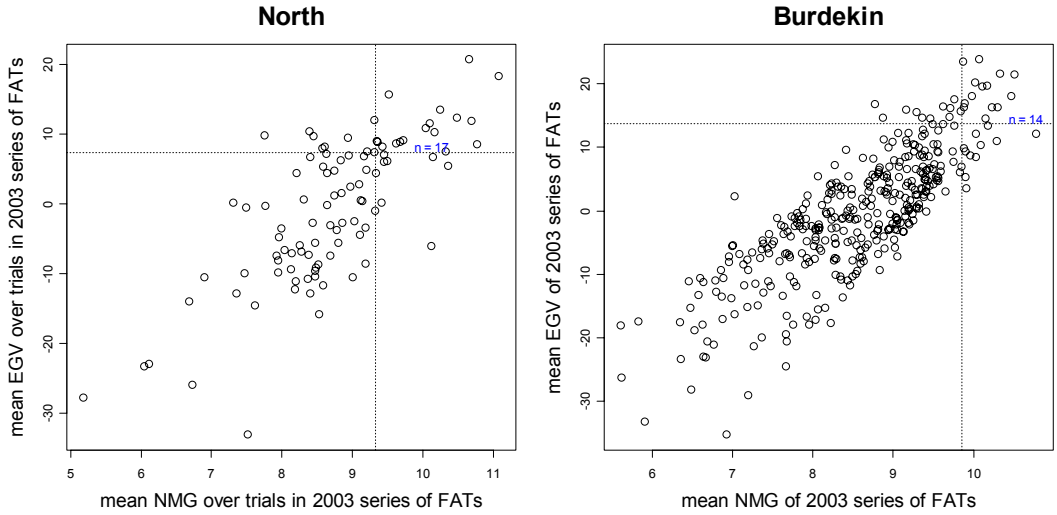
Figure 5 **Economic breeding values and net merit grade for parental clones to be used in 2008. Both values were derived from BLUPs that firstly predicted on PATs of one region and then combined with BLUPs from other regions by Eq [5]. Solid lines in each plot indicate the threshold where 96 clones were selected by EBV or NMG**

4.3.2 Selection for varieties

For the 2003 series, more profit would be expected from selection by EGV than by NMG. As an example shown in Table 13, if the top 25 clones were selected for further testing as suggested by Jackson and Wei (2006), gains from EGV were $1.97 (Northern) to $4.73 (Central) more than from NMG. Again the expected gains from EGV were higher for CCS and fibre content, but lower for TCH. The differences in selected clones were large from the two systems as illustrated in Figure 7. For instance, less than half of the selected clones were in common in the Central region.

Table 13 **Expected genetic gains ($/tonne of sugar) from selection by economic genetic value (EGV) and net merit grade (NMG) in four regions based on 2003 series of FATs. The impact or expected genetic gain of individual traits for CCS, TCH, fibre content and appearance grade as well as number of common clones selected are also listed**

Gains	Selection index	Northern	Burdekin	Central	Southern
$/tonne of sugar	Mean EGV	10.21	17.53	15.38	12.84
	Mean NMG	8.24	13.74	10.65	10.59
CCS	Mean EGV	0.68	0.83	1.02	0.68
	Mean NMG	0.45	0.68	0.41	0.36
TCH	Mean EGV	1.74	6.75	3.11	1.34
	Mean NMG	7.13	6.9	8.19	6.11
Fibre content	Mean EGV	-0.91	-0.12	-1.18	-0.32
	Mean NMG	-0.11	-0.13	-0.37	-0.07
Appearance grade	Mean EGV	0.11			
	Mean NMG	0.11			
Number of common clones selected (out of 25 selected clones)		17	14	10	14



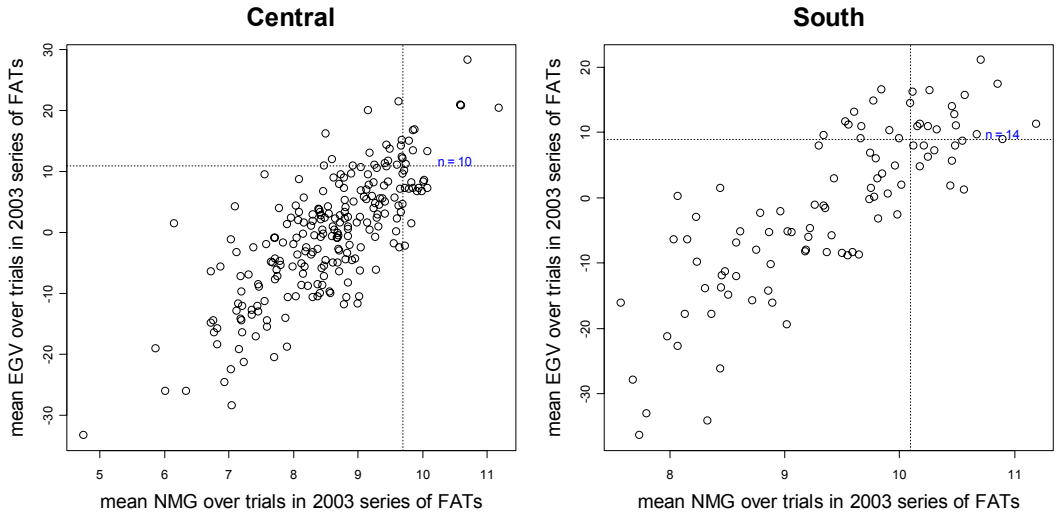


Figure 6 **Comparison of mean EGV and mean NMG for clones tested in 2003 series of FATs in four regions. Dotted lines represented the threshold where top 25 clones were selected by mean NMG (vertical) and mean EGV (horizontal)**

5.0 INTELLECTUAL PROPERTY

The copyright residing in all written materials, computer software and scripts is held by BSES, except in the case of an ASSCT publication where those rights have been assigned to the publisher. The statistical models development in the project and details of their application have been published or are being prepared for publication. Detailed economic information on costs of cane production, milling and marketing that was used to derive economic weights for sugarcane traits should remain confidential because of commercial sensitivity of this information.

6.0 ENVIRONMENTAL AND SOCIAL IMPACTS

The key environmental and social impacts concern sustaining the BSES-CSIRO variety improvement program at the forefront of plant breeding innovation globally, and thereby ensuring its ongoing contribution to enhancing the economic, environmental and social prosperity of the Australian sugarcane industry.

7.0 EXPECTED OUTCOMES

More profitable varieties for the Australian sugarcane industry are the ultimate outcome for this project. Because the outcomes have been implemented in the BSES-CSIRO variety improvement program, the GES will have an immediate to long term impact on varieties. In the short term this is exemplified by as the use of the GES for the identification of elite clones at the 2007 selection meetings, while in the long term, varieties will be bred and selected through the implementation of the GES for identifying parental combinations and elite clones for which further testing or release is justified.

It is difficult to quantify the impact of the GES on the industry, because varieties released are only a small part of the population the GES is trying to improve in spite of its paramount importance. We can only compare expected genetic gains using empirical data sets. Based on the selection intensity for parental clones used in Table 12 and for varieties in Table 13, there

will be an average of 19% more genetic gain in progeny for parental selection and an average of 29% more genetic gain in varieties from the GES than from NMG. The gains from GES are likely to be underestimated because traits such as diseases were not utilized in this calculation.

8.0 FUTURE RESEARCH NEEDS

Further improvement of the GES developed in this project is possible in the future, and the most significant ways this may be done are suggested below:

1. **Multivariate BLUP**

   In theory, multivariate BLUPs are required to construct a breeding objective as described in Eqs [2] and [3] in order to account for the genetic correlations among traits of interest in the breeding objective. We have assumed that the genetic correlations among traits under consideration were negligible. While it may be valid in some trials, (e.g. a correlation of zero was often observed between CCS and TCH), more often this is an invalid assumption / simplification. However, there is currently no statistical technology available for modelling complicated genetic and residual structures under a multivariate model.

2. **Alignment of breeding values or genetic values from different sources**.

   Because of the fragmented nature of the information available to this project and unavailability of a practicable statistical package to combine these sorts of data, a clone may have several predictions for its breeding or genetic values. For example, a clone could have its breeding value predicted based on its progeny from PAT trials and also based on its own performance from FAT trials. Similarly, a clone could have its genetic value predicted from several series of PAT trials that resulted in several values. Due to the nature of different predictors (eg various accuracies of the predictors themselves versus various associations of predictors with breeding objective), it is critical to validate the relative merits of these predicted values.

3. **Validation of genetic models**

   All genetic models used in this GES so far, and also those proposed by Oakey *et al.* (2007) to partition total genetic effects into additive and others which we propose to use, are based on diploid species. Although arguments were made about the suitability of theory from diploid species in sugarcane, further research is required, especially when attempting to exploit non-additive genetic effects.

4. **Economic weights for non-sugar product traits**.

   Although it was highly desirable to derive economic weights for the non-sugar products, electricity and ethanol, the lack of information has prevented us from completing this work. However, with the increasing likelihood of non-sugar products from sugarcane having a major role in future sugarcane production systems, it is important for breeders to understand which traits will be important to target and what effort should be channelled to them. Once a potential product is recognised for commercial production, a laboratory-scale experiment should be carried out to simulate the range of available processing options and then determine the economic importance of traits associated with those products.

9.0 RECOMMENDATIONS

Because most of the outputs of this project have already been implemented in the BSES-CSIRO variety improvement program for crossing and selection, recommendations here are concerned with steps that could immediately improve the GES.

9.1 Improving the connectedness among trials over stages, sites and regions

One of the most outstanding features in modern genetic evaluation is to predict genetic effects more accurately by combining information. We have applied this in final assessment trials (FATs) to some extent, which resulted in higher accuracy in predicted genetic values and more gains than the traditional method. However, this could be improved further if series of FATs across years could be combined. It was found in Queensland wheat breeding trials that the magnitude of the genotype by year interaction was higher than the between genotype and site in the same year. If the same pattern occurs in sugarcane, it is imperative to combine series to evaluate varieties.

One solution to improve the connectedness among FATs across series and regions as was proposed by the SRDC project, BSS250. For example, the top 25 clones from one series should be repeated in the next series for better information about the 25 clones as well as connectedness between the two series. Similarly, the top clones from one region should be tested in FATs in other regions to improve the connection of FATs between regions.

9.2 Retrospective analyses of all selection data to better predict breeding and genetic values

With the newly developed statistical model to partition total genetic effects into additive and non-additive genetic effects, it would be beneficial to the BSES-CSIRO variety improvement program if historical CAT/FAT data could be re-analysed. A large number of clones in these CAT/FATs are currently being used as parents but no breeding value is available. Retrospective analysis could provide information about the additive genetic effects of those clones, though these estimates need to be compared with those predicted from PATs (see 8.2).

9.3 Re-examination of economic weights for any important changes in the production system from planting to sugar markets

Although the selection index is fairly robust to changes in economic weights, genetic gains will be affected by economic weights, especially with changes in different directions, or inclusion or exclusion of important traits. With the experience of the orange rust outbreak in 2000 and smut in 2006, for example, it is necessary to evaluate economic weights every five years to reflect changes that might make current selection indices less efficient.

10.0 LIST OF PUBLICATIONS

The following is a list of papers that have been published or in preparation. More papers are expected for publication from this project.

Smith AB, Stringer J, Wei X, Cullis BR (2007) Varietal selection for perennial crops where data relate to multiple harvests from a series of field trials. *Euphytica* 157:253-266.

Smith, AB, Stringer, JK and Cullis, BR (2007) Predicting additive and non-additive genetic effects from a series of multi-environment plant breeding trials where traits are affected by interplot competition. *Theoretical and Applied Genetics* **in preparation**.

Wei X, Jackson P, Cox M, Stringer J (2006) Maximising economic benefits to the whole sugarcane industry from the BSES-CSIRO sugarcane improvement program. *Proceedings of Australian Society of Sugar Cane Technologist* **28**, 181-186.

Wei X, Jackson P, Cox M, Stringer J (2006) Maximising economic benefits to the whole sugarcane industry from the BSES-CSIRO sugarcane improvement program. *Sugar Cane International* **25**, 6-9.

Wei, X, Jackson P, Stringer, J, Cox M. (2008) rEGV – a replacement for NMG in the Australian variety improvement program. *Proceedings of Australian Society of Sugar Cane Technologist* **30**, in preparation.

11.0 ACKNOWLEDGEMENTS

We are grateful for the input on the economics of the Australian sugar industry from the project's Industry Consultative Group, which consists of experts from CANEGROWERS, several mills, QSL, and BSES Limited, as well as independent input from staff at BSES Limited, SRI and Mackay Sugar Limited. We would like to thank Drs Brian Cullis and Alison Smith from NSW DPI for their significant contributions in the development of statistical models. We would also like to thank Dr Robert Magarey and Mr Barry Croft for their invaluable contribution to the determination of yield loss from diseases. Finally we thank Dr Nuno Borralho and Dr Mark Dieters for their advice on predicting genetic effects of sugarcane clones.

12.0 REFERENCES

Berding N, Hurney AP (2000) Suckering: a facet of ideotype selection and declining CCS in the wet tropics. In 'Proceedings of the 2000 Conference of the Australian Society of Sugar Cane Technologists'. Bundaberg Queensland, 2-5 May 2000. (Ed. DM Hogarth) pp. 153-162

Bernardo R (2002) 'Breeding for Quantitative Traits in Plants.' (Stemma Press: Woodbury, Minnesota)

Chao CP, Hoy JW, Saxton AM, Martin FA (1990) Heritability of resistance and repeatability of clone reactions to sugarcane smut in Louisiana. *Phytopathology* **80**, 622-626.

Chapman SC, Rattey A, Jackson PA (2005) 'Evaluatoin and re-structuring of regional selection programs to maximise efficiency and speed of cultivar release.' SRDC Project CTA028 Final report.

Cotterill PP, Dean CA (1990) 'Successful Tree Breeding with Index Selection.' (CSIRO: Melbourne)

Cox M, Hogarth M, Smith G (2000) Cane breeding and improvement. In 'Manual of Canegrowing'. (Eds M Hogarth and P Allsopp) pp. 91-108. (Buearu of Sugar Experiment Stations: Brisbane)

Gilmour AR, Gogel BJ, Cullis BR, Thompson R (2006) 'ASReml User Guide Release 2.0.' (VSN International Ltd: Hemel Hempstead, HP1 1ES, UK)

Hammnod K, Graser H-U, McDonald A (Eds) (1992) 'Animal Breeding the Modern Approach: A Textbook for Consultants, Farmers, Teachers and for Students of Animal Breeding.' (University of Sydney: Sydney)

Hazel DL (1943) The genetic basis for constructing selection indexes. *Genetics* **28**, 476-490.

Hazel LN, Dickerson GE, Freeman AE (1994) The selection index - then, now, and for the future. *Journal of Dairy Science* **77**, 3236-3251.

Henderson CR (1984) 'Applications of Linear Models in Animal Breeding.' (University of Guelph)

Hogarth DM, Reimers JF, Ryan CC, Taylor PWJ (1993) Quantitative inheritance of Fiji disease resistance in sugarcane. *Field Crops Research* **34**, 175-186.

Jackson P, Bonnett G, Hogarth DM, Chudleigh P, Wood A, Massey W (1999) 'Whole of Industry Economic Evaluation of New Varieties.' SRDC Project CTA041 Final Report.

Jackson P, McRae TA (2001) Selection of sugarcane clones in small plots: Effects of plot size and selection criteria. *Crop Science* **41**, 315-322.

Jackson PA, Wei X (2006) 'Improved selection systems and data analysis in sugarcane-breeding programs.' BSES Limited, SRDC Final Report SD6003, Brisbane.

Mrode RA, Thompson R (2005) 'Linear Models for the Prediction of Animal Breeding Values.' (CAB International: Wallingford, UK)

Oakey H, Verbyla A, Cullis B, Wei X, Pitchford W (2007) Joint modeling of additive, dominance and epistatic (genetic line) effects in multi-environment trials. *Theoretical and Applied Genetics* **114**, 1319 - 1332.

Offmann B, Hoarau J-Y*, et al.* (2001) Genetic analysis of resistance to leaf scald disease in sugarcane. In 'Poster présenté dans le cadre de 2nd Symp Sci Res Indian Ocean, Reduit, Mauritius'. April, (2001)

Rattey AR, Jackson P, Wei X, Cox M, Stringer J (2004) Opportunities to increase rates of parent improvement in Australian sugarcane breeding programs. *Proceedings of Australian Society of Sugar Cane Technologist (CD-ROM)* **26**.

Schneeberger M, Barwick SA, Crow GH, Hammond K (1992) Economic indices using breeding values predicted by BLUP. *Journal of Animal Breeding and Genetics* **109**, 180-187.

Smith HF (1936) A discriminant function for plant selection. *Ann Engenics* **7**, 240-250.

Stringer JK (2006) Joint modelling of spatial variability and interplot competition to improve the efficiency of plant improvement. The University of Queensland.

VanRaden PM (2001) Methods to combine estimated breeding values obtained from separate sources. *Journal of Dairy Science* **84(E. Suppl.)**, E47-E55.

Wei X, Jackson P, Cox M, Stringer J (2006) Maximising economic benefits to the whole sugarcane industry from the BSES-CSIRO sugarcane improvement program. *Proceedings of Australian Society of Sugar Cane Technologist* **28**, 181-186.

Wu KK, Heinz DJ, Hogarth DM (1988) Association and heritability of sugarcane smut resistance to races A and B in Hawaii. *Theoretical and Applied Genetics* **75**, 754-760.

APPENDIX 1 – Project Report by Magarey (2007)

BSES Limited

ESTIMATING DISEASE-ASSOCIATED YIELD LOSSES IN BREEDING SELECTION TRIALS

Endemic diseases: Pachymetra root rot, orange rust and yellow spot

By

Dr Robert Magarey

PR07004

Contact:
Dr Robert Magarey
Principal Researcher
BSES Limited
PO Box 566
Tully QLD 4854
Telephone: 07 4088 0707
Facsimile: 07 4068 1907
Email: rmagarey@bses.org.au

Australian Government
Sugar Research and Development Corporation

BSES Limited Publication
Project Report PR07004 **December 2007**

CONTENTS

Page No

SUMMARY

Previous work has shown that plant improvement stage 3 selection trials can be used to assess the relationship between yield and disease resistance with particular Queensland endemic diseases - principally Pachymetra root rot, yellow spot and orange rust. Results provide a means for fine-tuning the plant improvement program - ensuring that commercial varieties have just the right level of resistance to maximise high yielding ability while minimising disease-associated yield losses.

In this study, data from 2003-series stage 3 (FATs) trials in northern and central districts were studied to determine the relationship between resistance and yield for Pachymetra root rot and orange rust. As yellow spot only occurs in the high rainfall areas of northern Queensland, analyses for this disease were restricted to this region only. Brown rust was to be included in the study but the difficulty is assessing disease resistance in FATs, and the lack of disease, made obtaining data to brown rust impossible. Additional data were available for Pachymetra root rot for northern series trials (1995-2004) providing more detailed information for this disease.

The data analysis showed that losses to Pachymetra root rot can be very significant in both northern and central districts (>40% for tonnes cane and tonnes sugar in individual trials) and that losses consistently are above 10%. CCS was largely unaffected while tonnes cane and tonnes sugar/ha were the main yield components affected.

Yellow spot caused inconsistent losses in northern trials, but on average still reduced yield (tonnes cane and tonnes sugar) by over 10%. In some years and in some locations losses were reduced considerably.

Orange rust caused huge yield losses in the year it was first detected (2000); at some locations losses in the 2000 plant crop were nearly 60% (tonnes cane and tonnes sugar). In later years, losses were greatly reduced and similar to those caused by yellow spot.

Environmental variables are very likely to have influenced yield losses caused by these diseases. Past analyses have shown that the central district favours spore germination conditions for *Puccinia kuehnii* (orange rust pathogen) and yield losses caused by orange rust seemed to be greater in that region. The high orange rust infection pressure in 2000, associated with very extensive plantings of the susceptible Q124, are very likely to have contributed to the large orange rust-associated yield losses seen in plant crops in that year. Lower losses later were probably a result of lower infection pressures associated with reduced cropping of the susceptible Q124 and an increase in bio-control of the disease. Further investigations are necessary to quantify the effects of the bio-controls. There was some linkage between Pachymetra root rot and annual rainfall though this needs to be investigated further.

A consideration of crop resistance profiles for northern and central crops in 2004, and assessment of the Resistance Index (RI) of clones in the plant improvement program, provided a gauge of the need to select for higher levels of disease resistance. RI values suggest there is a high level of orange rust resistance in FAT clones, intermediate level of Pachymetra root rot resistance, and less resistance to yellow spot.

It is recommended that FAT substation procedures be examined in the light of the interaction between residual Pachymetra spore populations and yield effects in crops subsequent trial plantings.

1.0 BACKGROUND

Sugarcane diseases exert a significant influence on the yield of commercial crops in Queensland. There are a number of endemic diseases that are widely dispersed through the industry that reduce commercial crop yields. A long-term, concerted disease control program in Queensland has reduced the influence of these diseases, but has not been able to entirely eliminate yield effects. The most important endemic diseases are listed in Table 1 and are caused by bacterial, fungal, viral and unknown causal agents.

Table 1 The most important endemic diseases in the Queensland sugarcane industry

Disease	Causal agent	Species
Brown rust	Fungus	*Puccinia melanocephala*
Orange rust	Fungus	*Puccinia kuehnii*
Yellow spot	Fungus	*Mycovellosiella koepkei*
Chlorotic streak	Unknown	Unknown
Mosaic	Virus	Potyvirus
Leaf scald	Bacterium	*Xanthomonas albilineans*
Fiji leaf gall	Virus	Fiji disease virus
Ratoon stunting disease	Bacterium	*Leifsonia xyli s.sp. xyli*
Pachymetra root rot	Fungus	*Pachymetra chaunorhiza*
Nematodes	Nematodes	Various species

Until recently, a limited amount of research had been undertaken examining the yield effects of these diseases, particularly the influence of disease resistance on yield losses. Yield loss research has been summarised by Magarey and Croft (1998). It is accepted that endemic diseases can cause significant yield losses in susceptible varieties, but losses in varieties of intermediate resistance, and the influence of the environment on these losses is largely unknown.

1.1 Factors affecting disease occurrence

A number of factors interact to affect the occurrence and severity and sugarcane diseases. These are important in governing yield losses and are described briefly below.
There are several key environmental factors that influence disease incidence (particularly leaf diseases); these are relative humidity, temperature and rainfall. The plant host also provides a key influence on disease incidence (varietal resistance), and pathogens may be unstable leading to variation in their ability to infest their host. Plant pathologists use the following diagram (Figure 1) to illustrate the relationship between these variables.

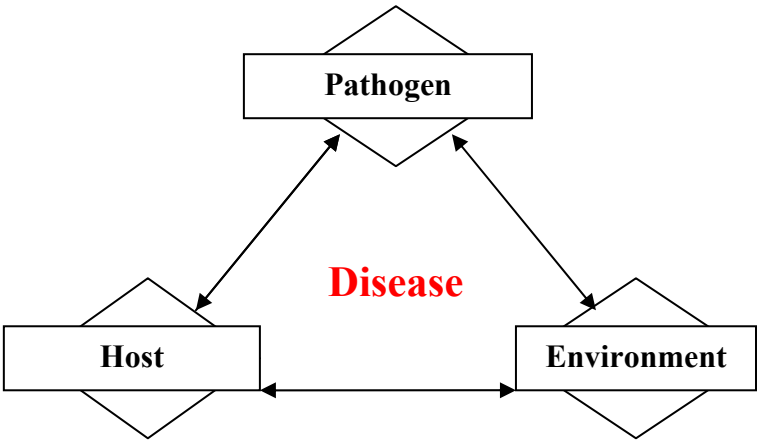


Figure 1 The relationship between the host, pathogen, environment and disease

1.1.1 Environmental variation

As the environment varies, the ability of the pathogen to invade the host also varies. This is very important with a number of diseases, especially leaf diseases. Relative humidity and temperature vary constantly and leaf pathogens have specific environmental requirements for different aspects of the disease cycle. This is particularly evident during the infection process, where conditions required for spore germination and hyphal spread into the host tissue are very specific and greatly influenced by the pervading environmental conditions. In years when rainfall is below average, low relative humidity may make conditions unsuitable for spore germination. As a consequence, leaf disease severity, and hence associated yield losses, will also be very low. In other years above average rainfall may favour the disease and lead to large yield losses. For this reason, assessing the effect of a leaf disease on yield in any one year (alone) will be insufficient for quantifying the long term yield effects of that disease. Losses would be better estimated by collating 10 years of data where below average, normal and above average rainfall and temperature conditions are experienced.

1.1.1.1 Climatic requirements for leaf pathogens

The conditions needed for disease to occur also vary between pathogens. Previous work has shown that environmental requirements for spore germination in *Puccinia melanocephala* (brown rust) and *P. kuehnii* (orange rust) vary significantly; *P. melanocephala* requires free water (as in a dew) as does *Mycovellosiella koepkei* (yellow spot pathogen) while *P. kuehnii* requires relative humidity over 97% (Staier et al, 2004). These requirements are illustrated in Figure 2. Temperature also exerts a major influence on disease incidence; the requirements for *Puccinia melanocephala, Puccinia kuehnii* and *Mycovellosiella koepkei* are illustrated in Table 2. These two interacting factors exert major effects on leaf disease occurrence. Brown rust occurs during spring months after cool nights (with dew) and with warm sunny days. Orange rust is favoured by wet season conditions where relative humidity and temperatures are high; these conditions also favour yellow spot.

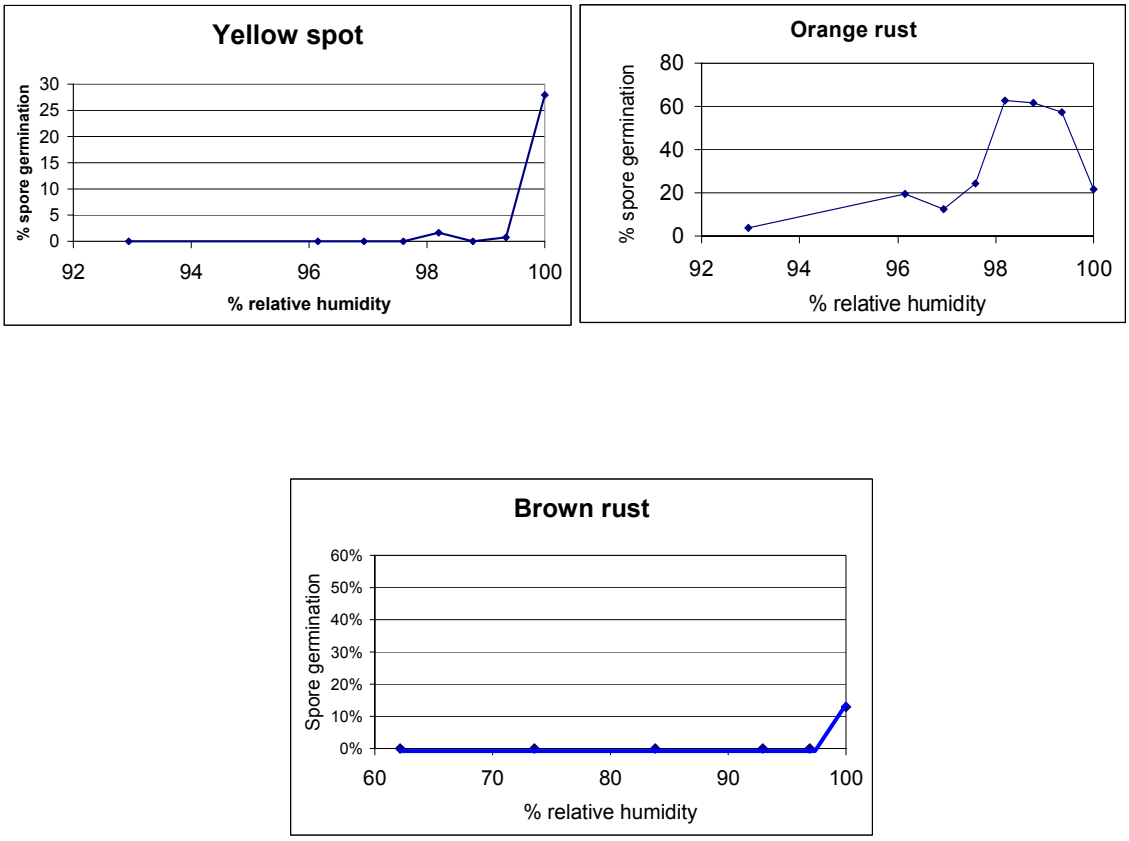


Figure 2 **Graphs illustrating the relative humidity requirements of the three major leaf disease pathogens in Queensland - *Mycovellosiella koepkei* (yellow spot), *Puccinia kuehnii* (orange rust) and *P. melanocephala* (brown rust). Germination of spores of *P. melanocephala* in free water (not shown) was optimal with 52% of spores germinating**

Table 2 **Optimum temperatures for spore germination in each pathogen**

Disease	Pathogen	Optimum temperatures
Brown rust	*Puccinia melanocephala*	11-27^{o}C
Orange rust	*Puccinia kuehnii*	17-23^{o} C
Yellow spot	*Mycovellosiella koepkei*	20-30^{o}C

Some analysis has been undertaken comparing the favourability of different locations for orange rust occurrence (Figures 3 and 4), based on optimum spore germination conditions. These data provide some indication as to where the disease may exert its greatest influence. Ideally, monitoring of weather conditions in each cane-growing area would provide the best information as to when (or if) optimum conditions for disease occurrence occur.

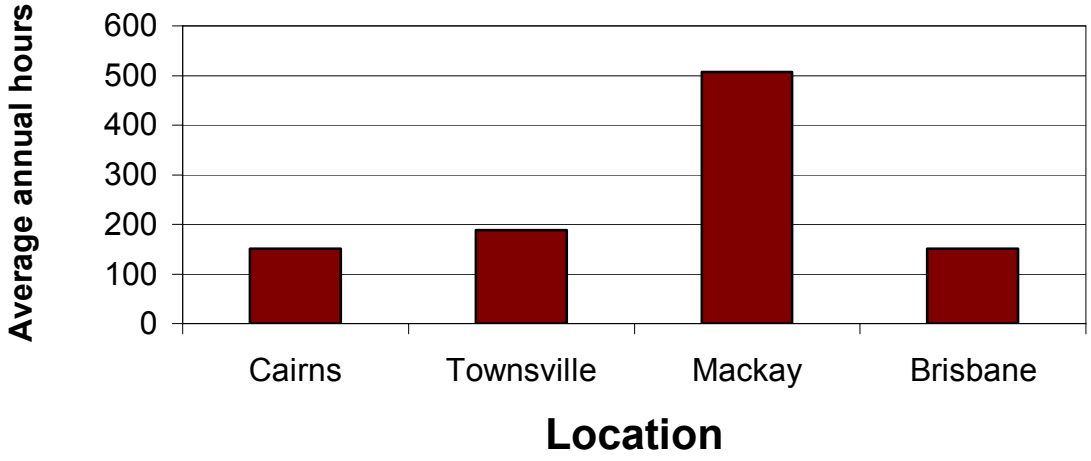


Figure 3 **Average annual number of hours suited to the germination of spores of *Puccinia kuehnii* in major Queensland centres**

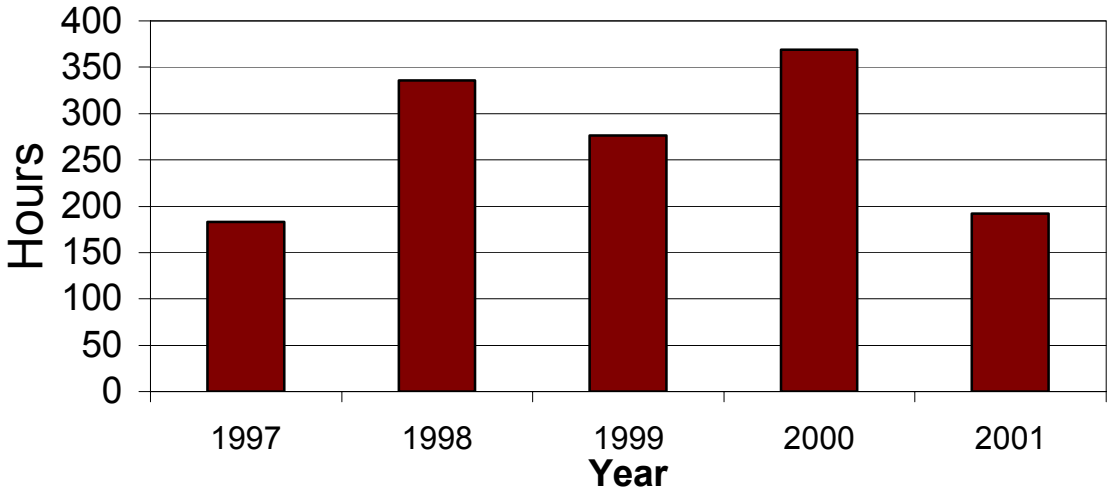


Figure 4 **A comparison of accumulated hours of suitable conditions in the Mackay area for *Puccinia kuehnii* (orange rust) spore germination in the 1997-2001 period**

1.1.1.2 Climatic requirements for soil borne pathogens

For soil-borne diseases, such as Pachymetra root rot, environmental variation is an important issue too. Research conducted in the early 1990s, on the wet tropical coast, suggested that rainfall had a very important role in governing disease incidence (Magarey and Soper, 1992). The authors found a relationship between spore population and rainfall beneath two varieties (Q117 and Q124) in the Gordonvale-Fishery Falls area. Higher rainfall was associated with increased spore populations, suggesting higher disease levels were present in higher rainfall districts.

The soil environment is more stable than atmospheric conditions, the latter may vary drastically within even a few minutes or hours. Soil conditions tend to buffered by much slower changes in soil moisture conditions. However soil environmental factors can again exert a significant influence on soil-borne disease incidence.

1.1.2 Pathogen variation

Pathogen variation can have an important influence on disease levels. The propensity of pathogens to change varies with species; some pathogens are stable while others are prone to mutation.

For many years, orange rust was a very rare and minor pathogen - so rare that few people had ever seen the disease. However, with the wide-spread planting of the variety Q124 in the 1990s, opportunity arose for the orange rust pathogen, *Puccinia kuehnii,* to mutate. Pathogen mutation is thought to have led to the previously resistant variety Q124 suddenly becoming susceptible to the disease; variation in the pathogen led to a strain that 'overcame' the disease resistance of Q124. Such variation in a pathogen has been seen regularly in stem rust of wheat caused by *Puccinia graminis*. Changes in *P. kuehnii* immediately had a huge influence on disease levels, which rose from minor to where the disease affected over 140,000 ha of crops in the year 2000 (Magarey, 2005). Orange rust caused one of the most significant disease epidemics in the history of the Australian sugar industry simply because pathogen variation made a previously resistant variety susceptible.

1.1.3 Host variation

Host variation is of great significance for the Australian sugar industry too, providing a key disease control strategy. The industry relies on this variation for selecting varieties with sufficient resistance to the major diseases, releasing only these to the industry. A major activity of sugar industry plant pathologists is to assess hosts (varieties) for resistance to the major endemic diseases in the Australian industry; the aim is to reduce disease-associated yield losses to negligible levels. The variation in disease resistance and how this affects yield losses with several major endemic diseases is the focus of the research described in this report.

Host selection is normally based on specific disease resistance screening trials. The BSES Experiment Station at Woodford undertakes resistance screening for major Australian sugarcane diseases; similar work is undertaken at the BSES Tully Experiment Station. There is also some natural selection for host resistance in breeding selection trials. The presence of major endemic diseases will lead to yield losses in susceptible clones; the selection of clones on yield will naturally select for disease resistance - if the endemic disease is significantly affecting sugarcane yield. The research detailed below aimed to quantify this very issue - how much are the major endemic diseases affecting the yield of clones in routine plant improvement selection trials.

1.1.3.1 Routine resistance screening trials

BSES pathologists have routinely screened for resistance with many of the endemic sugarcane diseases present in the Australian sugar industry. These include those listed in Table 3. In each case, pathogen inoculum is applied to each test clone, either mechanically or through the application of infested vectors.

Table 3 Detailed below are the routine resistance screening trials conducted by BSES, the timing of these in the plant improvement program and the nature of the test

Disease	When	Test
Fiji leaf gall	Early and late in selection program	Glasshouse and field
Mosaic	Late in selection program	Field
Red rot	Late in selection program	Field
Leaf scald	Early and late in selection program	Field
Pachymetra root rot	Mid-selection program (some areas)	Glasshouse
RSD	Not routine - late	Field

There is no specific resistance screening for ratoon stunting disease (RSD) as varietal resistance is not the major disease control strategy for this disease. Sanitation (sterilisation of contaminated equipment) and the planting of disease-free planting material are the two cornerstone control strategies for RSD.
For many years, there remained no resistance screening for yellow spot, except indirect screening associated with selection based on yield variation in clones in the breeding program. Specific brown rust selection occurred when the disease first appeared in the Australian sugar industry in 1978, but subsequently yield selection in the plant improvement program was considered sufficient to lead to the discard of highly susceptible clones. Up until 2000, selection for orange rust was also indirect - but the very rare occurrence of the disease meant that in reality selection was unnecessary.

1.1.3.1.1 Rating system for disease resistance

In all the routine resistance screening trials undertaken by BSES, standard varieties of known field reaction are included in each new trial. The incorporation of a range of varieties that vary from resistant to highly susceptible provides a basis for assessing the resistance of each test clone incorporated into the resistance screening trial. Such standards also assist in dealing with variation in environmental conditions that may be affecting disease incidence. If in some years low disease levels result from sub-optimal weather conditions, lower disease levels will also occur in the susceptible standard canes. By relating disease incidence in the standard varieties (through regression analyses), such variation is accounted for. It has been found that the relationship between **relative** disease incidence and clone susceptibility remains relatively constant no matter what levels of disease occur in the screening trial. Determining the relationship between disease incidence in the standard varieties and their resistance rating in the current trial therefore enables a resistance rating to be applied to each of the test clones. The rating provides an estimate as to how that clone will react to the presence of the disease under commercial crop conditions.

The standard international rating system for varieties is based on a sliding 1 to 9 scale where 1 implies a high level of resistance to the disease, and 9 implies a high level of susceptibility. This is outlined in Table 4.

Table 4 The resistance-susceptibility categories and how they relate to the standard international 1 to 9-based rating system

Resistance category	Resistant	Intermediate	Susceptible
Resistance rating (1 to 9 scale)	1, 2 or 3	4, 5 or 6	7, 8 or 9

Negligible commercial losses could be expected in commercial varieties rated 1, while very high yield losses could be expected in varieties with a 9 rating. BSES has ensured that for the most important diseases, all susceptible clones in the plant improvement program are discarded (not released to industry), so as to avoid significant commercial yield losses. This varies a little between diseases and also depends in some cases on inoculum levels present in commercial fields. A sub-optimal disease environment in a district may allow more susceptible varieties to be grown compared to districts where conditions are highly favourable.

The objectives of the study reported here were to relate disease-associated yield losses with varietal resistance with several of the more important, but lesser researched endemic diseases. For some of the major diseases, other factors besides current commercial yield losses decide the basis for clone selection; these include epidemiological considerations and disease control measures. Examples include leaf scald, Fiji leaf gall, mosaic, chlorotic streak and red rot. These diseases were not included in this study.

1.2 Other factors affecting yield losses associated with endemic diseases

Other factors besides the immediate environmental conditions, pathogen variation and host resistance may influence disease-associated yield losses.

1.2.1 Timing of disease occurrence

The onset of suitable environmental conditions for disease may vary considerably by year. For instance, wet season conditions may begin in December, rather than February on the wet tropical coast. This leads to high relative humidity, warm temperatures and good spore germination conditions when the growing crop is still small. For orange rust, this leads to high disease levels during the major crop growing period. In this case there is much opportunity for the disease to significantly affect biomass production - and reduced tonnages (tonnes cane per ha) are likely. If these conditions prevail for many months (a long wet season) the crop canopy may be diseased well into the normal maturity period, when CCS levels normally rise. If the canopy is badly affected, CCS will also be reduced. A number of combinations (scenarios) may arise, each influencing the yield effects caused by leaf diseases. Possible outcomes are described in Table 5.

Table 5 The potential effect of a leaf disease on crop production, depending on when environmental conditions favour disease occurrence

Disease occurrence	Crop biomass effects	Sugar content effects
Early through to late	Yes	Yes
Early only	Yes	No
Late only	No	Yes
Negligible occurrence	No	No

Timing of disease incidence, and disease severity through the growing period, therefore have a huge influence on yield losses. The effect of a disease on yield is therefore best provided not by one single disease assessment during the season, but by the continuous

monitoring of disease severity during the growth of the crop. Of course this requires a very large resource input which is rarely available in the current research environment.

In the analyses reported below for orange rust and yellow spot, it is not surprising therefore that in some years biomass yield (tonnes cane per ha) is reduced by these diseases, while in other years sugar content only is affected, and in still others - there is no associated yield effect.

1.2.2 Spore production and dispersal

For a pathogen to influence crop yields the pathogen must first reach the susceptible crop. This is related to spore production in already diseased crops and dispersal of these spores to previously disease-free crops.

1.2.2.1 Leaf diseases

The presence of pathogen spores is an important factor influencing disease incidence. A long period of favourable conditions for a leaf disease will lead to a large number of disease cycles - where initial pathogen infection is followed by disease development and spore production from infested tissue. The population of spores in the atmosphere increases considerably as the length of the period favouring the disease increases. With the rust pathogens, spore production can be extensive, and 'clouds' of spores may hover or blow across cane growing districts applying intense disease pressure to susceptible crops.

This was the case with orange rust in the Mackay district in the year 2000. Farmers reported that after moving through diseased crops, their shirts were dis-coloured orange by rust spores. Enormous atmospheric spore populations were associated with over 100,000ha of badly diseased crops of the susceptible Q124 being cultivated in the Mackay area. In the Herbert district, even house veranda floors became orange due to large populations of *P. kuehnii* spores. Back then we had no means to quantify the atmospheric populations. As the proportion of Q124 decreased so too did the spore populations; the intensity of disease infection pressure decreased as a result.

1.2.2.2 Pachymetra root rot

As for leaf diseases, spore populations are a key factor in controlling Pachymetra root rot incidence and severity. Previous work by BSES has shown that varietal resistance significantly influences spore populations under commercial crops; there is a very strong relationship between varietal resistance and spore counts (Magarey, 1991; Figure 5).

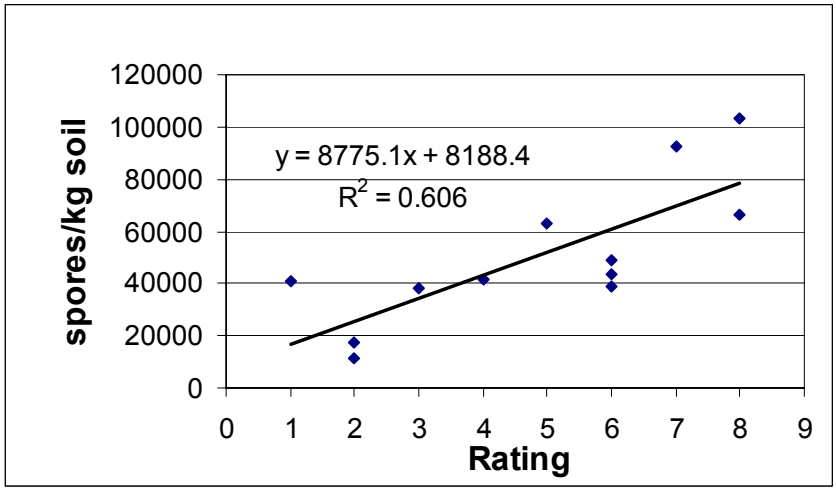


Figure 5 Pachymetra root rot spore populations developing over a plant and first ratoon crop under varieties of differing resistance (1-9 scale) in the Mackay district

Research has also shown that yield losses in susceptible varieties are also related to spore populations (Magarey, 1994; Figure 6).

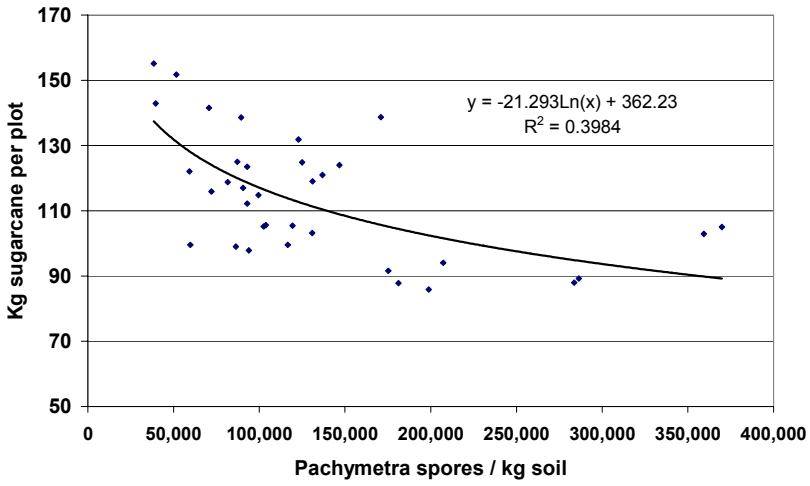


Figure 6 The effect of increasing Pachymetra soil spore inoculum populations on the yield of a susceptible variety (Q90) in northern Queensland

BSES Tully provides a soil testing service for farmers that quantifies the spore population under commercial crops in Queensland (Magarey, 1989). Likely yield effects are predicted from spore count information so that farmers can select varieties with sufficient resistance to the disease to minimise yield losses.
In plant improvement selection trials, the influence of clones on spore populations has been investigated. Large variation in spore populations across previous selection trial plots has been found (from 38,000 spores / kg to 350,000 spores / kg).

The replanting of this site with a susceptible variety, and monitoring of spore populations in the plant, first and second ratoon crops illustrates how spore populations may increase

dramatically in successive crops. Average populations for plant, first ratoon and second ratoon crops are illustrated in Figure 7.

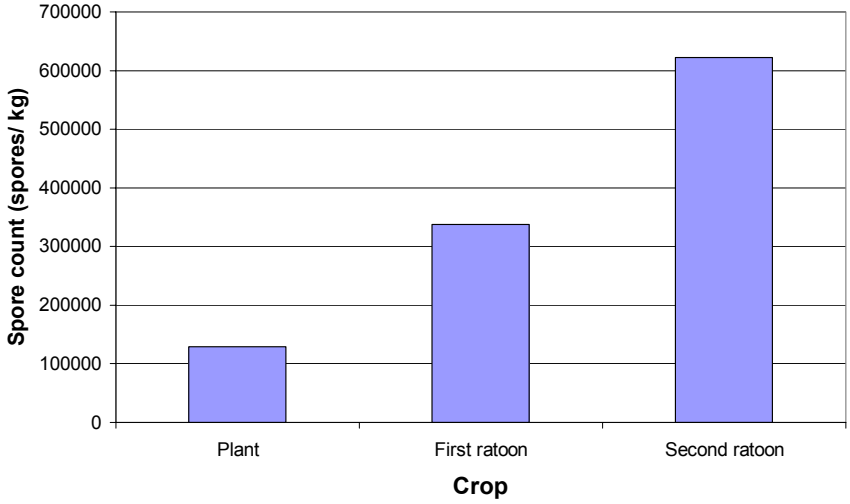


Figure 7 Average Pachymetra spore population increases in plant, first and second ratoon crops under a susceptible variety (Q90) at Miriwinni, northern Queensland

Planting selection experiments sites without a very long fallow period (five years or longer) will therefore lead to very significant interaction between initial spore populations, clone susceptibility and final yield. Some clones will be planted on plots with low initial Pachymetra populations while others will be planted on plots with high initial counts - depending on the resistance of the clones planted previously in those plots. Selection for yield will therefore be compromised by the spore populations present at the initiation of the selection trial

1.3 Rainfall during the study period

In considering the effect of the endemic diseases, a factor to be considered is the rainfall received during the study period. Rainfall affects relative humidity and the length of time water is present on leaf surfaces - as well as affecting soil moisture conditions. Both affect the severity of diseases, either leaf diseases or Pachymetra root rot. Rainfall during the 1995-2004 period for Tully is presented in Figure 8.

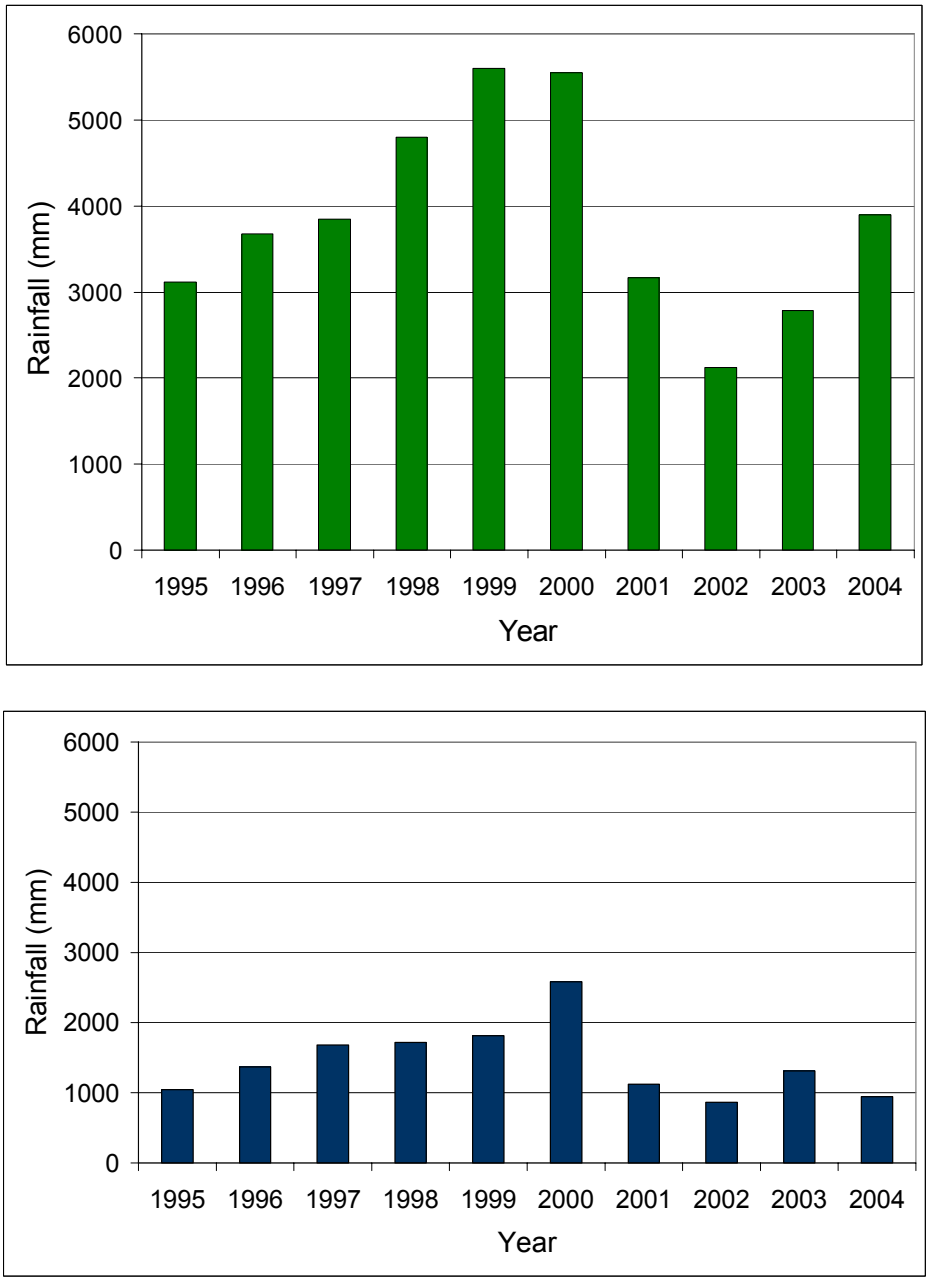


Figure 8 Rainfall during the 1995-2004 study period for Tully (top) and Mackay (bottom)

1.4 Assessing yield losses in plant improvement selection trials

1.4.1 Basis of the method

The basis of using plant improvement trials to investigate the relationship between varietal resistance and endemic disease-associated yield losses was explored only in recent times by Magarey and Bull (2001, 2003) and Magarey et al (2002, 2004). Briefly the method relied on the fact that where experiments incorporating a large number of clones are planted, the average yield of susceptible clones will be lower than the average yield of resistant clones - if the disease is exerting significant yield effects at that site. The following diagram illustrates this point (Figure 9)

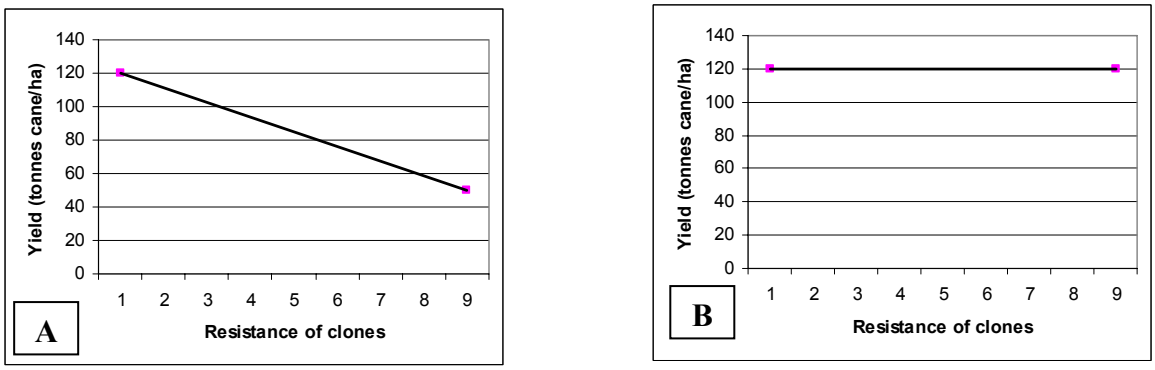


Figure 9 Theoretical relationships between resistance of clones and the yield (tonnes cane per ha) of clones in plant breeding selection trials. Graph A illustrates the situation where the endemic disease is influencing the yield of clones, while B illustrates no yield effect

1.4.2 Statistical considerations

Although individual clones may vary considerably in their genetic yielding ability, the use of the average yield for all clones of the same resistance category enables this variation to be 'removed' from the regression analysis and the effect of the disease on yield parameters to be visualised. If a regression between the yield of all clones (not the average yield of clones of the same resistance rating) was adopted, some statistical inconsistencies would arise. Yield variation incorporated into the regression would vary with resistance rating; resistant clones (1 rating) would not be influenced by the disease and variation in yield would be controlled by genetic factors for yield plus the influence of susceptibilities to other diseases and environmental influences. On the other hand, susceptible clones would include the same variation plus variation arising from yield effects from the endemic disease.

In considering the disease resistance-yield relationship, no yield effect of a disease will result in no significant regression between yield and resistance (a scatter of points); a significant regression would be expected with a disease-associated effect on yield. There may be either positive or negative relationships between resistance and yield parameter. Examples include the following; it is known that crop biomass reduction associated with some diseases causes a rise in sugar content (CCS) in affected crops. This may be evidenced by a positive relationship between susceptibility and CCS. In this instance a negative relationship between tonnes cane / ha and susceptibility would also be expected.

1.4.3 Disease assessed

The ability to use this method to assess yield losses obviously depends on the uniform and broad distribution of pathogen inoculum in plant improvement selection trials. This in turn depends on the nature of the disease. In considering these parameters, regression analyses were undertaken for orange rust, yellow spot, brown rust and Pachymetra root rot in selected districts. These diseases are not evenly distributed through the industry because the environment in some districts favours the disease while in others it does not. Table 6 provides details on where these diseases are found in the Queensland sugar industry.

Table 6 The general incidence of yellow spot, orange rust, brown rust and Pachymetra root rot in Queensland sugarcane districts. Some local variation occurs - for instance in some parts of southern Queensland, Pachymetra root rot is not present

Sugarcane regions / ***Disease***	**Northern** *(Coast)*	**Northern** *(Tableland)*	**Burdekin**	**Central**	**Southern**
Orange rust	+	+	+	+	+
Brown rust	[1]+	+	+	+	+
Yellow spot	[2]+	+	-	-	-
Pachymetra root rot	+	-	-	+	+

Notes:

1. Brown rust incidence is highly variable - in some years environmental conditions favour disease incidence in August, other times in November, other times in-between these months while in other years there is little disease occurring.
2. Yellow spot is favoured by high rainfall and the highest disease levels occur in the Babinda-Tully districts. In wet years, the disease may also be significant in the Herbert, and be found in the Burdekin and Central districts.

It should be borne in mind that the incursion of sugarcane smut in June 2006 caused a rapid re-deployment of pathology staff and the inability to undertake some project activities. This particularly applied to measuring environmental data and relating these to disease-associated yield losses.

2.0 OBJECTIVES

- Assess the resistance of clones to Pachymetra root rot, orange rust, yellow spot and brown rust in plant improvement stage 3 trials
- Relate disease resistance to the yield of clones (using regression analyses)
- Summarise losses from each disease in the 2003 series trials.
- Provide information to the plant breeders to ensure appropriate breeding strategies.

3.0 RELATING YIELD AND DISEASE RESISTANCE

Research was conducted in several sugarcane regions over several years to investigate the relationships between the different endemic diseases and the resistance of clones. With orange rust and yellow spot, this required the assessment of clone resistance within the plant improvement selection trials (relying on natural disease incidence) while with Pachymetra root rot, results from routine glasshouse screening trials were used for northern district analyses.

In the plant improvement program, four or five trials are planted each year (the series is labelled by the year in which the clones are planted) in each district in widely dispersed commercial fields; the same clones are usually planted in each trial. Identification of consistently high yielding clones in most trials in a district provides the basis for selecting clones for possible commercial release. In the leaf disease-related work undertaken, identification of the resistance of clones in one trial in a series was sufficient to obtain disease resistance ratings for use in analyses in each of the other trials of the same series.

Accordingly, appropriate trials in each series were selected for assessing clonal resistance. Some trials were unsuitable for assessment because most plots were lodged (making access to clones impossible) or there was insufficient disease present due to poor environmental conditions operating at that site (this would be expected to result in no relationship between disease resistance and yield at that site).

3.1 Analyses

The same procedure was applied to analyses with each disease; analyses included the following: -

- Regression analyses between yield parameters (CCS / tonnes cane per ha / tonnes sugar per ha) and clone resistance for individual trials.
- Identification of regressions where the r^2 was 0.20 and above (i.e. the disease susceptibility could explain at least 20% of the yield variation)
- Calculation of maximum yield losses in each selection trial based on the regression equation.
- Determination of the average yield loss for each yield parameter using data from all selection trials in the same series. In undertaking this, yield data for each resistance rating were first expressed as a percentage of the mean yield for the 1-rated clones; this enabled easy calculation of the percent yield losses for clones of any resistance rating. A regression was undertaken for each set of mean data to provide an overall assessment of the yield losses associated with that disease in that particular selection series.

The research conducted with each disease, and the results, are outlined in individual disease sections below. Over 270 regressions were undertaken using data from over 90 individual selection trials.

4.0 PACHYMETRA ROOT ROT

4.1 Introduction

Pachymetra root rot (caused by *Pachymetra chaunorhiza*) is of major concern in the northern, Herbert, central and Bundaberg (part of the southern) districts (Magarey et al, 2004). The disease has been found on only one farm on the Atherton Tableland (why this is mentioned will become clear later) and only in some parts of southern Queensland. Pachymetra root rot is of limited occurrence in New South Wales.

4.2 Method

4.2.1 Resistance screening

4.2.1.1 Northern program

The main resistance screening program for Pachymetra root rot is undertaken at BSES Tully and incorporates a glasshouse-based screen that includes artificial soil infestation, maintenance of constant soil environmental conditions and growth of test plants in small pots for 12 weeks (Croft, 1989). As a result, resistance ratings have a high level of repeatability and the method provides a reliable means for assessing the field resistance of commercial varieties. Magarey (1991) investigated the relationship between glasshouse-

based resistance ratings and the production of spores beneath field plots of the same varieties. The relationship was generally very good, though there were a few exceptions - where the glasshouse rating was different for a commercial variety compared to what was expected from spore population studies.

Tully-based resistance screening of clones in stage 3 trials is only possible for clones from the northern program. Quarantine issues associated with Fiji leaf gall (and now sugarcane smut) prevent all clones from stage 3 trials in the central and southern programs from being assessed in routine glasshouse trials. In analyses of the northern trials reported here, regression analyses utilised existing stored resistance data on clones planted in northern stage 3 trials, and yield data for the same trials collected and stored by plant breeders. These data are available from 1995 to 2004.

4.2.1.2 Central Program

In general, clones from the central and southern programs were not able to be assessed for Pachymetra resistance at BSES Tully, though a few of the more important ones are sent through quarantine and are eventually screened in Tully. To undertake yield loss research for stage 3 trials in central and southern districts, an alternative method was trialled. This was time, labour and resource intensive and required the collection of soil samples from beneath individual plots of all clones in a selection trial and assay for Pachymetra spores. The following method was employed.

4.2.1.2.1 Soil sampling and resistance ratings

Soil samples were collected from beneath all plots (two replicates) of each clone in one trial from the series being analysed. Details of the trials sampled are included in Table 7. Soil sampling was to 25cm depth and samples were collected using 4.5 cm 'Dutch-head' augers. Soils from each plot were dispatched to Tully where they were sieved, mixed thoroughly, sampled and processed for Pachymetra root rot assay as described by Magarey (1989). Mean data were calculated for each clone and the lowest and highest populations identified; the clone with the lowest spore count was identified as a 1-rated clone ('standard') while the clone with the highest spore population was identified as a '9-rated' standard. The equation of the straight line linking these two points (resistance rating and spore population) was used to describe the relationship between resistance rating and spore population in all clones. Resistance ratings for each clone were then applied using this equation.

Where glasshouse-based resistance ratings were available for the most important clones and varieties, correlation of the two ratings (glasshouse and field spore-based ratings) was undertaken.

4.3 Plant improvement series investigated

Table 7 provides information of the selection trial series assessed for the relationship between Pachymetra resistance and yield.

Table 7 Plant improvement series trials analysed for the relationship between Pachymetra root rot resistance and yield

District	Northern *(Coast)*	Northern *(Tableland)*	Central
Series / Year			
1995	[1]+		
1996	+		
1997	+		
1998	+		
1999	+	+?	+
2000	+		
2001	+		+
2002	+		
2003	+	+	+
2004	+		

[1]+ denotes that all trials (usually 4-5) in that series were analysed for the relationship between resistance and yield parameters.

4.4 Results

4.4.1 Northern trials

There was a fairly consistent relationship between Pachymetra root rot resistance and yield parameters in the northern series trials investigated. Pachymetra root rot was not associated with yield losses in all trials, but there were a number where disease resistance explained a sizable proportion of the variation in clone yield (using average clonal yields). The r-squared values (>0.20) for each trial regression between yield parameter and Pachymetra resistance, details of the regression equations and maximum yield losses occurring in 9-rated clones in each trial where the r-squared was >0.20, are contained in the Appendix.

For each trial at a certain location within a district and series (year), the mean yield data for clones of the same resistance rating were expressed as a percentage of those for the 1-rated clones. In the northern district, this led to either 4 or 5 separate sets of information where the % yield loss for yield parameters could be related to each yield component (CCS, tonnes cane per ha, and tonnes sugar per ha). The mean percentage yield data was then calculated over all trials for the one series (year) in each district. Mean data combining plant and first ratoon data were calculated. This provides an overall assessment of the relationship between Pachymetra resistance and % yield loss. For Pachymetra root rot, average loss figures were calculated for the 1999 series trials in northern Queensland, and for the 2003 series trials in the same district; graphs of these data are presented in Figure 10.

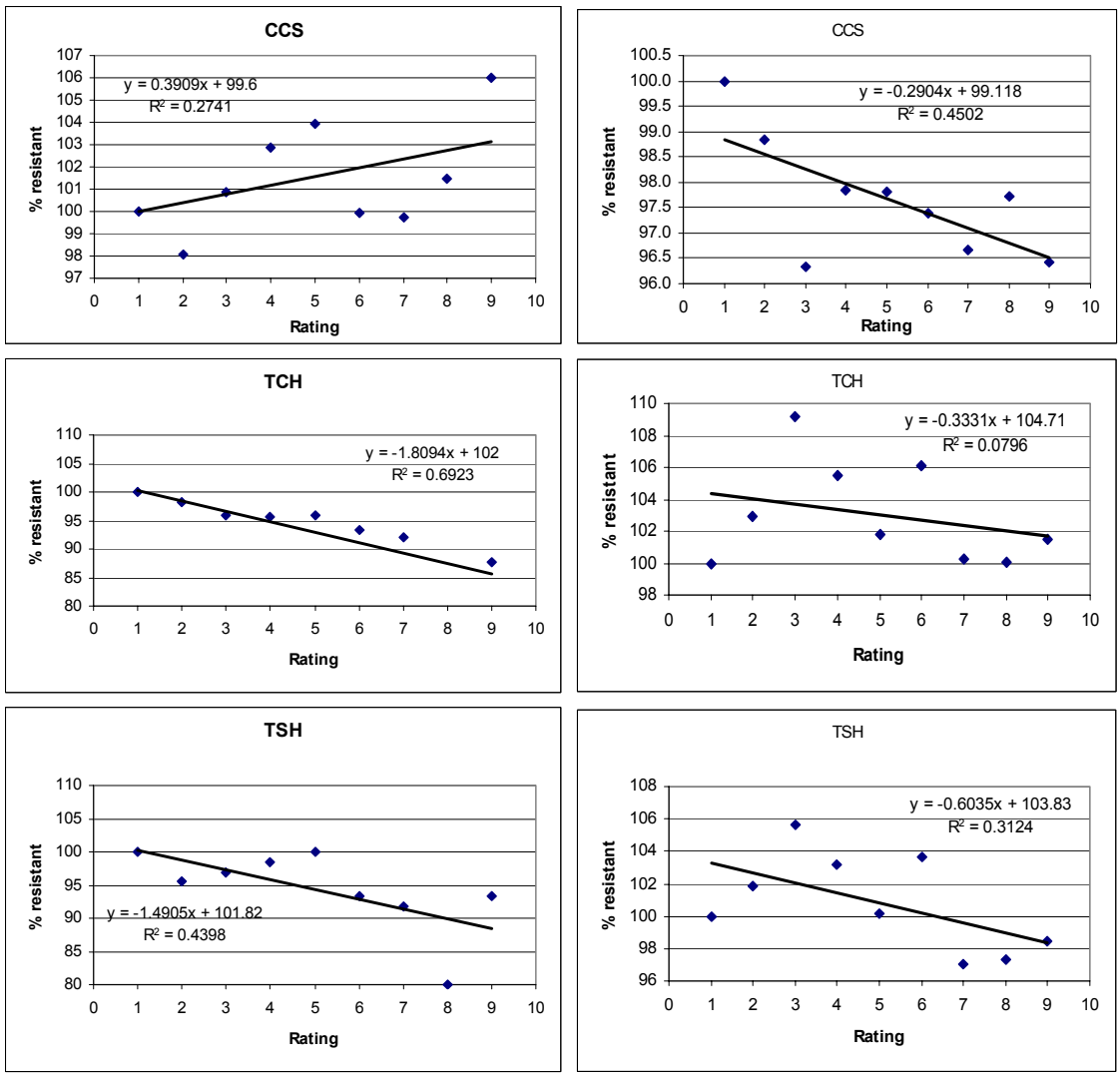


Figure 10 Average yield data (CCS, tonnes cane and tonnes sugar) for all northern trials in the 1999 (left) and 2003 (right) trials; regressions indicate the relationship between Pachymetra root rot resistance and yield

Table 8 Calculated percent Pachymetra root rot-associated yield losses for susceptible clones (compared to resistant clones) in plant improvement selection trials: 1999 and 2003 series. Data are mean figures for plant and first ratoon crops

Yield parameter Trial series	Tonnes cane/ha	Tonnes sugar/ha
1999	14.5	11.9
2003	2.7	4.8

Average data were also calculated for all trials in the period 1995-1999, and for the following five years, 2000-2004. These are included in Figure 11. Mean data for all years (1995-2004) were also calculated.

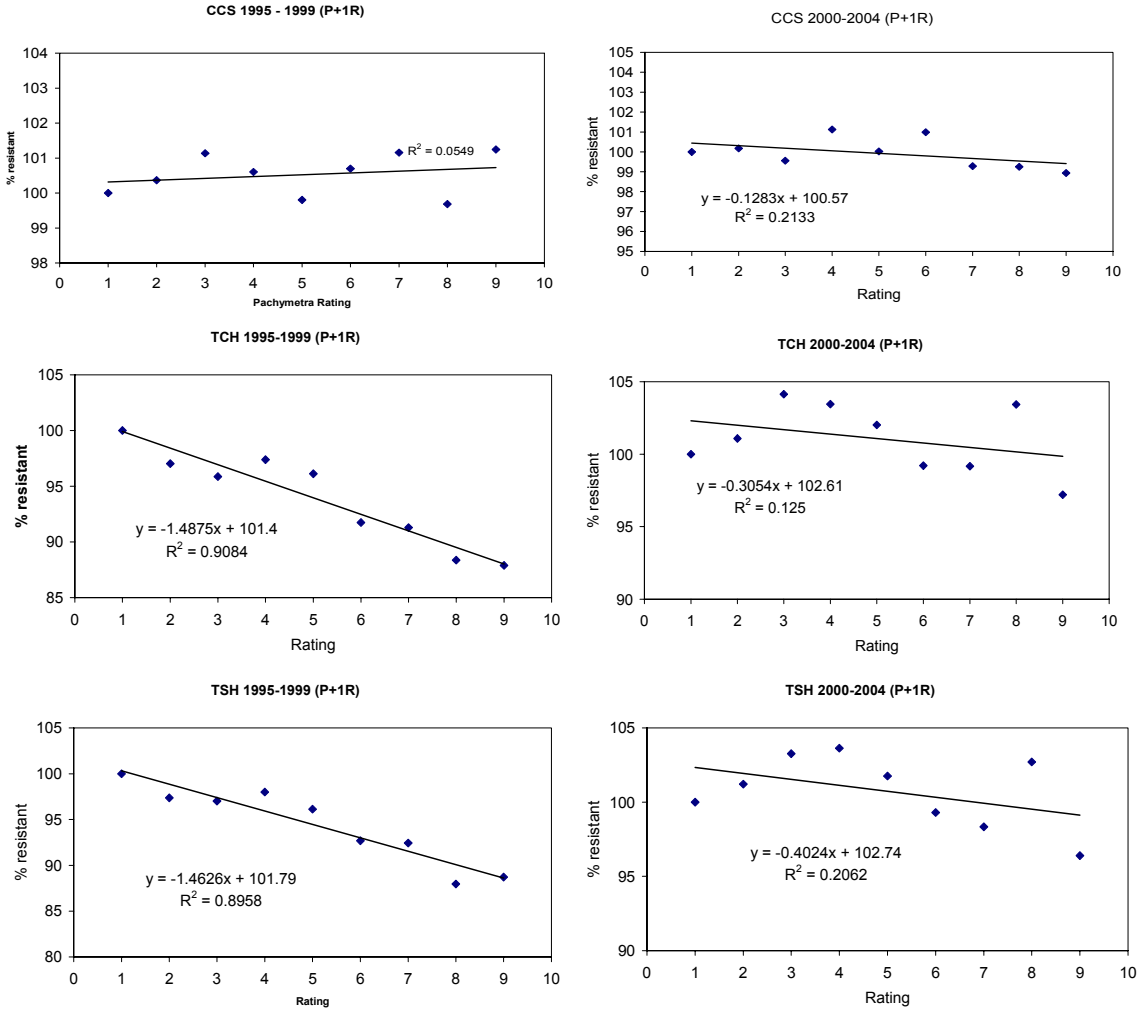


Figure 11 Five year average data for the relationship between Pachymetra root rot and yield parameters in northern selection trials (average of all sites, and plant and first ratoon crops). The 1995-1999 series averages are illustrated at left, and 2000-2004 series averages at right

These data suggest a strong relationship between Pachymetra root rot and tonnes cane per ha (biomass), and tonnes sugar per ha - especially for the mean data for the period 1995-1999, and for the 1999 series analyses. CCS was generally not related to Pachymetra

resistance though there were cases where CCS increased with susceptibility, while in other cases reduced CCS was associated with susceptibility.

The maximum yield losses associated with Pachymetra root rot resistance at each FAT trial location in the northern district are illustrated in Table 9. These data show very large losses maybe associated with susceptibility to Pachymetra root rot in individual trials.

Table 9 Maximum yield losses associated with Pachymetra root rot in each of the northern FAT series trials (1995-2004 data). Details of the series and crop class are provided for the maximum yield loss at each location (Tch = tonnes cane/ha; Tsh = tonnes sugar/ha)

Location	**Babinda**		**Mulgrave 1**		**Mulgrave 2**		**Mourilyan 1**		**Mourilyan 2**		**Tully**	
Yield parameter	Tch	Tsh	Tch	Tsh	Tch	Tsh	Tch	Tsh	Tch	Tsh	Tch	Tsh
% loss	41.2	40.6	49.8	48.4	47.2	43.1	36.8	41.4	24.3	24.4	49.4	46.3
Series	1998		2000		1997		1997		1996		1997	
Crop class	2R		2R		1R		1R		2R		2R	

The percent yield loss associated with Pachymetra root rot in northern trials between 1995-2004 (average of plant and first ratoon data) is illustrated in Figure 12 (tonnes cane/ha) and Figure 13 (tonnes sugar/ha). Yield losses for each trial series (labelled according to year of planting) in this period are compared to Tully rainfall recordings in Figure 14, and the data are regressed to assess the association between rainfall and % yield (tonnes cane) losses in Figure 15. The resistance index (RI) of clones in each trial series is presented in Figure 16.

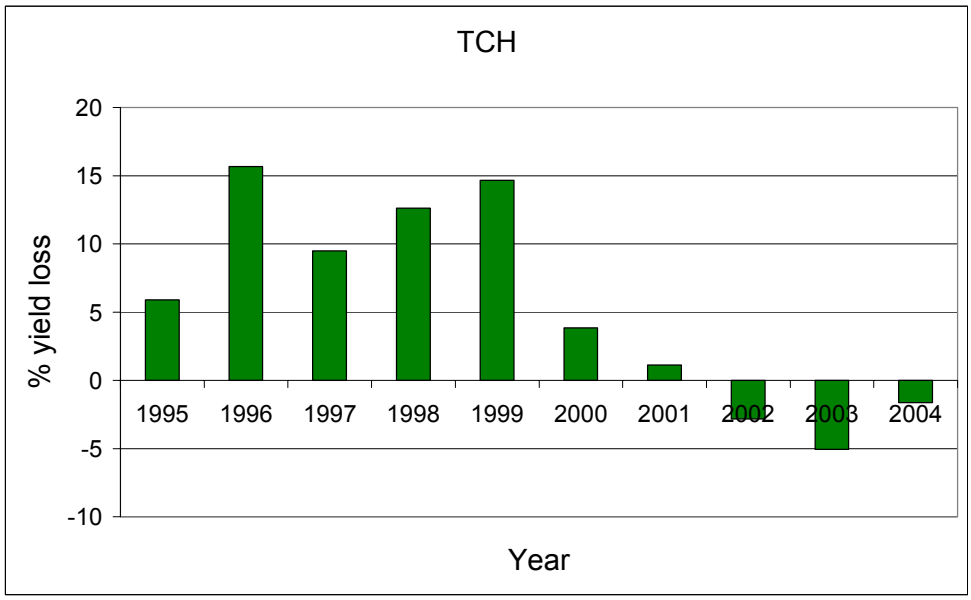


Figure 12 Percent yield loss associated with Pachymetra root rot for tonnes cane/ha in each series (average of plant and first ratoon data) from 1995-2004

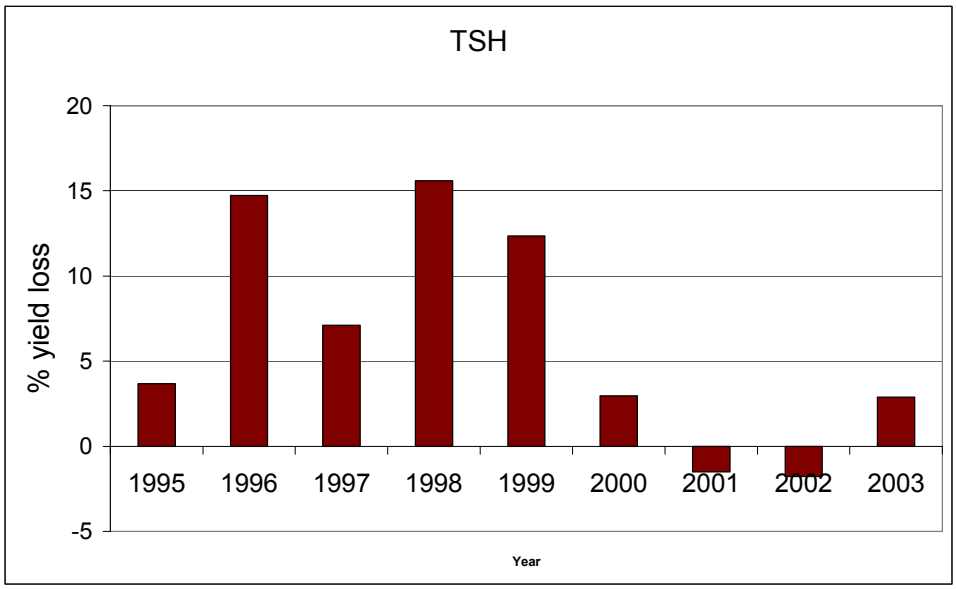


Figure 13 **Percent yield loss associated with Pachymetra root rot for tonnes sugar/ha in each series (average of plant and first ratoon data) from 1995-2003**

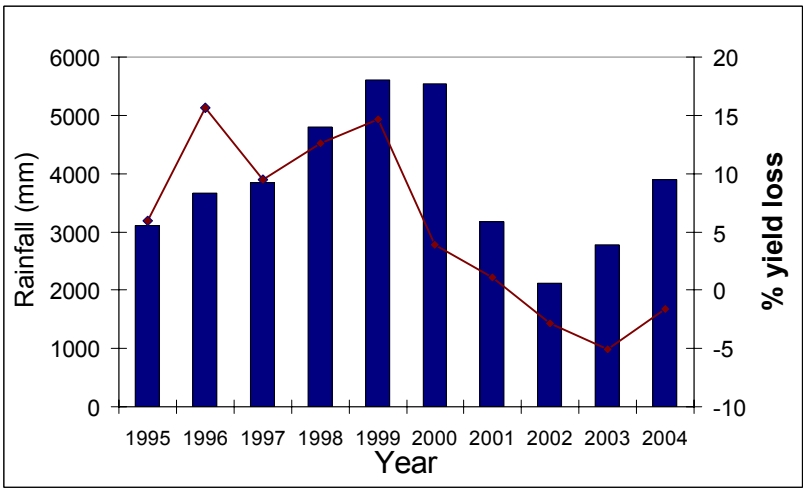


Figure 14 **The relationship between % yield losses associated with Pachymetra root rot (line) and rainfall (bars) at one site in northern Queensland (Tully)**

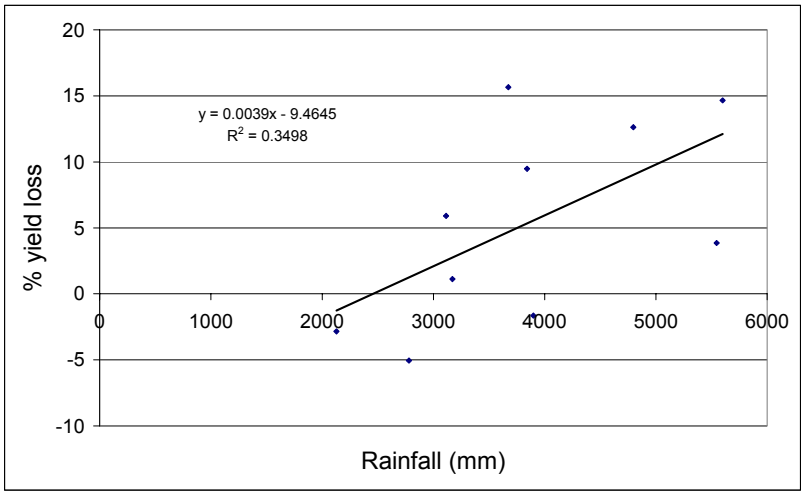


Figure 15 The regression relationship between rainfall in Tully and the average yield losses associated with Pachymetra root rot in northern Queensland selection trials in the 1995-2004 period

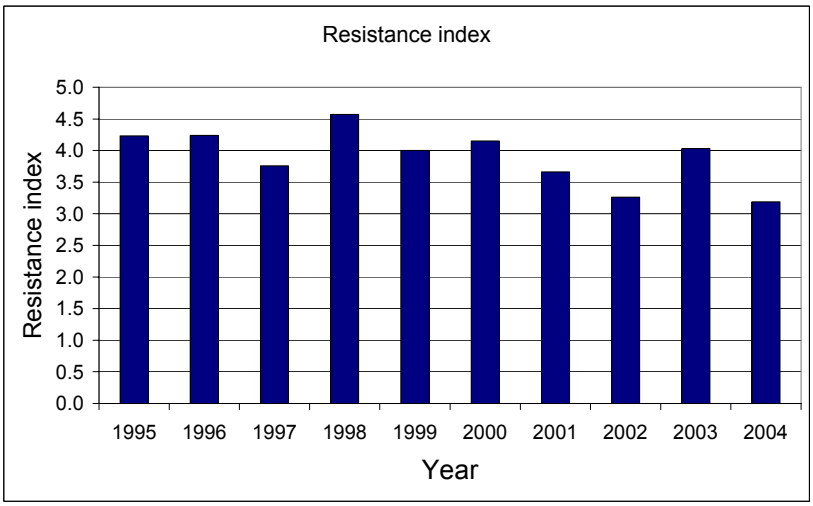


Figure 16 The Pachymetra resistance index (RI) of the clones included in each FAT series in northern Queensland (1995-2004). RI refers to the average resistance of clones within that population (clones in an individual series)

Of interest are the analyses conducted using Tableland selection trial data. These show no, or a poor, relationship between the Pachymetra resistance of clones and yield parameters. Very little Pachymetra root rot has been observed in Tableland cane fields. The analyses for the 1999 (2003?) series trials are outlined in Figure 17.

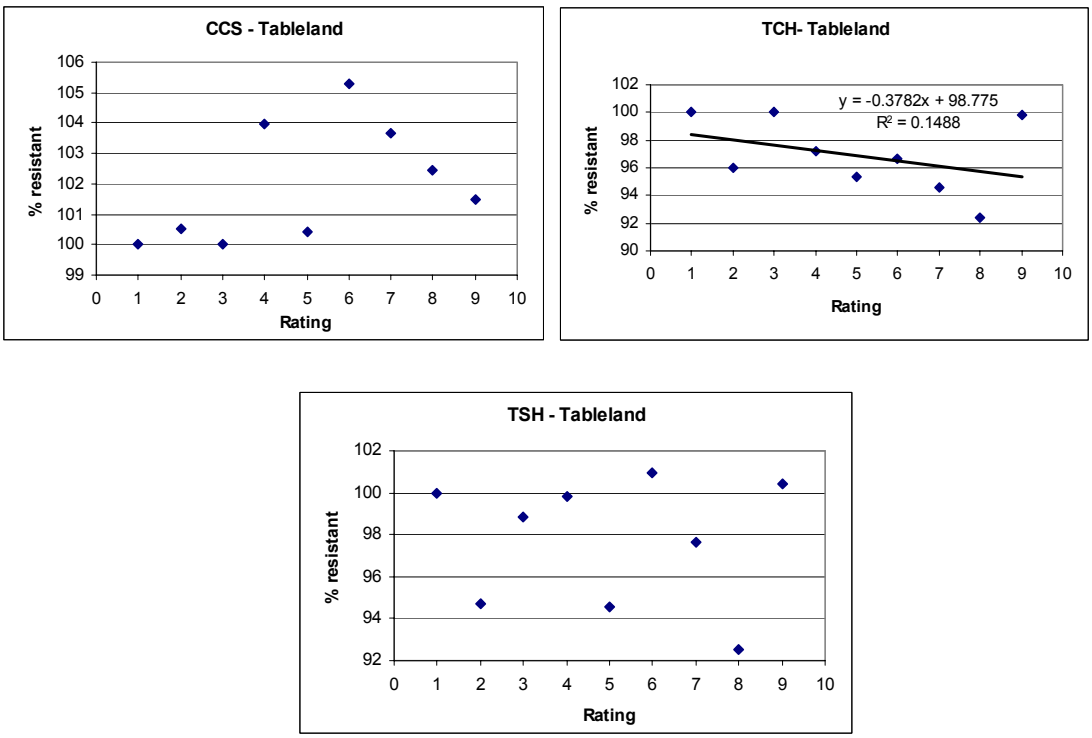


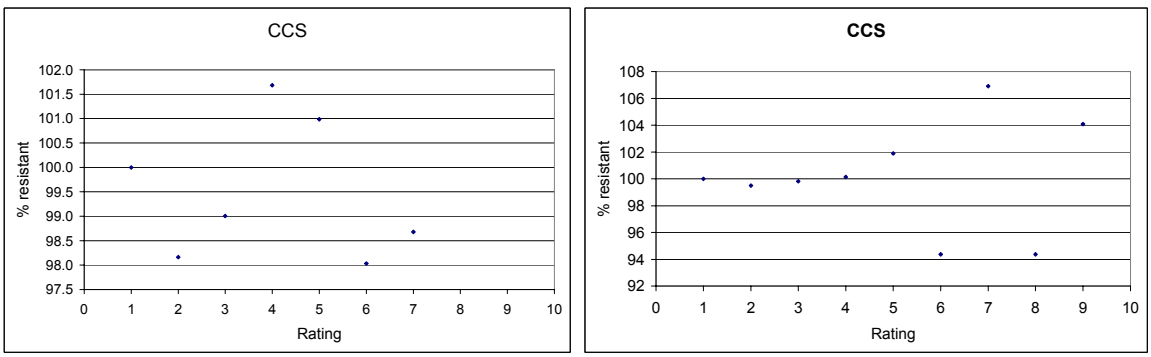
Figure 17 The relationship between Pachymetra root rot resistance and yield parameters in Tableland trials in the 1999-planted series. Pachymetra root rot has only been detected on the Atherton Tablelands at one site; at that site inoculum densities were very low

4.4.2 Central district trials

4.4.2.1 Resistance ratings

In central district analyses, there was an inconsistent relationship between spore-based resistance ratings and those arising from glasshouse resistance screening. In some years, the correlation was good while in 2000 series trials, the relationship was poor (data not shown).

Yield analyses:
In the 2003 series trial analysed, there were strong relationships between tonnes cane/ha and tonnes sugar/ha and resistance ratings obtained either from spore counts or glasshouse resistance screening. These are illustrated in Figure 18 and Table 10.



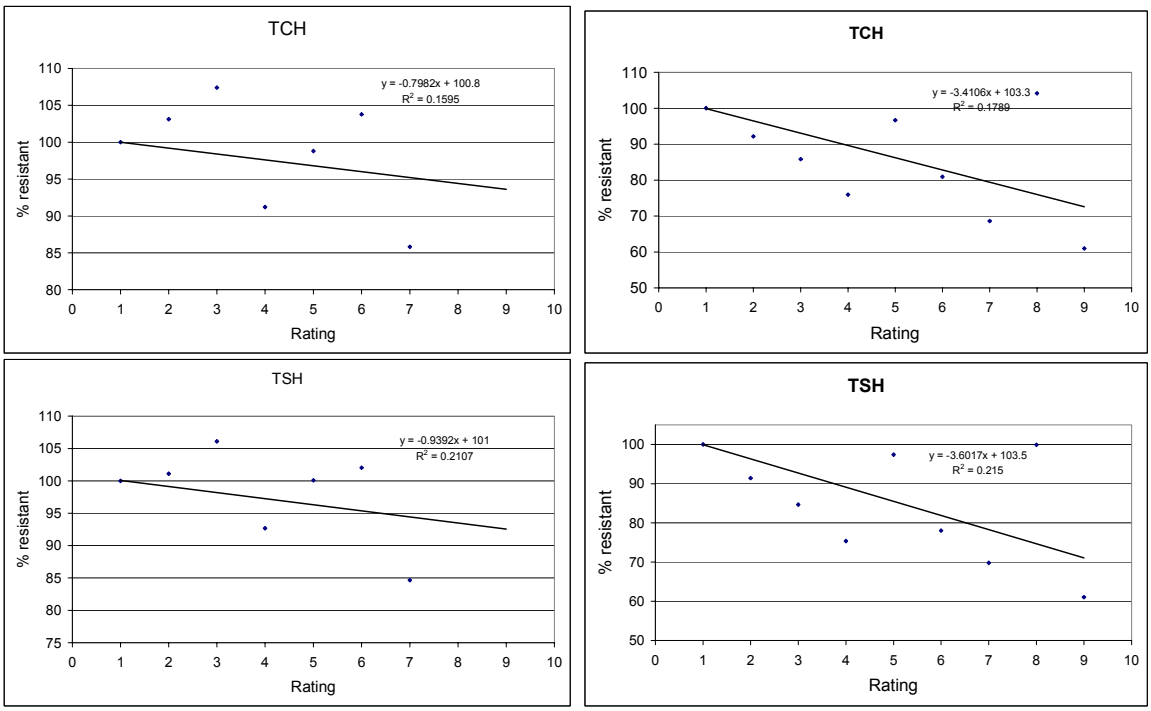


Figure 18 Average yield data (CCS, tonnes cane and tonnes sugar) for a 2003 series central district trial (Racecourse). Yield data using glasshouse resistance ratings (left) were compared with yield loss data based on resistance ratings originating from spore counts (right); limited glasshouse ratings meant less data are included in the analyses presented at left

Table 10 Calculated percent Pachymetra root rot-associated yield losses for susceptible clones (compared to resistant clones) in a plant improvement selection trial in Central Queensland, 2003 series. Data refer to plant crop results and have been analysed using spore count-derived vs. glasshouse resistance ratings

Yield parameter Rating method	CCS	Tonnes cane/ha (% loss)	Tonnes sugar/ha (% loss)
Spore ratings	ns	21.0	22.2
Glasshouse ratings	ns	19.2	13.4

4.5 Discussion

There seemed to be a clear association between Pachymetra resistance and yield parameters in breeding selection trials in the northern district, especially in some years and locations. It is generally accepted that Pachymetra root rot spore populations in the northern area are high (Magarey et al, 2004) and that some natural selection for disease resistance has been occurring in plant improvement selection trials over the last 20 years. The data confirm that the disease is of importance in selecting high yielding varieties and that varietal resistance is important for minimising disease-associated yield losses.

The absence of a relationship between Pachymetra root rot resistance and yield parameters in a Tableland trial confirms that the there is no inherent relationship between clonal resistance to Pachymetra and yielding ability.

Of concern was the variation in the data between the 1995-1999 and those post-2000. When further investigations were made, it was found that selection trial procedures changed for northern trials in the year 2000. In that year, sub-stations were adopted for the staging of selection trials. Instead of using 'new' commercial fields each time for selection trials, the same farmer's field was used for all trials after 2000 (four sub-stations per region). Trials were planted in rotation each year into the same field; new trials were planted onto 'old trial sites' after a short fallow of 6-8 months. Previous research has shown that substantial differences in Pachymetra inoculum densities will exist in plots in these fields when new trials are planted; this variation no doubt exerts a significant yield effect on clones in each new trial. Such yield effects will disrupt the relationship between clone resistance and yield and could lead to the poor relationships seen in this study.

Not only will this practice affect disease yield loss relationships, but will also add to the 'noise' associated with selecting clones based on yield data. Some clones of intermediate resistance may not be selected due to excessive yield loss associated with high initial Pachymetra inoculum densities in plots; other intermediate clones may be selected based on superior yield characteristics because they were planted in plots with unusually low Pachymetra inoculum densities - these being associated with resistant clones in the previous trial. These effects should be taken into consideration when revising plant breeding trial procedures.

It should also be borne in mind that climatic variation may also have influenced the strength of the resistance-yield relationship. Rainfall in particular showed some relationship with yield losses - though further study will be needed to distinguish causation. Distinguishing the influence of each factor was not possible here - though it is likely the previous clone effect will have been very significant.

Of interest were the resistance index (RI) values for Pachymetra root rot for clones in northern FAT trials. The data suggest that although RI values decreased slightly from 1995 to 2004, the differences were not great and that these would not account for the reduced yield losses subsequent to 2000.

The lack of relationship between Pachymetra root rot spore counts and glasshouse ratings in some trials hindered obtaining details of the effect of Pachymetra root rot in central district trials. However, data from the 2003 series trials suggests losses of around 20% in both tonnes cane/ha and tonnes sugar/ha. These are significant losses. It should be noted that FAT trials in the central district are not planted on sub-stations but in previously 'un-used' commercial fields. Other research has addressed the relationship between

Pachymetra root rot and yield in the Mackay district (Magarey et al, 2003). In this research, varieties of differing resistance to Pachymetra were planted in a field where there was a distinct difference in Pachymetra inoculum density (created through the previous cropping of a resistant and susceptible variety in each half of the field). A range of varieties varying in resistance were then planted over the inoculum boundary. Harvest of the plant crop provided Pachymetra associated yield loss data and information on the effect of resistance of these losses. These data are presented below in Figure 19.

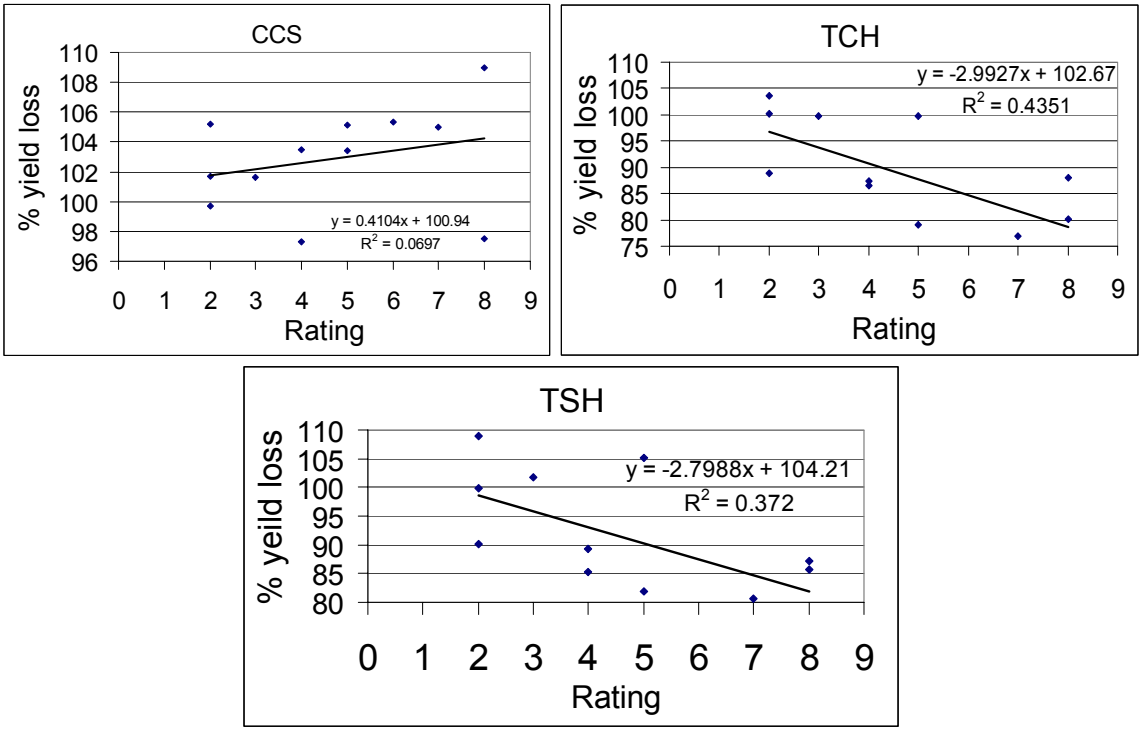


Figure 19 The relationship between Pachymetra root rot resistance and yield losses in an experiment conducted in Mackay where yield of varieties varying in resistance were recorded at a single site influenced by high and low inoculum densities

Interestingly, losses attributed to the disease in this experiment were similar to the average yield loss data from the 2003 series central district trials. The major effect was exerted through biomass reduction, while CCS was largely unaffected by Pachymetra root rot. Other yield loss research (Magarey, 1994) provided a similar result - high biomass losses but little effect of Pachymetra on sugar content.

These data collected using different methods are consistent and confirm the importance of Pachymetra roo rot on sugarcane yield in both northern and central Queensland.

5.0 YELLOW SPOT

5.1 Introduction

Yellow spot is of major concern principally in the northern (wet tropical coast) region. Some wetter parts of the Herbert district are also regularly affected, but not the drier areas

in that region. The disease is of principal concern in the higher rainfall areas and is definitely worse in the years with highest rainfalls. In previous years (late 1960s-early 1970s) some varieties were not released for commercial production because of their susceptibility to yellow spot. Data analyses were limited to the northern district because of the limited distribution of the disease.

5.2 Method

5.2.1 Resistance screening

In the series examined, resistance screening was based on the assessment of leaf area affected by the disease in clones within one plant improvement trial (for each series / year). A trial was selected where the plots of individual clones could be accessed. In some trials, high disease levels were present but lodged plots prevented entry to gain data on disease incidence, and hence resistance data. The method for assessing resistance relied on the identification of a leaf at the same relative position (for instance, the seventh down from the spindle leaf) in the canopy in each clone. Four leaves in the same relative position in each of four separate stalks were selected in each plot. The method has been described in detail elsewhere (Magarey et al, 2002); this ensured a good comparative assessment between all clones. Two assessors each visually assessed the percent leaf area affected by yellow spot; all assessments by each assessor for each of the four leaves was recorded and used for application of resistance ratings. These data were collected when yellow spot was approaching the peak of disease occurrence; this was usually toward the end of the wet season in the April-May period.

Mean values for percent leaf area affected were then calculated for each clone and clones with the lowest and highest disease levels identified. After appropriate arcsin transformation of the data, a 1 rating was applied to the clone with the lowest disease level and a 9 rating to the clone with the highest disease level. Resistance ratings for all other clones were applied on the basis of the equation of the line relating these two ratings / disease levels.

5.3 Plant improvement series investigated

Table 11 provides information on the series (based on year) analysed for the relationship between yellow spot and yield.

Table 11 Plant improvement series trials analysed for the relationship between yellow spot resistance and yield

District Series / Year	Northern
1999	+
2000	-
2001	-
2002	-
2003	+

5.4 Results

The relationship between yellow spot resistance and yield parameters was not as consistent as the effect of Pachymetra root rot in the northern series trials investigated.

Yellow spot was associated with yield losses in some trials, but the effect was inconsistent. The r-squared values (>0.20) for each trial regression between yield parameter and yellow spot resistance, plus details of the regression equations are presented for the same trials in the Appendix.

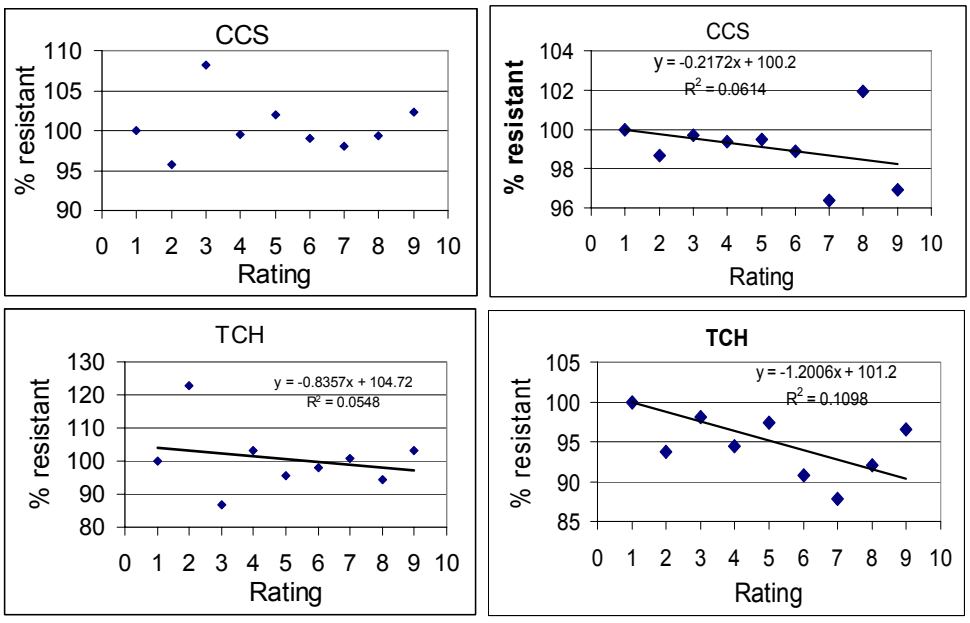
As for Pachymetra root rot, for each trial at a certain location within a district and series (year), the mean yield data for clones of the same resistance rating were expressed as a percentage of those for the 1-rated clones. This led to either 4 or 5 separate sets of information where the % yield loss for yield parameters could be related to each yield component (CCS, tonnes cane per ha, and tonnes sugar per ha). The mean percentage yield data were then calculated over all trials for the one series (year) in each district. Mean figures combining plant and first ratoon data were calculated. This provided an overall assessment of the relationship between yellow spot resistance and % yield loss. Maximum yield losses occurring in 9-rated clones in each trial where the r-squared was >0.20 are contained in the Appendix.

The average yield losses for each yield parameter (CCS, tonnes cane/ha and tonnes sugar/ha) over all trials in the 1999 and 2003 series are contained in Table 12.

Table 12 Calculated percent yellow spot-associated yield losses for susceptible clones (compared to resistant clones) in plant improvement selection trials: 1999 and 2003 series. Data are mean figures for plant and first ratoon crops

Yield parameter Trial series	Tonnes cane/ha	Tonnes sugar/ha
1999	13.9	9.8
2003	5.3	7.8

The regressions relating average loss figures for both the 1999 and 2003 series trials in northern Queensland are presented in Figures 20 and 21.



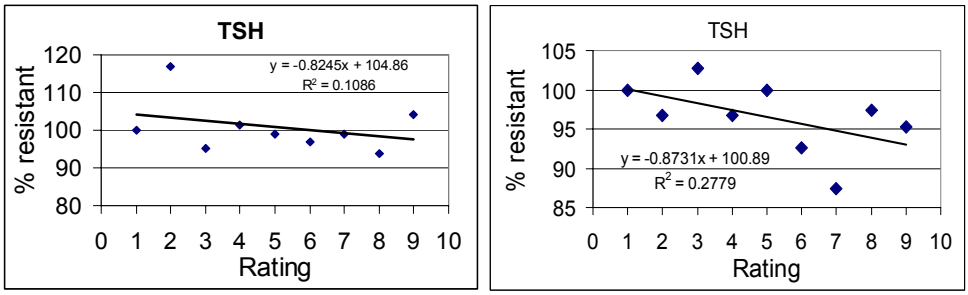


Figure 20 Summary yield data relating yellow spot resistance to yield parameters in 1999 (left) and 2003 (right) series trials in northern Queensland

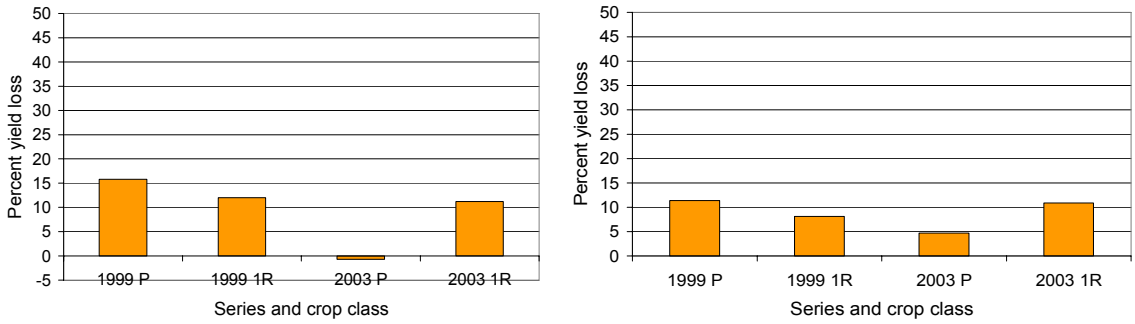


Figure 21 Magnitude of yellow spot-associated yield losses in northern trials by year and crop class (left TCH; right TSH)

These data suggest some relationship between yellow spot and CCS, tonnes cane per ha (biomass) and tonnes sugar per ha.

The maximum yield losses associated with yellow spot resistance at each FAT trial location in the northern district are illustrated in Table 13. These data show that large losses maybe associated with susceptibility to yellow spot in some locations - but the losses are not of the same magnitude in individual trials as for Pachymetra.

Location	**Babinda**		**Mulgrave 1**		**Mulgrave 2**		**Mourilyan 1**		**Mourilyan 2**		**Tully**	
Yield parameter	Tch	Tsh	Tch	Tsh	Tch	Tsh	Tch	Tsh	Tch	Tsh	Tch	Tsh
% loss	22.72	28.3	18.5	14.3	ns	ns	ns	ns	24.2	ns	27.0	22.8
Series	1999		1999						1999		1999	
Crop class	2R		P						P		1R	

Ns = not significant

Table 13 Maximum yield losses associated with yellow spot in each of the northern FAT series trials (1999 and 2003 data). Details of the series and crop class are provided for the maximum yield loss at each location (Tch = tonnes cane/ha; Tsh = tonnes sugar/ha)

5.5 Discussion

There seemed to be only some association between yellow spot resistance and yield parameters in breeding selection trials in the northern district. There were far fewer trials (compared to Pachymetra root rot) where yellow spot resistance explained more than 20% of the variation in the yield of clones. Even so when mean figures over all trials and series were calculated, the losses associated with yellow spot susceptibility were still quite large, and on a comparable scale as for Pachymetra root rot. Why the r squared values were much lower with yellow spot is unknown.

The relatively high level of susceptibility in stage 3 clones in breeding selection trials tends to suggest the lack of a strong selection pressure for yellow spot resistance in stage 1 and stage 2 trials. This could either be because the disease does not greatly affect sugarcane yield or that the disease occurs sporadically and inconsistent selection based on varying environmental conditions leads to a lack of elimination of susceptible clones. There are no doubt occasions when the disease does reduce yields and when commercial crop yields suffer. This can be seen in individual trial data in the 2003 series (for instance the Tully trial). This has been shown in other studies (Magarey et al, 2004; Egan, 1972). Further research with fungicides may shed more light on the importance of the disease and how this varies with weather /climate variation.

The highest yield losses associated with yellow spot were in the 1999 series trials, and this coincides with higher rainfall; the lowest Tully rainfall (1925-2006) on record for Tully was in 2002, while rainfalls in 1999 and 2000 were higher than average. Higher levels of disease were noticeable in this period in susceptible commercial crops.

The use of breeding selection trials for assessing the effects of yellow spot should be continued for a number of years. In this study only several years (series) of data were able to be analysed; by recording yield loss information over 5-10 years, the long-term effect of the disease would become much clearer.

6.0 ORANGE RUST

6.1 Introduction

Orange rust is of major concern right throughout the Queensland industry. Data presented earlier in this report suggest that some districts are more favourable to the disease than others. Observations in 2000 suggest that the disease may severely affect crops in each major cane growing region. Data analyses were mainly limited to the northern and central districts; application of resistance ratings for clones in Burdekin trials was attempted - but access to these crops when the disease was at moderate-severe levels was impossible due to the large crops and extensive lodging. The relationship between resistance and yield is likely to be similar to the outcomes from the analyses conducted with northern and central district data.

6.2 Method

6.2.1 Resistance screening

Resistance screening for orange rust was undertaken in exactly the same way as for yellow spot. As the two diseases occur at similar times of the year, some assessments (for instance in northern trials) occurred at the same time.

6.3 Plant improvement series investigated

Table 14 provides information of the series (based on year) assessed for the relationship between orange rust and yield.

Table 14 Plant improvement series trials analysed for the relationship between orange rust and yield

District Series / Year	Northern	Central
1999	+	+
2003	+	+

6.4 Results

6.4.1 Northern trials

The relationship between orange rust and yield parameters varied according to the series (year) and location. On some occasions the relationship was very strong while in other years the relationship was relatively weak. The r-squared values (>0.20) for each regression between yield parameter and clone, plus details of the regression equations for the same trials are presented in the Appendix.

As for Pachymetra root rot, for each trial at a certain location within a district and series (year), the mean yield data for clones of the same resistance rating were expressed as a percentage of those for the 1-rated clones. This led to either 4 or 5 separate sets of information where the % yield loss for yield parameters could be related to each yield

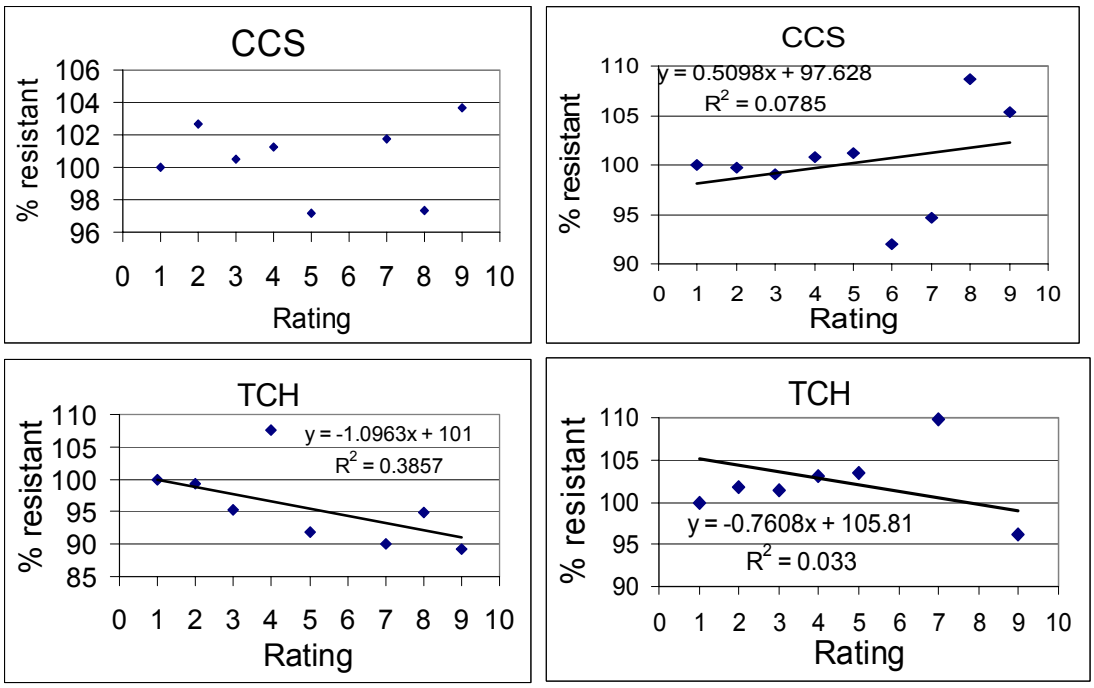
component (CCS, tonnes cane per ha, and tonnes sugar per ha). The mean percentage yield data were then calculated over all trials for the one series (year) in each district. Mean data combining plant and first ratoon data were calculated. This provided an overall assessment of the relationship between orange rust resistance and % yield loss.
The average yield losses for each yield parameter (CCS, tonnes cane/ha and tonnes sugar/ha) over all trials in the 1999 and 2003 series are contained in Table 15.

Table 15 Calculated percent orange rust-associated yield losses for susceptible clones (compared to resistant clones) in northern plant improvement selection trials: 1999 and 2003 series. Data are mean figures for plant and first ratoon crops

Yield parameter Trial series	Tonnes cane/ha	Tonnes sugar/ha
1999	11.4	12.3
2003	3.8	3.2

Maximum yield losses occurring in 9-rated clones in each trial where the r-squared was >0.20 are contained in the Appendix.

For orange rust, average loss figures were calculated for the 1999 and 2003 series trials in northern Queensland; graphs based on these data are presented in Figure 22. The magnitude of yield losses in each analysed series by plant and first ratoon crop are presented in Figure 23.



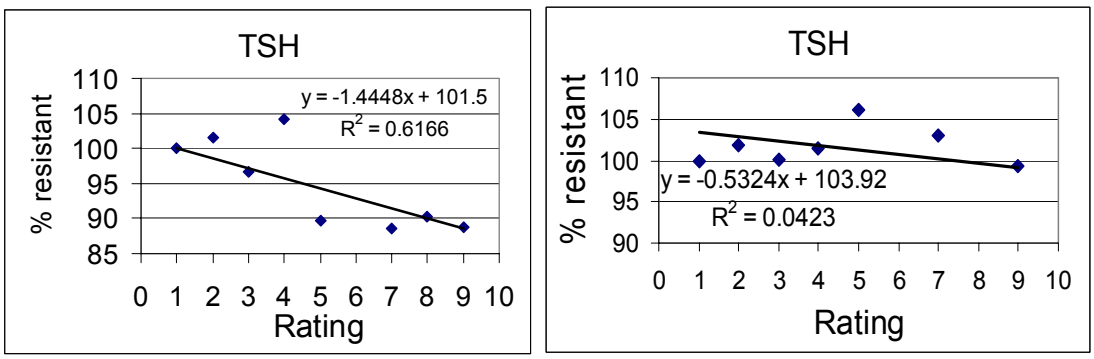


Figure 22 Summary yield data relating orange rust resistance to yield parameters in 1999 (left) and 2003 (right) series trials in northern Queensland

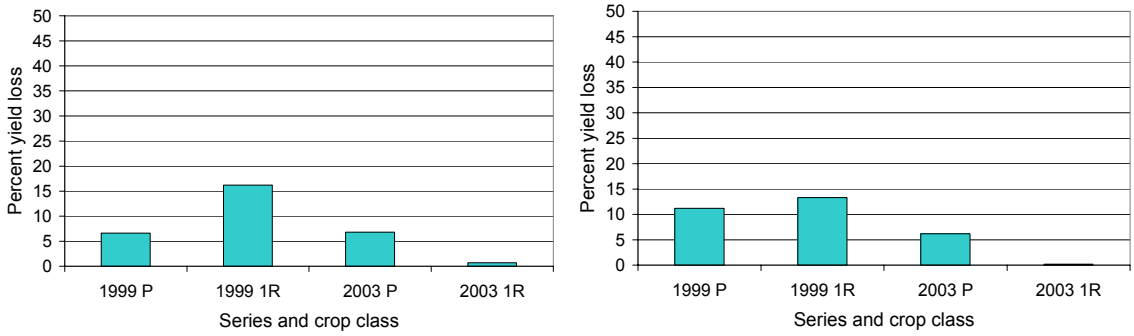


Figure 23 Magnitude of yield losses in northern trials by year and crop class (left TCH; right TSH)

The maximum yield losses associated with orange rust resistance at each FAT trial location in the northern district are illustrated in Table 16. These data show that significant losses may be associated with susceptibility to orange rust.

Table 16 Maximum yield losses associated with orange rust in each of the northern FAT series trials (1999 and 2003 data). Details of the series and crop class are provided for the maximum yield loss at each location (Tch = tonnes cane/ha; Tsh = tonnes sugar/ha)

Location	**Babinda**		**Mulgrave 1**		**Mulgrave 2**		**Mourilyan 1**		**Mourilyan 2**		**Tully**	
Yield parameter	Tch	Tsh	Tch	Tsh	Tch	Tsh	Tch	Tsh	Tch	Tsh	Tch	Tsh
% loss	25.4	22.4	34.2	33.3	ns	14.6	ns	ns	19.8	16.2	ns	ns
Series	1999		1999		1999				2003			
Crop class	1R		1R		P				1R			

6.4.2 Central trials

In the central district analyses, there was a very strong relationship between orange rust resistance and yield loss, particularly in plant crops of the 1999-planted series. The r-squared values (>0.20) for each regression between yield parameter and clone, plus details of the regression equations, are presented in the Appendix.

Maximum yield losses occurring in 9-rated clones in each trial where the r-squared was >0.20 are also contained in the Appendix. The relationship between yield loss and resistance for the 1999 and 2003 series are presented in Figures 24 and 25. Mean yield loss figures are included in Table 16.

Table 16 Calculated percent orange rust-associated yield losses for susceptible clones (compared to resistant clones) in plant improvement selection trials: 1999 and 2003 central district series. Data are mean figures for plant and first ratoon crops

Yield parameter Trial series	Tonnes cane/ha	Tonnes sugar/ha
1999	28.5	27.6
2003	-3.4	-7.6

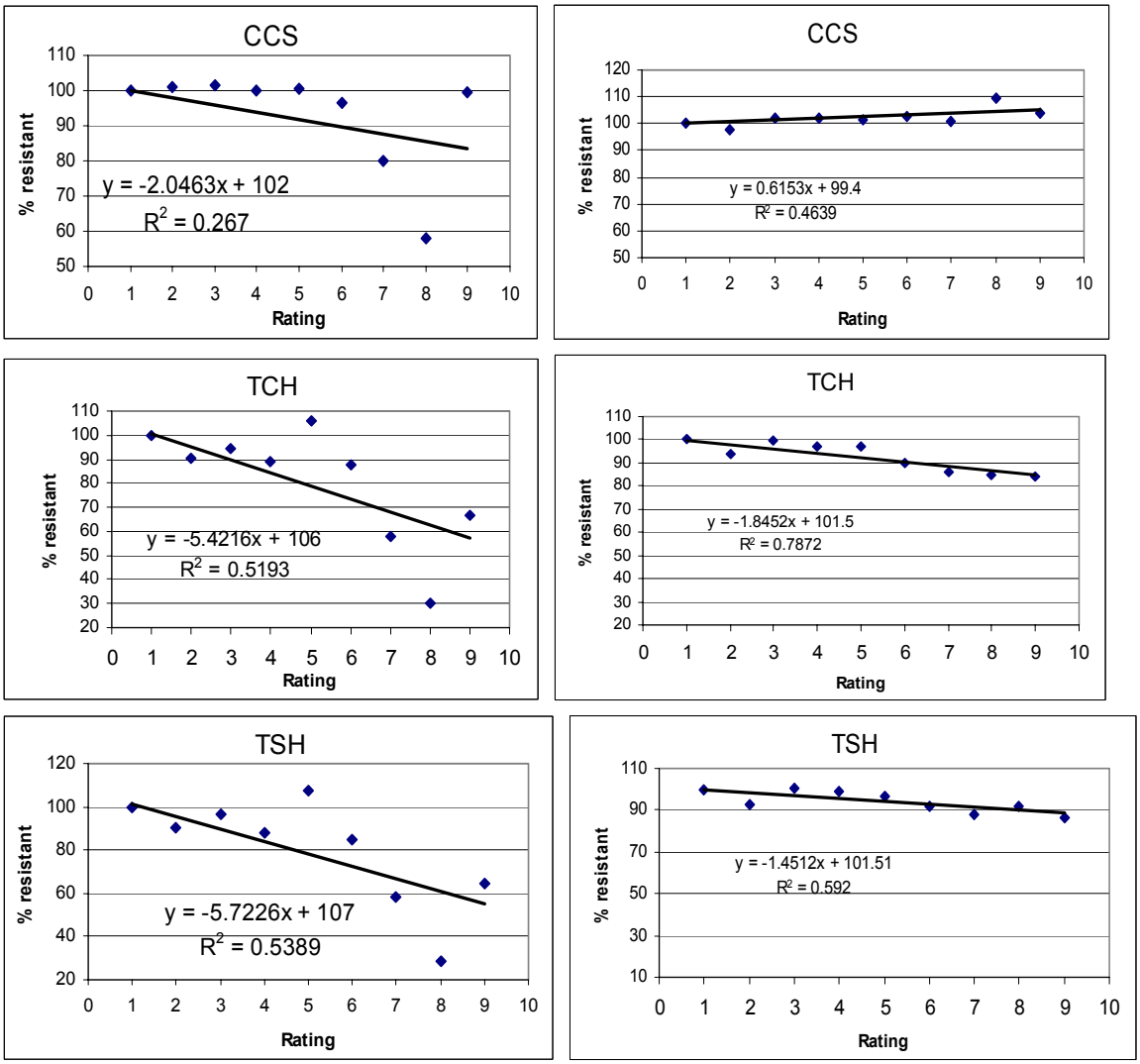


Figure 24 Summary yield data relating orange rust resistance to yield parameters in 1999 series plant crops (left) and first ratoon (right) in central Queensland

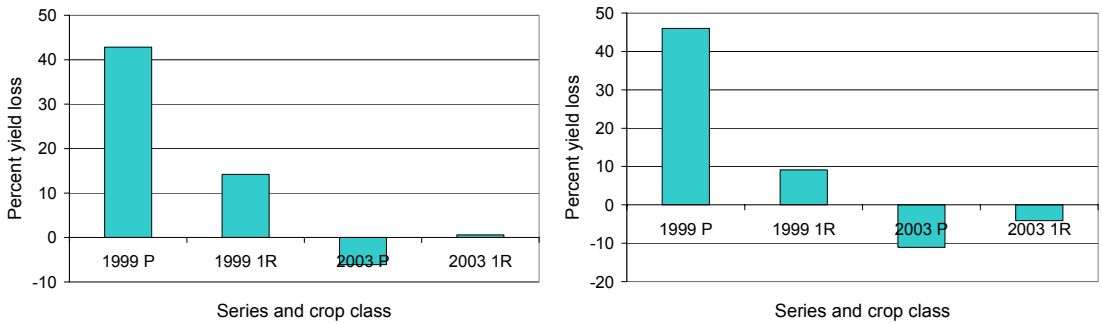


Figure 25 Magnitude of yield losses in central trials by year and crop class (left TCH; right TSH)

The maximum yield losses associated with orange rust resistance at each FAT trial location in the central district is illustrated in Table 17. These data show that very large losses maybe associated with susceptibility to orange rust under favourable conditions.

Table 17 Maximum yield losses associated with orange rust in each of the central FAT series trials (1999 and 2003 data). Details of the series and crop class are provided for the maximum yield loss at each location (tch = tonnes cane/ha; tsh = tonnes sugar/ha)

Location	**Proserpine**		**Farleigh**		**Marian**		**Pleystowe**		**Racecourse**		**Plane Creek**	
Yield parameter	tch	tsh	tch	tsh	tch	tsh	tch	tsh	tch	tsh	tch	tsh
% loss	33.8	32.1	58.8	59.6	29.9	31.0	58.9	58.8	11.6	31.1	25.7	29.9
Series	1999		1999		1999		1999		2003	1999	2003	
Crop class	P		P		P		P		1R	P	1R	

These data suggest a variable relationship between orange rust and yield parameters - with variation between years and districts.

6.5 Discussion

The data reported for both northern and central districts capture a precise moment in history, when orange rust first appeared in the industry. At this point in time, two factors were operating: i. there was a huge area planted to a very susceptible variety (Q124). This meant that the amount of disease present within districts was at a very high level. Magarey (2005) reported on industry assessments and an estimated 140,000ha of sugarcane was severely affected by orange rust during 2000. This is likely to be the highest single-year disease occurrence in the history of the Australian industry; ii. The recent introduction of this strain of orange rust meant that biological controls affecting the disease were not operating at a high level; disease incidence was therefore relatively unconstrained by bio-control agents. Recent observations in the Tully area suggest that some hyper-parasitic fungi are commonly found associated with crops affected by orange rust. In the central district, some insect larvae have also been found feeding on orange rust spores in recent years.

Together, both these factors suggest that in the first year of the disease epidemic, very high inoculum pressure was affecting susceptible varieties leading to high disease levels.

In the analyses undertaken here, yield effects match these observations. The greatest yield losses were in the 1999-planted trial series (both central and northern districts). In the central district, yield losses were as high as 40% in some trials in plant crops. This is far higher than what was observed for Pachymetra root rot and yellow spot and is likely to be related to the high levels of disease inoculum. Later analyses suggest the effect of orange rust was less, and this could have been associated with the lower inoculum production (associated with reduced areas of commercial crops of Q124) coupled with a build up in biological control mechanisms. It is possible that slightly reduced clonal resistance may be needed in the future (compared to the year 2000) because of this. Higher rainfalls in the 1999-2000 period could also have contributed to the higher yield losses associated with orange rust in the 1999 series trials. The accumulated hours of conducive conditions (see Figure 4) also suggest this period was very suitable for orange rust escalation.

The highest yield losses from orange rust were in the central district. As mentioned climatic variables strongly favoured the disease in that region and there was a very high infection pressure associated with the large area planted to the susceptible Q124. Losses in northern Queensland were less as there was a much smaller proportion of the susceptible Q124 planted in the north compared to the central district. The combination of less Q124 coupled with less conducive environmental conditions led to lower yield losses in 2003 series trials. The obvious potential for orange rust to cause significant yield losses is highlighted by the data presented. Ongoing losses however are likely to be small.

In future analyses of the kind adopted here, generally high levels of resistance in the Australian germplasm (especially after the year 2000) will pose problems for this type of analysis - there were too few clones of high disease susceptibility to accurately gauge the yield of susceptible clones.

7.0 BROWN RUST

7.1 Introduction

It was not possible to undertake brown rust analyses using the breeding selection trials. Attempts at assessing the resistance of clones in plant breeding selection trials failed due to the lack of disease in field plots when trials were inspected. It was especially difficult to undertake these inspections in trials remote from Tully, as the timing of travel to locations such as Mackay when brown rust was affecting crops was difficult to arrange. There were occasions when the disease was affecting commercial crops, but inspection of breeding selection trials failed to locate sufficient disease to assess clones for resistance.

Some research with brown rust yield losses and the effect of varietal resistance has been undertaken in the past (Taylor et al, 1985). This research was undertaken when the disease was first identified in Australia and centred on the use of fungicides to create high and low disease comparative plots. Two trials were undertaken, one in northern Queensland and one in southern Queensland. The southern Queensland data were reanalysed to relate varietal resistance with yield losses.

7.2 Method

The experimental method will not be outlined in detail here but briefly it involved a split plot experiment where fungicide (oxycarboxin) was applied to some plots and not to paired plots of the same variety. Brown rust was assessed at different intervals during the

course of the growing crop in each of five varieties that differed in resistance to the diseases. Yield (CCS, tonnes cane/ha and tonnes sugar/ha) was assessed in the mature crop.

In these analyses, the percent leaf area affected in the 7th leaf from the top of the stalk was used to assess the resistance of each of the varieties (Q110, Q87, Q108, Q90 and QS70-77) present in the experiment. The resistance of the clones was regressed against the percent yield loss (tonnes cane/ha; tonnes sugar/ha) to determine the relationship between resistance and yield losses. Because only a limited number of varieties were used in the regression, there is limited interpretation that should be applied to the relationship between resistance and yield.

7.3 Results

Figure 26 below illustrates the relationship between brown rust resistance and losses in tonnes cane/ha and tones sugar/ha. In both cases, losses were significant in the most susceptible variety (QS70-77).

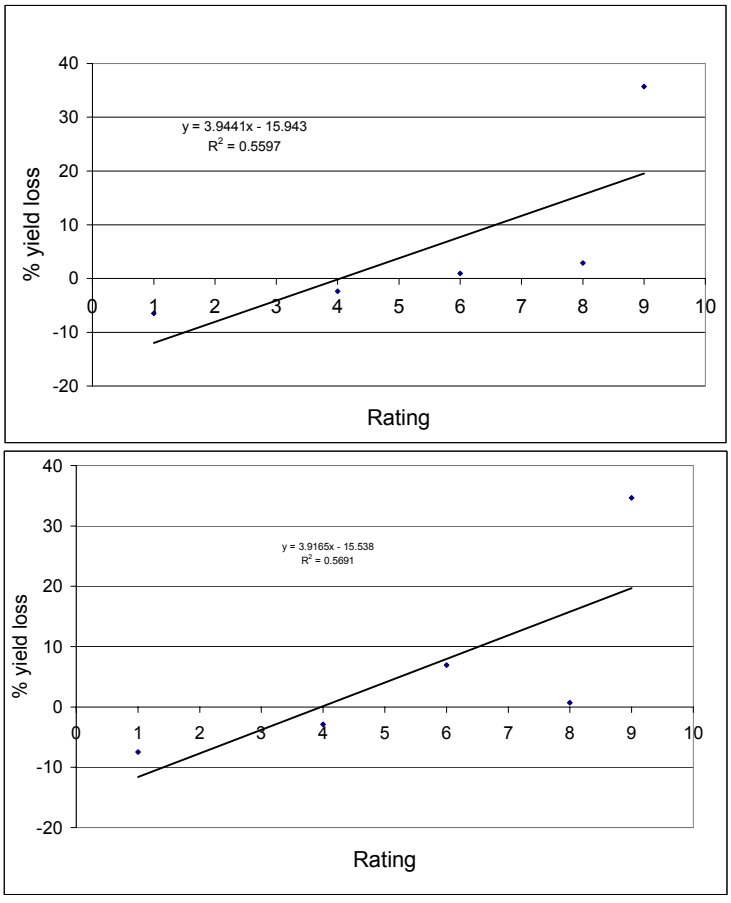


Figure 26 Brown rust associated yield losses in the Isis Mill area (1982-83) - tonnes cane/ha (top) and tonnes sugar/ha (bottom)

7.4 Discussion

Only limited interpretation should be placed on the analyses reported for brown rust as a very limited data set was used to investigate the yield loss x resistance relationship. These data do suggest a similar relationship to orange rust - roughly linear relationship with maximum losses around 30% (tonnes cane and tonnes sugar). It is obvious that further data capture is needed and further investigation of the relationship between resistance and yield.

Other evidence suggests that brown rust can exert a significant effect on sugarcane yield and that breeding for resistance is necessary in order to maximise commercial yields of sugarcane crops. Experience around the world has shown that the disease has made the commercial cropping of susceptible varieties uneconomic, and has necessitated the use of resistant or intermediate resistance varieties (Raid and Comstock, 2000).

8.0 AVERAGE CROP RESISTANCE AND DISEASE RESISTANCE PROFILES

Of interest in any discussion on yield effects of diseases is a consideration of the general resistance of commercial crops to those diseases and how the resistance of seedlings produced in the breeding program compares to expected disease-associated yield losses.

Magarey (2006) calculated the average resistance of crops in each region of Queensland. This was undertaken by obtaining data on the proportion of the crop produced in each area by each variety, using disease resistance ratings for each variety, and calculating a weighted mean resistance rating for all crops produced in the region based on the resistance data. Further details relating to these calculations are described by Magarey (2006). Profiles for the northern and central districts for all diseases for the 2004 crop are illustrated in Figure 27.

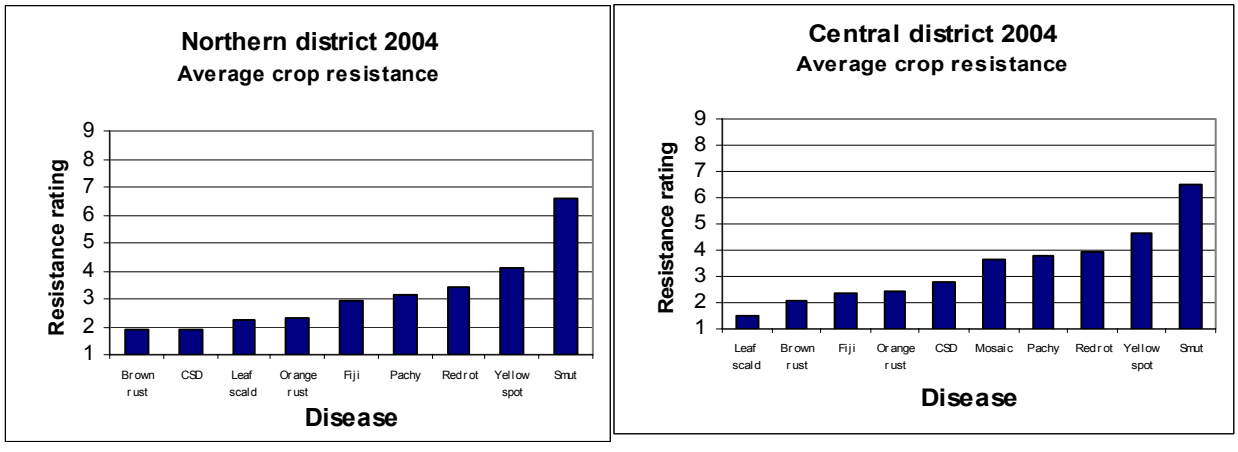
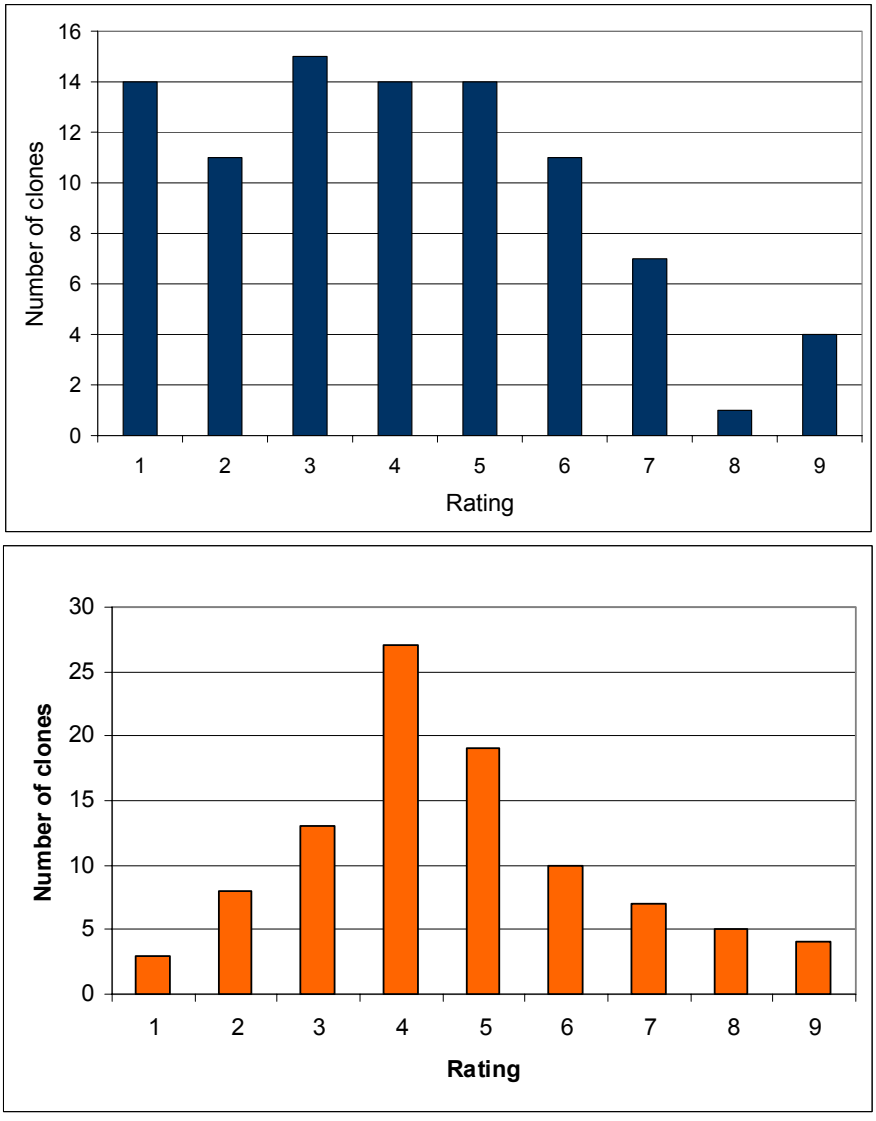


Figure 27 The average 2004 crop disease resistance to various endemic diseases in northern and central Queensland

These data illustrate the relatively high level of resistance present for orange rust, and the lower level of resistance to Pachymetra root rot and yellow spot (for the latter this is relevant for northern Queensland only).

Yield loss data obtained form plant improvement selection trials could be used to estimate the percentage yield losses occurring regularly in crops in each region, assuming losses in breeding trials were indicative of those suffered on average in commercials fields. For example if maximum losses for Pachymetra root rot with a 9 rated variety were 15%, then average crop resistance of 4 would suggest district losses are 4/9 x 15% = 6.7% annually. If the general resistance of the crop within a region rose, then losses would be reduced proportionally.

Also of interest are the resistance profiles of clones in plant improvement selection trials, as this provides a background understanding of what breeders have to select for future commercial varieties. If high levels of susceptible varieties are coming through the system, then there is little room to select for a high level of resistance. Data for Pachymetra root rot, yellow spot and orange rust for the 2003 series northern trials are included in Figure 28.



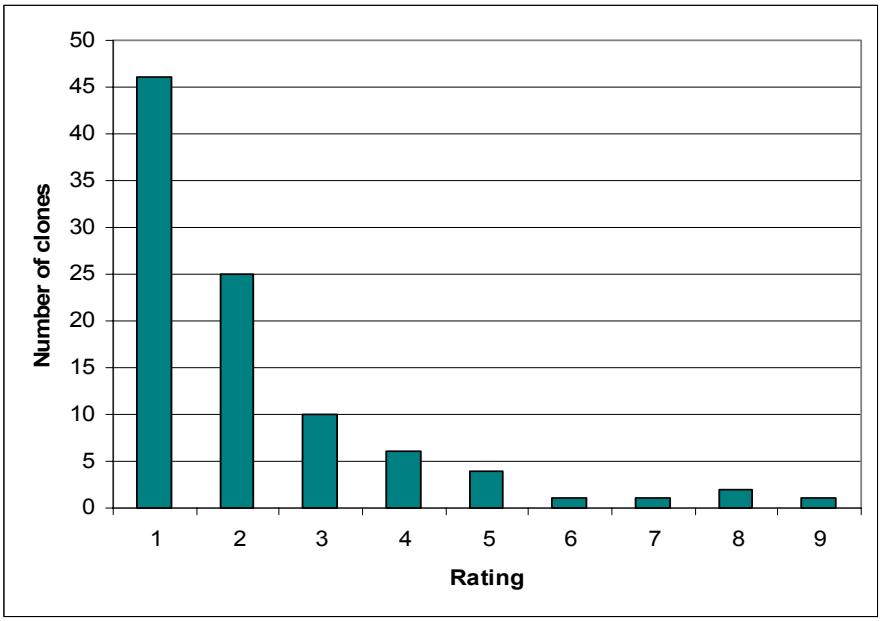


Figure 28 The resistance profiles of the same 2003 northern series clones to Pachymetra root rot (top), yellow spot (middle) and orange rust (bottom) illustrating the diversity in inherent resistance to the three diseases

The Resistance Index (RI) for each disease in the 2003 northern series is illustrated in Table 18. This again illustrates the higher level of resistance present in the germplasm to orange rust, and the lower level of resistance to yellow spot.

Table 18 The resistance index for each disease in the 1999 and 2003 series clones in northern (Pachymetra root rot / yellow spot / orange rust) and central (orange rust) FATs

Disease / Series	Pachymetra root rot	Yellow spot	Orange rust	
			Northern	*Central*
1999	4.0	5.5	2.1	3.1
2003	4.0	4.6	2.2	2.8

In plant improvement selection strategies, not only is the potential yield losses caused by each disease in a region an important consideration, but also the urgency to produce new commercial varieties with higher levels of resistance (if a disease is causing excessive yield losses in commercial crops) and the level of resistance present in seedling clones passing through the selection program. All the information could be gathered from the type of data presented in this report.

9.0 COMPARISON OF YIELD LOSSES CAUSED BY EACH DISEASE

The analysis of the same series of trials allows some comparison of the relative influence of each disease on yield. It is clear from overall mean yield loss figures that each disease reduced yield (tonnes cane/ha or tonnes sugar/ha) on average by between 10-15%. In individual trials, much greater losses were recorded for each disease - particularly orange

rust in plant crops in 1999 in the central district and Pachymetra root rot in northern trials in the 1995-1999 period. The strength of the relationship between Pachymetra root rot and yield loss was stronger than for the other diseases. Ad hoc observations suggest that natural selection for Pachymetra root rot resistance has been occurring in northern FAT selection trials over the last 20 years, and this supports the proposition that the disease is reducing yield consistently in these trials.

The lack of selection for yellow spot resistance tends to suggest the opposite - that the disease affects yield in a sporadic way - perhaps being more dependant on weather conditions, and therefore less consistently affecting yield. This would reduce natural selection in breeding trials. Higher RI values for yellow spot illustrate the lower level of resistance for yellow spot in the seedling population.

Of the two leaf diseases, orange rust - especially in the central district - caused the most severe average yield losses with yield reduced by up to 40%. However, in later trials the effect of orange rust and yellow spot was comparable and more minor in effect. Lower rainfall in the northern district may have contributed to reduced yield effects in this region, but there is also no doubt that lower levels of inoculum contributed. Increased bio-control is also likely to be a factor. Table 18 details the average yield losses (tonnes cane/ha and tonnes sugar/ha) for the 1999 and 2003 series trials for northern Queensland.

Table 18 Average yield losses associated with each disease in northern series trials in 1999 and 2003

Series (year)	1999			2003		
Crop class	Disease	Tonnes cane / ha	Tonnes sugar / ha	Disease	Tonnes cane / ha	Tonnes sugar / ha
Plant	Yellow spot	15.8	11.4	Yellow spot	-0.7	4.7
	Orange rust	6.6	11.2	Orange rust	6.8	6.2
First ratoon	Yellow spot	12.0	8.1	Yellow spot	11.2	10.9
	Orange rust	16.2	13.3	Orange rust	0.7	0.2
Mean (P+1R)	Yellow spot	13.9	9.8	Yellow spot	5.3	7.8
	Orange rust	11.4	12.3	Orange rust	3.8	3.2
	Pachymetra	14.5	11.9	Pachymetra	2.7	4.8

These are illustrated graphically in Figure 29 for the 1999 series trials.

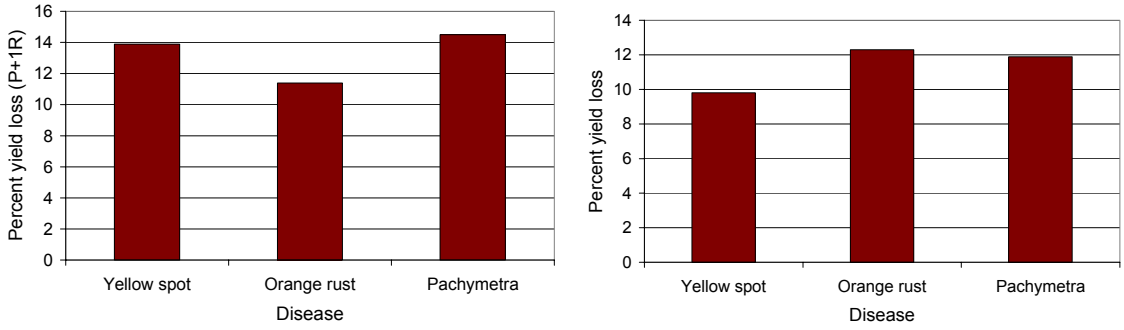


Figure 29 Magnitude of yield losses caused by the different diseases in northern trials (left TCH; right TSH). The results are mean figures for plant plus first ratoon crops in the 1999 series trials

10.0 GENERAL DISCUSSION

It is clear from these analyses that endemic diseases are significantly affecting the yield of clones in plant breeding selection trials. This suggests that incorporating sufficient resistance to these diseases in commercial varieties is important for maximising commercial yields. Of the three diseases, Pachymetra root rot has consistently reduced yields in FATs and is the most important on-going disease influence. However, the effect of orange rust in the central district in 2000-harvested plant crops was also very large - but losses were much lower in first ratoon crops of the same series.

The relationship between resistance and yield in all three diseases appeared similar; there was a linear relationship between losses and yield. In other research conducted with Pachymetra root rot in the central district (Magarey et al, 2003), losses in highly resistant varieties were negligible and it is likely negligible losses would have been associated with highly resistant clones with each disease in these analyses.

Climate also is likely to have had a significant influence on yield losses in these studies. Higher yield losses were seen with the leaf diseases in the 1999 series trials compared to the 2003 series - rainfall is likely to have influenced the favourability of the environment for these diseases. The higher inoculum levels associated with large areas of the susceptible Q124 would have contributed to higher orange rust losses in that year also.

The Pachymetra data suggest that plant breeders should consider revising, or at least giving further contemplation to the operation of breeding sub-stations. The interaction of residual Pachymetra inoculum with yield, is at best contributing ‘noise’ to the data and at worst leading to the selection of clones that will perform sub-optimally in the presence of the disease. Practical issues need to be combined with disease considerations in the design of sub-station sites.

Possible problems with this type of analysis include having a suitable range of resistance in clones in FAT trials. With orange rust, the high level of resistance present in clones meant that there tended to be few clones in the susceptible category. This decreased the reliability of yield values at this end of the scale in the regression analyses. For Pachymetra root rot and yellow spot this was generally of lesser significance. The lack of relationship between resistance and yield with Pachymetra root rot in Tableland trials illustrates that there was no inherent relationship between resistance to Pachymetra root rot and yield parameters.

11.0 CONCLUSION

- There were significant yield losses associated with endemic diseases in many FATs planted in northern and central Queensland
- Losses to Pachymetra root rot were the most consistent and were up to 40% in some locations in northern Queensland.
- Losses associated with orange rust were around 40% in central Queensland in the year 2000, but lower in subsequent years.
- Yellow spot caused significant losses in some years but was less consistent in its effects

- When mean data for all trials and all series were analysed it was found that each disease caused losses of between 10-15% (tonnes cane/ha and tonnes sugar/ha).
- Rainfall, inoculum pressure and bio-control effects are likely to have contributed to variation in disease-associated yield losses.
- It is recommended that further yield loss studies are undertaken over a longer period to establish the long term effect of these diseases, especially for the leaf diseases.
- Further monitoring of climatic conditions coupled with more extensive monitoring of leaf disease levels during the growing season will enable stronger conclusions to be drawn of the relationship between weather and yield losses.
- This work coupled with knowledge of the resistance of parents and clones will enable optimum levels of disease resistance to be incorporated into commercial varieties.
- FAT sub-station site procedures should be reviewed to take into account residual Pachymetra inoculum influences on clone yield.

12.0 FUTURE WORK

- There are further analyses that could be undertaken with these data, including the following: -
  - Determining the mean yield losses occurring for the endemic diseases at each FAT location within a district
  - Comparing average losses at each site with weather conditions (such as rainfall) to further determine the relationship between rainfall and yield losses.
- Additional work could include assessing soil moisture conditions (as opposed to simple rainfall measurements) to investigate the linkage between soil moisture and severity of Pachymetra root rot.
- Recording of temperature, relative humidity, leaf wetness and rainfall at each FAT site and to relate these conditions to the severity of leaf diseases at each site. This will provide much further information on the requirements for leaf diseases.
- The above information, relating yield losses to disease resistance, if recorded over an extended period will allow predictions on what percentage of years in each area will be prone to significant yield losses from that disease. This in turn will provide further guidance to plant breeders on what level of resistance to incorporate into commercial varieties.
- Further analyse central district Pachymetra FAT data for individual trial losses and the influence of regional conditions on these losses.

13.0 ACKNOWLEDGEMENTS

My sincere thanks go to Judi Bull and other Tully Pathology Technicians for their assistance in field and office activities. I also thank both SRDC and BSES for the financial

support of the work. I thank Dr Xianming Wei for patience in anticipation of the outcomes from this research.

14.0 REFERENCES

1. Croft, B.J. 1989. A technique for screening sugarcane cultivars for resistance to Pachymetra root rot. Plant Disease 73: 651-654.

2. Egan, B.T. 1972. The 1971 yellow spot epidemic in north Queensland. Queensland Society of Sugar Cane Technologists, 39: 201-207.

3. Magarey, R.C. 1989. Quantitative assay of *Pachymetra chaunorhiza,* a root pathogen of sugarcane in Australia. Phytopathology 79:1302-1305.

4. Magarey, R.C. 1991. The effect of varietal resistance on the epidemiology of Pachymetra root rot. Proceedings of the Australian Society of Sugar Cane Technologists, 13:95-102.

5. Magarey, R.C. 1994. Effect of Pachymetra root rot on sugarcane yield. Plant Disease 78: 475-477.

6. Magarey, R.C. 2005. The incidence of sugarcane diseases in Queensland. Proceedings of the Australian Society of Sugar Cane Technologists, 27: 252-265.

7. Magarey, R.C. and Bull, J.I. 2001. The effect of Pachymetra root rot on sugarcane yield in plant breeding trials. In: Conference handbook, 13th Biennial Conference, Australasian Plant Pathology Society, p69, Cairns, Australia.

8. Magarey, R.C., Bull, J.I., Camilleri, J.R., Cripps, L., Staier, T.N. and Magnanini, A.J. (2004). Pachymetra root rot severity in Queensland canefields assessed through data from the Tully soil assay laboratory. Proceedings of the Australian Society of Sugar Cane Technologists, 26 (CD-ROM) ..p

9. Magarey, R.C. and Bull, J.I., Neilsen, W.A., Camilleri, J.R. and Magnanini, A.J. 2004. Relating cultivar resistance to sugarcane yield using breeding trial analyses – orange rust and yellow spot. Australian Journal of Experimental Agriculture 44: 1057-1064.

10. Magarey, R.C. and Bull, J.I. 2003. Relating cultivar Pachymetra root rot resistance to sugarcane yield using breeding selection trial analyses. Australian Journal of Experimental Agriculture. 43: 617-622.

11 Magarey, R.C. and Croft, B.J. 1998. A review of yield losses caused by Australian and exotic diseases. Proceedings of the Australian Society of Sugar Cane Technologists, 20:76-84.

12. Magarey, R.C., Bull, J.I., Neilsen, W.A. and Magnanini, A.J. 2002. The use of breeding trials to estimate disease-induced yield losses and to refine selection strategies. Proceedings of the Australian Society of Sugarcane Technologists, 24.

13. Magarey, R.C. and Soper, S.A. 1992. Involvement of Pachymetra root rot in stool tipping in varieties Q117 and Q124 in north Queensland. Proceedings of the Australian Society of Sugar Cane Technologists, 14: 68-74.

14. Magarey, R.C., Sarich, C.E., Turner, J.D. and Bull, J.I. 2003. Influence of Pachymetra root rot resistance on yield losses in central Queensland. Proceedings of the Australian Society of Sugarcane Technologists, 25.

15 Raid, R. N. and Comstock, J.C. 2000. Common rust. In: A Guide to Sugarcane Diseases. Eds. P. Rott, R.A. Bailey, J.C. Comstock, B.J. Croft, and A.S. Saumtally. CIRAD and ISSCT, pp 85-89.

16. Staier, T.N., Magarey, R.C. and Neilsen, W.A. 2004. Meteorological data collection, analysis and sugarcane disease forecasting for orange rust. Proceedings of the Australian Society of Sugarcane Technologists, 26.

17. Taylor, P.W.J., Croft, B.J. and Ryan, C.C. 1985. Effect of rust on cane yield. BSES Project Report No. 335.

15.0 APPENDICES

Appendix 1 – Yield loss calculations for Pachymetra root rot using average northern trial data for 1995-1999 and 2000-2004

5 Year Series	Trial											
	All Northern TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1995-1999	P	100.65	-1.144	100.65	99.506	89.21	**10.3**	101.57	-1.0979	101.57	100.4721	90.591	**9.8**
	1R	102.15	-1.8309	102.15	100.3191	83.841	**16.4**	102.01	-1.8274	102.01	100.1826	83.736	**16.4**
	P+1R	101.4	-1.4875	101.4	99.9125	86.525	**13.4**	101.79	-1.4626	101.79	100.3274	87.164	**13.1**
2000-2004	P	101.85	-0.3552	101.85	101.4948	98.298	**3.1**	102	-0.4927	102	101.5073	97.073	**4.4**
	1R	103.36	-0.2557	103.36	103.1043	100.803	**2.2**	103.49	-0.3122	103.49	103.1778	100.368	**2.7**
	P+1R	102.61	-0.3054	102.61	102.3046	99.556	**2.7**	102.74	-0.4024	102.74	102.3376	98.716	**3.5**

Appendix 2 - R-squared values for the regressions between Pachymetra resistance and yield component for each FAT in northern Queensland (1995-2004 series)

Year	Trial	Babinda			Mulgrave 1			Mulgrave 2			Mourilyan 1		
		CCS	TCH	TSH	CCS	TCH	TSH	CCS	TCH	TSH	CCS	TCH	TSH
1995	P	0.26									0.45		
	1R								-0.37	-0.39		-0.51	-0.47
	2R												
1996	P					-0.84	-0.80		-0.27	-0.19		-0.42	-0.27
	1R		-0.24	-0.39	-0.22	-0.55	-0.66		-0.39	-0.35	0.24	-0.53	-0.39
	2R					-0.52	-0.45	0.41	-0.71	-0.62		-0.66	-0.71
1997	P		-0.48	-0.47					-0.51	-0.41	-0.62		
	1R	0.44	-0.49	-0.38		-0.52	-0.42	0.44	-0.52	-0.46		-0.92	-0.90
	2R				-0.62	-0.21	-0.33	0.25	-0.20		-0.30	-0.40	-0.50
1998	P	-0.46			-0.28	-0.26	-0.42		-0.55	-0.60		-0.73	-0.74
	1R	-0.32	-0.78	-0.80	-0.40	-0.58	-0.81		-0.35	-0.28		-0.38	-0.49
	2R		-0.49	-0.45		-0.31	-0.42	0.23			0.35	-0.38	-0.29
1999	P		-0.59	-0.54	0.32	-0.47	-0.29	0.53	-0.62			-0.65	-0.71
	1R		-0.47	-0.49	0.22	-0.20		0.24	-0.44	-0.35		-0.65	-0.71
	2R		-0.72	-0.70				0.3	-0.45	-0.37		-0.37	-0.29
2000	P		-0.29	-0.25									
	1R					-0.49	-0.46					-0.41	-0.46
	2R		-0.49	-0.38		-0.81	-0.76						-0.25
2001	P		-0.23									-0.24	
	1R	0.23	-0.28									-0.20	
	2R					-0.73	-0.65				0.30		0.27
2002	P										0.21		
	1R										-0.20		
	2R					-0.27	-0.20					-0.49	-0.65
2003	P	-0.34	0.21			-0.31	-0.50				-0.47		
	1R	-0.64					-0.22					-0.29	-0.53
	2R											0.31	0.48
2004	P			-0.21							-0.41		
	1R	-0.37		-0.23								-0.25	-0.21

Year	Trial	Mourilyan 2			Tully		
		CCS	TCH	TSH	CCS	TCH	TSH
1995	P				-0.33		
	1R						
	2R						
1996	P		-0.45	-0.50			
	1R		-0.30	-0.33		-0.31	-0.43
	2R	-0.20	-0.50	-0.51		-0.24	
1997	P					0.24	0.22
	1R						
	2R					-0.62	-0.58
1998	P						
	1R						
	2R					-0.61	-0.52
1999	P		-0.41	-0.33	0.51	-0.28	
	1R		-0.46	-0.40	0.21	-0.61	-0.55
	2R		-0.48	-0.48		-0.20	
2000	P				0.43		
	1R						
	2R						
2001	P						
	1R				0.34		
	2R				0.75	0.21	0.57
2002	P				-0.23		
	1R						
	2R				0.29	-0.25	
2003	P						
	1R				-0.33		
	2R						
2004	P				-0.30		

Appendix 3 - Yield loss calculations for Pachymetra root rot using data from each location in northern trial data for 1995-2004 (blank spaces occur where the r-squared <0.20)

Year	Trial	Babinda TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1995	P												
	1R												
	2R												
1996	P												
	1R	72.4	-1.32	72.4	71.08	59.2	**16.7**	9.4	-0.185	9.4	9.215	7.55	**18.1**
	2R												
1997	P	119.8	-2.94	119.8	116.86	90.4	**22.6**	15.2	-0.312	15.2	14.888	12.08	**18.9**
	1R	101.8	-3.04	101.8	98.76	71.4	**27.7**	14.8	-0.349	14.8	14.451	11.31	**21.7**
	2R												
1998	P												
	1R	60.5	-2.22	60.5	58.28	38.3	**34.3**	8.9	-0.35	8.9	8.55	5.4	**36.8**
	2R	69	-3.02	69	65.98	38.8	**41.2**	11.7	-0.505	11.7	11.195	6.65	**40.6**
1999	P	64.8	-1.21	64.8	63.59	52.7	**17.1**	9.3	-0.164	9.3	9.136	7.66	**16.2**
	1R	67.7	-1.1	67.7	66.6	56.7	**14.9**	10.9	-0.185	10.9	10.715	9.05	**15.5**
	2R	101.3	-2.49	101.3	98.81	76.4	**22.7**	17.9	-0.545	17.9	17.355	12.45	**28.3**
2000	P	60.5	-0.783	60.5	59.717	52.67	**11.8**	9.9	-0.139	9.9	9.761	8.51	**12.8**
	1R												
	2R	105.6	-3.15	105.6	102.45	74.1	**27.7**	17.3	-0.525	17.3	16.775	12.05	**28.2**
2001	P	109.3	-0.801	109.3	108.499	101.29	**6.6**	17.3	-0.118	17.3	17.182	16.12	**6.2**
	1R	103.4	-0.732	103.4	102.668	96.08	**6.4**						
	2R												
2002	P												
	1R												
	2R												
2003	P												
	1R												
	2R												
2004	P							13.6	-0.133	13.6	13.467	12.27	**8.9**
	1R							9.6	-0.089	9.6	9.511	8.71	**8.4**

Year	Trial	Mulgrave 1 TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1995	P												
	1R												
	2R												
1996	P	116.7	-2.7	116.7	114	89.7	**21.3**	19.5	-0.459	19.5	19.041	14.91	**21.7**
	1R	116.7	-1.7	116.7	115	99.7	**13.3**	17.9	-0.313	17.9	17.587	14.77	**16.0**
	2R	102.5	-2.3	102.5	100.2	79.5	**20.7**	16.1	-0.341	16.1	15.759	12.69	**19.5**
1997	P	106.5	-0.988	106.5	105.512	96.62	**8.4**	16.3	-0.153	16.3	16.147	14.77	**8.5**
	1R	109	-1.68	109	107.32	92.2	**14.1**	17.2	-0.298	17.2	16.902	14.22	**15.9**
	2R	82	-1.38	82	80.62	68.2	**15.4**	12.7	-0.265	12.7	12.435	10.05	**19.2**
1998	P	124.6	-1.39	124.6	123.21	110.7	**10.2**	18.2	-0.237	18.2	17.963	15.83	**11.9**
	1R	92.8	-1.1	92.8	91.7	81.8	**10.8**	13.9	-0.215	13.9	13.685	11.75	**14.1**
	2R	101.3	-1.96	101.3	99.34	81.7	**17.8**	18	-0.386	18	17.614	14.14	**19.7**
1999	P	80.1	-1.76	80.1	78.34	62.5	**20.2**	11.7	-0.192	11.7	11.508	9.78	**15.0**
	1R	93.5	-1.78	93.5	91.72	75.7	**17.5**	15.7	-0.228	15.7	15.472	13.42	**13.3**
	2R												
2000	P												
	1R	80.6	-1.13	80.6	79.47	69.3	**12.8**	13.6	-0.181	13.6	13.419	11.79	**12.1**
	2R	104.1	-5.46	104.1	98.64	49.5	**49.8**	16.7	-0.852	16.7	15.848	8.18	**48.4**
2001	P												
	1R												
	2R	129.7	-5.39	129.7	124.31	75.8	**39.0**	23.6	-0.942	23.6	22.658	14.18	**37.4**
2002	P												
	1R												
	2R	139.7	-3.84	139.7	135.86	101.3	**25.4**	21.9	-0.532	21.9	21.368	16.58	**22.4**
2003	P	101.3	-1.53	101.3	99.77	86	**13.8**	13.1	-0.261	13.1	12.839	10.49	**18.3**
	1R							22.3	-0.148	22.3	22.152	20.82	**6.0**
	2R												
2004	P												

Year	Trial	Mulgrave 2 TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1995	**P**												
	1R	113.7	-2.62	113.7	111.08	87.5	**21.2**	18.6	-0.147	18.6	18.453	17.13	**7.2**
	2R												
1996	**P**	132.2	-1.13	132.2	131.07	120.9	**7.8**	20.1	-0.149	20.1	19.951	18.61	**6.7**
	1R	129.6	-1.72	129.6	127.88	112.4	**12.1**	20.8	-0.239	20.8	20.561	18.41	**10.5**
	2R	107.1	-3.82	107.1	103.28	68.9	**33.3**	17.6	-0.571	17.6	17.029	11.89	**30.2**
1997	**P**	112.8	-2.08	112.8	110.72	92	**16.9**	16.1	-0.239	16.1	15.861	13.71	**13.6**
	1R	107.2	-5.34	107.2	101.86	53.8	**47.2**	16.7	-0.764	16.7	15.936	9.06	**43.1**
	2R	71.5	-1.88	71.5	69.62	52.7	**24.3**						
1998	**P**	122.5	-1.57	122.5	120.93	106.8	**11.7**						
	1R	89.5	-1.18	89.5	88.32	77.7	**12.0**	12.6	-0.161	12.6	12.439	10.99	**11.6**
	2R												
1999	**P**	108.1	-2.61	108.1	105.49	82	**22.3**	13.9	-0.118	13.9	13.782	12.72	**7.7**
	1R	108.8	-2.41	108.8	106.39	84.7	**20.4**	18.4	-0.355	18.4	18.045	14.85	**17.7**
	2R	93.5	-3.29	93.5	90.21	60.6	**32.8**	16.1	-0.482	16.1	15.618	11.28	**27.8**
2000	**P**												
	1R												
	2R												
2001	**P**												
	1R												
	2R												
2002	**P**												
	1R												
	2R												
2003	**P**												
	1R												
	2R												
2004	**P**												

Year	Trial											
	Mourilyan 1											
	TCH							TSH				
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1995	P												
	1R	92.6	-1.11	92.6	91.49	81.5	**10.9**						
	2R												
1996	P	76.5	-0.92	76.5	75.58	67.3	**11.0**	11.5	-0.147	11.5	11.353	10.03	**11.7**
	1R	75.9	-1.34	75.9	74.56	62.5	**16.2**	11	-0.15	11	10.85	9.5	**12.4**
	2R	69.4	-2.1	69.4	67.3	48.4	**28.1**	10.7	-0.327	10.7	10.373	7.43	**28.4**
1997	P												
	1R	68.2	-2.68	68.2	65.52	41.4	**36.8**	10	-0.44	10	9.56	5.6	**41.4**
	2R	39.5	-1.34	39.5	38.16	26.1	**31.6**	6.1	-0.228	6.1	5.872	3.82	**34.9**
1998	P	107.4	-3.13	107.4	104.27	76.1	**27.0**	16.5	-0.496	16.5	16.004	11.54	**27.9**
	1R	107.8	-1.41	107.8	106.39	93.7	**11.9**	16.2	-0.273	16.2	15.927	13.47	**15.4**
	2R	99.1	-2.65	99.1	96.45	72.6	**24.7**	17.1	-0.392	17.1	16.708	13.18	**21.1**
1999	P	86.1	-3.77	86.1	82.33	48.4	**41.2**	15.6	-0.656	15.6	14.944	9.04	**39.5**
	1R												
	2R	100.6	-0.757	100.6	99.843	93.03	**6.8**	18.4	-0.131	18.4	18.269	17.09	**6.5**
2000	P												
	1R	108.1	-1.51	108.1	106.59	93	**12.7**	18.5	-0.251	18.5	18.249	15.99	**12.4**
	2R							16.2	-0.241	16.2	15.959	13.79	**13.6**
2001	P	121.1	-1.51	121.1	119.59	106	**11.4**						
	1R	101.5	-0.976	101.5	100.524	91.74	**8.7**	15.3	-0.134	15.3	15.166	13.96	**8.0**
	2R												
2002	P												
	1R												
	2R	117.9	-3.12	117.9	114.78	86.7	**24.5**	19.5	-0.559	19.5	18.941	13.91	**26.6**
2003	P												
	1R	93.3	-0.85	93.3	92.45	84.8	**8.3**	14.7	-0.148	14.7	14.552	13.22	**9.2**
	2R												
2004	P							17.9	-0.131	17.9	17.769	16.59	**6.6**
	1R	73.8	-0.842	73.8	72.958	65.38	**10.4**	10.3	-0.111	10.3	10.189	9.19	**9.8**

Year	Trial	Mourilyan 2 TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1995	P												
	1R												
	2R												
1996	P	119.8	-1.47	119.8	118.33	105.1	**11.2**	19.4	-0.29	19.4	19.11	16.5	**13.7**
	1R	90.8	-1.86	90.8	88.94	72.2	**18.8**	13.2	-0.297	13.2	12.903	10.23	**20.7**
	2R	52.5	-1.38	52.5	51.12	38.7	**24.3**	8	-0.211	8	7.789	5.89	**24.4**
1997	P												
	1R												
	2R												
1998	P												
	1R												
	2R												
1999	P	88.7	-1.41	88.7	87.29	74.6	**14.5**	12.6	-0.141	12.6	12.459	11.19	**10.2**
	1R	92.6	-1.87	92.6	90.73	73.9	**18.5**	15.1	-0.254	15.1	14.846	12.56	**15.4**
	2R	116.8	-1.1	116.8	115.7	105.8	**8.6**	21.3	-0.254	21.3	21.046	18.76	**10.9**
2000	P												
	1R												
	2R												
2001	P												
	1R												
	2R												
2002	P												
	1R												
	2R												
2003	P												
	1R												
	2R												
2004	P												

Year	Trial Tully TCH	Intercept	M	0 Rating	1 rating	9 rating	% loss	TSH Intercept	M	0 rating	1 rating	9 rating	% loss
1995	**P**												
	1R												
	2R												
1996	**P**												
	1R	91.2	-0.314	91.2	90.886	88.06	**3.1**	12.6	-0.429	12.6	12.171	8.31	**31.7**
	2R	100.2	-1.23	100.2	98.97	87.9	**11.2**						
1997	**P**												
	1R												
	2R	72.1	-3.75	72.1	68.35	34.6	**49.4**	10.1	-0.494	10.1	9.606	5.16	**46.3**
1998	**P**												
	1R												
	2R	115.2	-3.09	115.2	112.11	84.3	**24.8**	18.4	-0.39	18.4	18.01	14.5	**19.5**
1999	**P**	71.4	-1.63	71.4	69.77	55.1	**21.0**	10.4	-0.2	10.4	10.2	8.4	**17.6**
	1R	102.2	-1.99	102.2	100.21	82.3	**17.9**	16.5	-0.278	16.5	16.222	13.72	**15.4**
	2R	114.4	-0.381	114.4	114.019	110.59	**3.0**						
2000	**P**												
	1R												
	2R												
2001	**P**												
	1R												
	2R												
2002	**P**												
	1R												
	2R	101.7	-1.98	101.7	99.72	81.9	**17.9**						
2003	**P**												
	1R												
	2R												
2004	**P**												

Appendix 4 - Yield loss calculations for Pachymetra root rot using average northern trial data for 1999 and 2003 data

Year	Trial											
	Northern Average											
	TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P	78.36	-0.5707	78.36	77.7893	72.653	**6.6**	11.404	-0.1402	11.404	11.2638	10.002	**11.2**
	1R	91.363	-1.6183	91.363	89.7447	75.18	**16.2**	15.082	-0.2191	15.082	14.8629	12.891	**13.3**
	2R												
2003	P	91.128	-0.6816	91.128	90.4464	84.312	**6.8**	13.523	-0.0923	13.523	13.4307	12.6	**6.2**
	1R	112.97	-0.0923	112.97	112.8777	112.047	**0.7**	18.461	-0.0042	18.461	18.4568	18.419	**0.2**
	2R												

Appendix 5 - R-squared values for the regressions between yellow spot resistance and yield component for each FAT in northern Queensland (1999 and 2003 series; blank spaces occur where the r-squared <0.20)

Year	Trial	Babinda			Mulgrave 1			Mulgrave 2			Mourilyan 1		
		CCS	TCH	TSH	CCS	TCH	TSH	CCS	TCH	TSH	CCS	TCH	TSH
1999	P					-0.44	-0.49						
	1R	0.48				-0.38	-0.22						
	2R												
2003	P											-0.26	-0.40
	1R										0.21		
	2R												

Year	Trial	Mourilyan 2			Tully		
		CCS	TCH	TSH	CCS	TCH	TSH
1999	P	0.63	-0.25			-0.36	-0.54
	1R		-0.18		0.21	-0.57	-0.69
	2R						
2003	P						
	1R						
	2R				-0.4		

Appendix 6 - Yield loss calculations for yellow spot in northern trials for 1999 and 2003 series trials (blank spaces occur where the r-square for the regression was <0.20)

Year	Trial	Babinda											
		TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P						0.0						0.0
	1R						0.0						0.0
	2R	101.3	-2.49	101.3	98.81	76.4	22.7	17.9	-0.545	17.9	17.355	12.45	28.3
2003	P												
	1R												
	2R												

Year	Trial	Mulgrave 1											
		TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P	84.7	-1.71	84.7	82.99	67.6	18.5	12.2	-0.191	12.2	12.009	10.29	14.3
	1R	96.7	-1.53	96.7	95.17	81.4	14.5	15.5	-0.141	15.5	15.359	14.09	8.3
	2R												
2003	P						0.0						0.0
	1R												0.0
	2R												

Year	Trial	Mulgrave 2											
		TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P						0.0						0.0
	1R						0.0						0.0

	2R					0.0							0.0
2003	P												
	1R												
	2R												

Year	Trial	Mourilyan 1											
		TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P						0.0						0.0
	1R						0.0						0.0
	2R						0.0						0.0
2003	P												
	1R						0.0						0.0
	2R												

Year	Trial	Mourilyan 2											
		TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P	99.2	-2.6	99.2	96.6	73.2	24.2						0.0
	1R	88.1	-0.476	88.1	87.624	83.34	4.9						0.0
	2R						0.0						0.0
2003	P						0.0						
	1R						0.0						
	2R												

Year	Trial	Tully						TSH					
	TCH	Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P	76.9	-1.94	76.9	74.96	57.5	23.3	11.2	-0.252	11.2	10.948	8.68	20.7
	1R	118.3	-3.44	118.3	114.86	83.9	27.0	18.5	-0.457	18.5	18.043	13.93	22.8
	2R												
2003	P												
	1R	126.5	-1.89	126.5	124.61	107.6	13.7	22.1	-0.414	22.1	21.686	17.96	17.2
	2R						0.0						

Appendix 7 - Yield loss calculations for yellow spot using average northern trial data for 1999 and 2003

Year	Trial	Northern TCH					TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	**P**	86.004	-1.4852	86.004	84.5188	71.152	**15.8**	11.87	-0.1491	11.87	11.7209	10.379	**11.4**
	1R	93.738	-1.2306	93.738	92.5074	81.432	**12.0**	15.087	-0.1344	15.087	14.9526	13.743	**8.1**
2003	**P**	83.74	0.0606	83.74	83.8006	84.346	**-0.7**	13.596	-0.0702	13.596	13.5258	12.894	**4.7**
	1R	111.12	-1.3707	111.12	109.7493	97.413	**11.2**	18.27	-0.2191	18.27	18.0509	16.079	**10.9**

Appendix 8 - R-squared values for the regressions between orange rust resistance and yield component for each FAT in northern Queensland (1999 and 2003 series; blank spaces occur where the r-square for the regression <0.20)

Year	Trial	Babinda			Mulgrave 1			Mulgrave 2			Mourilyan 1		
		CCS	TCH	TSH	CCS	TCH	TSH	CCS	TCH	TSH	CCS	TCH	TSH
1999	P	-0.38		-0.22		-0.73	-0.61	-0.2		-0.33			
	1R		-0.47	-0.49	0.22	-0.20		0.24	-0.44	-0.35			
	2R		-0.72	-0.70				0.3	-0.45	-0.37			
2003	P											-0.26	-0.40
	1R										0.22		
	2R	0.76	-0.89	-0.83									

Year	Trial	Mourilyan 2			Tully		
		CCS	TCH	TSH	CCS	TCH	TSH
1999	P				-0.23		
	1R		-0.37				
	2R						
2003	P						
	1R						
	2R					0.32	0.43

Appendix 9 - Yield loss calculations for orange rust for northern trials for the 1999 and 2003 series (blank spaces occur where the r-square for the regression <0.20)

Year	Trial	Babinda											
		TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P						**0.0**	9.1	-0.131	9.1	8.969	7.79	**13.1**
	1R	70.7	-1.94	70.7	68.76	51.3	**25.4**	11.2	-0.272	11.2	10.928	8.48	**22.4**
							0.0						
2003	P												
	1R												
		74.3	-2.82	74.3	71.48	46.1	**35.5**	10.9	-0.358	10.9	10.542	7.32	**30.6**

Year	Trial	Mulgrave 1											
		TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P	78.8	-1.78	78.8	77.02	61	**20.8**	11.8	-0.276	11.8	11.524	9.04	**21.6**
	1R	99.5	-3.64	99.5	95.86	63.1	**34.2**	17	-0.606	17	16.394	10.94	**33.3**
2003	P						**0.0**						**0.0**
	1R												**0.0**

Year	Trial	Mulgrave 2 TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P						0.0	14	-0.224	14	13.776	11.76	14.6
	1R						0.0						0.0
							0.0						0.0
2003	P												
	1R												

Year	Trial	Mourilyan 1 TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P						0.0						0.0
	1R												0.0
							0.0						0.0
2003	P												0.0
	1R						0.0						0.0

Year	Trial	Mourilyan 2 TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P						0.0						0.0
	1R	87.7	-1.1	87.7	86.6	76.7	11.4						0.0
							0.0						0.0
2003	P												
	1R	86.5	-1.86	86.5	84.64	67.9	19.8	13.5	-0.239	13.5	13.261	11.11	16.2

Year	Trial	Tully					TCH						TSH
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	**P**						**0.0**						**0.0**
	1R						**0.0**						**0.0**
2003	**P**												
	1R												
		65.9	2.44	65.9	68.34	90.3	**-32.1**	9.6	0.411	9.6	10.011	13.71	**-36.9**

Appendix 10 - Yield loss calculations for orange rust using average central trial data for 1999 and 2003 series

Year	Trial	Central Average TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P	98.662	-4.4804	98.662	94.1816	53.858	**42.8**	16.235	-0.7902	16.235	15.4448	8.333	**46.0**
	1R	99.401	-1.5483	99.401	97.8527	83.918	**14.2**	15.127	-0.1517	15.127	14.9753	13.61	**9.1**
2003	P	66.875	0.4546	66.875	67.3296	71.421	**-6.1**	10.59	0.1324	10.59	10.7224	11.914	**-11.1**
	1R	65.745	-0.0404	65.745	65.7046	65.341	**0.6**	11.249	0.0514	11.249	11.3004	11.763	**-4.1**

Appendix 11 - R-squared values for the regressions between orange rust resistance and yield component for each FAT in central Queensland (1999 and 2003 series; blank spaces occur where the r-square for the regression <0.20)

Year	Trial	Farleigh			Marian			Pleystowe			Racecourse		
		CCS	TCH	TSH	CCS	TCH	TSH	CCS	TCH	TSH	CCS	TCH	TSH
1999	P		-0.66	-0.62		-0.45	-0.46		-0.72	-0.71	-0.56		-0.53
	1R	-0.39	-0.46	-0.36	-0.26			-0.65	-0.61	-0.51	-0.41		
2003	P												
	1R	0.24			0.68	0.77	0.74				0.23	0.25	

		Plane Creek			Proserpine		
		CCS	TCH	TSH	CCS	TCH	TSH
1999	P		-0.28			-0.39	-0.45
	1R		-0.69	-0.53			
2003	P				-0.66		
	1R					0.72	0.85

Appendix 12 - Yield loss calculations for orange rust using data from each trials in the central district for 1999 and 2003 series trials (blank spaces occur where the r-square for the regression <0.20)

Year	Trial											
	Farleigh											
	TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P	122.88	-7.541	122.88	115.339	47.47	**58.8**	19.479	-1.209	19.479	18.27	7.389	**59.6**
	1R	141.71	-4.231	144.71	140.479	102.4	**27.1**	22.69	-0.5959	22.69	22.0941	16.731	**24.3**
2003	P												
	1R												

Year	Trial											
	Marian											
	TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P	96.421	-3.1009	96.421	93.3201	65.412	29.9	15.459	-0.514	15.459	14.945	10.319	31.0
	1R												
2003	P												
	1R	44.636	2.265	44.636	46.901	67.286	-43.5	7.0076	0.4481	7.0076	7.4557	11.4886	-54.1

Year	Trial											
	Pleystowe											
	TCH						TSH					
		Intercept	M	0 Rating	1 rating	9 rating	% loss	Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P	63.693	-3.9092	63.693	59.7838	24.601	**58.9**	10.395	-0.6372	10.395	9.7578	4.023	**58.8**
	1R	106.28	-2.5167	106.28	103.7633	81.113	**21.8**	17.406	-0.3114	17.406	17.0946	14.292	**16.4**
2003	P												
	1R												

Year	Trial	Racecourse TCH Intercept	M	0 Rating	1 rating	9 rating	% loss	TSH Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P							17.815	-0.5946	17.815	17.2204	11.869	**31.1**
	1R												
2003	P												
	1R	65.029	-0.8257	65.029	64.2033	56.772	**11.6**						

Year	Trial	Plane Creek TCH Intercept	M	0 Rating	1 rating	9 rating	% loss	TSH Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P	104.65	-2.3628	104.65	102.2872	81.022	**20.8**						
	1R	102.49	-2.8447	102.49	99.6453	74.043	**25.7**	14.382	-0.4621	14.382	13.9199	9.761	**29.9**
2003	P												
	1R												

Year	Trial	Proserpine TCH Intercept	M	0 Rating	1 rating	9 rating	% loss	TSH Intercept	M	0 rating	1 rating	9 rating	% loss
1999	P	81.054	-2.9377	81.054	78.1163	51.677	**33.8**	13.509	-0.4649	13.509	13.0441	8.86	**32.1**
	1R												
2003	P												
	1R	74.468	3.8249	74.468	78.2929	112.717	**-44.0**	12.497	0.7185	12.497	13.2155	19.682	**-48.9**

APPENDIX 2 – Paper by Wei *et al.*. (2006)