

ORANGE RUST DISEASE OF SUGARCANE

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KEYWORDS: Orange rust, *Puccinia kuehni*,
Resistance, Early Warning, Fungicides.

Abstract

AN OUTBREAK of orange rust (*Puccinia kuehni*) in the Australian sugarcane industry in 2000 caused devastating yield losses and significant financial hardships to cane farmers, sugar factories and the local communities that were reliant on the crop. The epidemic was first recognised in the year 2000, but later analyses suggest that orange rust was present in central (Mackay) region crops at least 1–2 years (in 1998–1999) prior to detection. This was evident in comparative sugar factory productivity data for several varieties and in yield loss analyses in breeding selection trials. Yield loss studies (breeding selection trials and fungicides) suggested losses of around 40% from orange rust. Resistance assessments showed there to be a high level of disease resistance in many commercial varieties. Fungicide research led to the application of fungicides to commercial crops to minimise yield losses. Large quantities of resistant seed-cane were transported long distances from neighbouring regions to assist farmers in replanting susceptible crops. Genetic analyses suggested that there were minor variations in the pathogen ‘pre-’ and ‘post-epidemic’; some variation was observed in pathogen isolates collected from its geographical range around the world. Possibilities for remote detection of the disease were investigated and techniques could be used to provide early warning of the presence of the disease.

Introduction

Recent detections of orange rust (*Puccinia kuehni*) in North and Central America have continued to emphasise the new-found importance of this disease in commercial crops.

Originally restricted to south Asia, south east Asia, and the Pacific (Ryan and Egan, 1989; Magarey *et al.* (2002b, 2003a)), the disease has now been detected in the western hemisphere in North and Central America (Chavarria *et al.* (2009), Comstock *et al.* (2008), Ovalle *et al.* (2008), Flores *et al.* (2009)).

Australian evidence suggests that the recent outbreak, as reported by Magarey (2000), Magarey *et al.* (2001a, 2001b, 2002a, 2003b), are more serious than those occurring earlier in history.

A new, more aggressive strain of the pathogen may be the reason for these observations (Braithwaite *et al.*, 2009).

In this paper, epidemiological considerations associated with the disease, the major effects of orange rust, the framework used in Australia for implementation of management strategies, potential early warning techniques and the presence of disease resistance in Australian germplasm are reviewed.

History of Orange Rust in Australia

Orange rust has been reported within the Australian sugarcane industry since the late 1800s, though the accuracy of some disease records has been questioned (Ryan and Egan, 1989). With the disease common in neighbouring Papua New Guinea, the centre of diversity of several *Saccharum* species, it is likely that orange rust entered Australia with PNG germplasm. Despite this, 20th century reports of diseased commercial crops were rare. When brown rust (*Puccinia melanocephala*) first entered the Australian industry in 1978, that disease became known as 'rust' – there was no recognised need to distinguish brown rust from orange rust in commercial crops emphasising the minor importance of orange rust. As such, there were no purposeful orange rust disease assessments, screening for resistance, or regular reporting of the area affected in commercial crops.

Strains of *Puccinia kuehnii*

The rapid appearance of the disease in a widely-planted variety that had for a number of years shown no evidence of the disease lent weight to the hypothesis that the epidemic arose from a new pathogen strain. Molecular analysis of historical Australian samples (1898), samples collected from Indonesia, Papua New Guinea, Central America and from within the Australian sugarcane industry could not clearly pinpoint the nature of the strain change, but did show pathogen variation (Braithwaite *et al.*, 2009).

Braithwaite *et al.* (2009) concluded that isolates from commercial crops clustered together and this included the 1898 Australian isolate plus more recent isolates. Two other clades (groups) included *P. kuehnii* isolates from wild and garden canes in Indonesia and PNG. Though morphologically similar to *P. kuehnii*, there was more variation in these samples and they pose a potential further threat to the Australian (and other) sugarcane industry. They concluded there is a greater diversity in *P. kuehnii* isolates than thought previously (Braithwaite *et al.*, 2009).

Orange Rust epidemic in 2000

The status of orange rust changed in the year 2000 with its detection in the widely-planted commercial Australian variety, Q124. The epidemic was severe, crop canopies were largely dead, and biomass production stifled. Many very poorly-grown crops were present in the central (Mackay) region; symptoms included thin, rubbery, pithy, shortened stalks and senescing leaves.

Farmers venturing into their fields on machinery emerged with orange-coloured shirts and hats, and there were reports of concrete veranda floors covered with an orange film. Urgent action was needed to minimise crippling yield losses, and a major industry program involving research, strategic economic incentives and on-farm strategies was implemented to minimise economic effects.

Orange rust was first reported in Q124 crops in January 2000 in the Mackay district of central Queensland. Q124 is susceptible to yellow spot (*Mycovellosiella koepkei*) disease and previous high rainfall years favoured this disease. Observations in January 2000 suggested, however, that the observed symptoms were different to those of yellow spot; samples were dispatched to a pathologist who identified the disease as orange rust based on pustule and spore characteristics.

Rapid escalation in disease severity accompanied progress of the normal wet season and reports soon came of the disease in most other parts of the industry, including northern, Herbert, Burdekin and southern production areas. Orange rust was not seen in the southern-most Australian production area of northern New South Wales until the following year. By March 2000, the regions where Q124 was widely planted (central and Herbert) were badly affected with crop canopies in very poor health. Crops yields plummeted compared to previous years and individual farmers, sugar factories and service companies all were badly affected financially.

Evidence for early incidence

There is strong evidence that the disease first affected sugarcane crops in the central district before January 2000, there being two lines of evidence. Yield loss assessments in BSES plant improvement selection trials showed a relationship between orange rust resistance and yield in 1999-harvested crops in the central part of the Mackay district (Magarey *et al.*, 2009a).

These losses were not seen further afield but in central Mackay trials only. Additionally, relative commercial crop yields in two significant Mackay varieties (one susceptible during the epidemic (Q124) and one that remained resistant (Q135)) showed that Q124 very significantly out-yielded Q135 in the mid-1990s, but by 1998 a trend to lower relative yield in Q124 emerged; the difference grew significantly post-2000.

With the presence of two leaf diseases, it is reasonable to conclude that orange rust was not detected in Q124 for 1–2 years. Close observation of individual leaves is needed to separate orange rust from yellow spot and this didn't occur until orange rust severity rose significantly.

This provides an object lesson – that vigilant crop observations by pathologists trained in endemic and exotic diseases are needed to ensure an early warning of impending disease epidemics.

Yield loss research

Yield loss data have been published elsewhere, but a brief summary provides an indication of the extent of losses.

Breeding selection trials

Breeding selection trial analyses are based on relating the yield of a range of clones (80+) to their orange rust resistance in individual trials (Magarey *et al.*, 2003b). The technique has been used to quantify yield losses caused by a range of endemic Australian diseases (Magarey *et al.*, 2003b, 2009a).

Losses at individual sites in the Mackay region in the year 2000 reached 58% (tonnes cane / ha), though the mean figure was around 40%.

This was further emphasised by the relative performance of Q124 within selection trials; before 2000 Q124 was the most productive variety but, during and after 2000, it became one of the lowest yielding (Magarey *et al.*, 2003b). Losses were not restricted to biomass, but included sugar content (CCS) with a reduction of 1 to 2 units.

Fungicide studies

Fungicide studies have also been reported elsewhere (Magarey *et al.*, 2002a; Staier *et al.*, 2003). Several experiments undertaken near Mackay showed yield improvements between treated and untreated plots of 40%, reinforcing the selection trial estimates. Reductions in sugar content of around 1–2 units again were reported (Staier *et al.*, 2003).

Management of the epidemic

Magarey *et al.* (2001a) report on the management strategies associated with the epidemic. A focused, industry-based strategy, implemented as soon as the epidemic was recognised, greatly assisted with epidemic management.

The main components were:

- i. the supply of orange rust resistant varieties that were free of systemically transmitted pathogens, from as far afield as 500 km away,
- ii. the acceleration of resistant clones within the plant improvement program,
- iii. the gaining of resistance data on both clones and parent canes and modified selection and crossing programs, and
- iv. the application of effective fungicides to susceptible crops, thus minimising losses during the establishment of resistant crops.

Varieties

There was no problem identifying susceptible clones in variety trials in badly affected districts; senescing crop canopies in very poor health were very obvious. Quantitative resistance screening techniques were developed which utilised comparative standard varieties of known disease reaction (Magarey and Bull, 2009). Plant breeders discarded susceptible clones from selection programs and pathologists screened parent varieties for resistance to ensure suitably resistant crosses were made between parents (Magarey and Bull, 2009). Data showed that a high level of resistance was present within the Australian sugarcane germplasm (Magarey *et al.*, 2003b; Magarey *et al.*, 2009a, Magarey, 2006). The replacement of the highly susceptible Q124 in district crops effectively ended the epidemic. It has not been difficult since then for plant breeders to release high-yielding commercial varieties possessing a high level of disease resistance.

In the central and Herbert (the two most affected) districts, planting material of replacement resistant varieties was quickly made available in 2000 and 2001. The Mackay sugar factories offered financial incentives for farmers to replant susceptible crops with resistant varieties and the economics of the situation drove a rapid transition to resistant crops. In other districts, the situation was not as severe and farmers transitioned to resistant crops without direct assistance.

Fungicides

There have been no historical fungicide applications for leaf disease control in Australian crops. In fact, there are only rare reports of this on a world basis (Autrey *et al.*, 1983). The need for a rapid disease control was so urgent in the Mackay region that widespread fungicide application occurred in-lieu of transition to resistant varieties. Staier *et al.* (2003) report on research undertaken in 2001 with the fungicides propiconazole, cyproconazole and mancozeb. Good control was achieved with as little as two spray applications, as long as the applications were made sufficiently early (when leaf area diseased reached 5%). Knowledge of the effect of weather conditions (Magarey *et al.*, 2004, Staier *et al.*, 2004) and predictive modelling assisted in minimising fungicide applications. The products were granted temporary registration and over 30 000 ha treated (Staier *et al.*, 2003). Aerial applications were undertaken in 2001, but low sugar prices and the successful transition to resistant canes led to no, or very limited, further fungicide applications in the following years.

Epidemic considerations

Previous disease epidemics in Australia

There have been many disease epidemics in the Australian sugarcane industry, including those caused by sugarcane mosaic, leaf scald, Fiji leaf gall, brown rust, *Pachymetra* root rot and more recently sugarcane smut. Almost all began when the affected region had been widely cropped with one variety for a period of around 10 years.

Examples include Fiji leaf gall in southern Queensland where NCo310 dominated plantings (>80% of crops) for at least 10 years (Egan, 1982). The variety Q90 was widely planted in the northern region (>60% production) in the 1970–1980 period before *Pachymetra* root rot and brown rust both drastically reduced commercial yields. In central Queensland, the highly productive Q124 was widely grown, and constituted >80% of the regional production before the orange rust outbreak. It seems that whenever a single variety exceeds, on an extended basis, approximately 60% of production, a disease epidemic is initiated.

Progress of the orange rust epidemic

Progress of the orange rust epidemic was very rapid; the disease spread the length of the Australian sugarcane industry (approximately 2100km) in just over 12 months – though pre-detection disease occurrence suggests spread is likely to have begun in 1998 or 1999. Similar spread was observed with the Australian brown rust incursion; Ryan and Egan (1989) reported that *P. melanocephala* also spread through the industry within 12 months.

In contrast, spread of Fiji leaf gall (Egan, 1982) and sugarcane smut (Magarey *et al.*, 2009b) have been much slower thus allowing more time for the transition to resistant varieties. The slower spread is no doubt a consequence of the involvement of a slower spreading vector with limited infective ability (Fiji leaf gall) and the limited target infection court for sugarcane smut (compared to orange rust). The orange rust yield effects quickly drove Mackay region farmers to transition to more resistant varieties and this is illustrated in Figure 1.

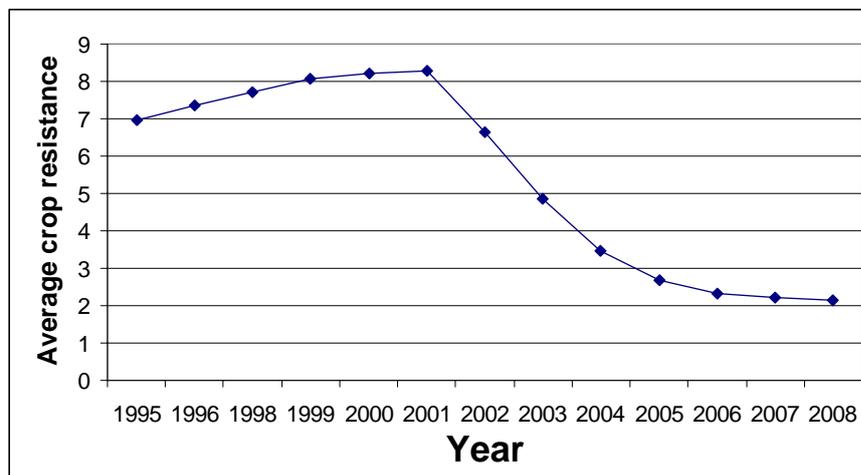


Fig. 1—The orange rust crop resistance profile for Central Queensland 1995–2009: this provides a measure of the average resistance of crops grown in the region. The 2000 epidemic greatly influenced farmer variety choice, thus leading to a sudden surge toward resistant crops.

Comparison to smut

Smut has not spread as quickly (Magarey *et al.*, 2009b). The lower initial percentage of smut resistant varieties in the Australian industry has made the smut epidemic variety transition more difficult. Magarey *et al.* (2009b) suggest that smut will spread across an individual region (to reach all farms) within 2–3 years from first detection and will build up approximately 10-fold annually leading to very significant yield losses in susceptible crops 2–3 years from the first crop detection in that crop.

Remote detection

Mackay Sugar Limited (operating Mackay sugar factories) cooperated with researchers to develop remote sensing systems for orange rust detection (Apan *et al.*, 2004). Several methodologies were investigated.

The authors concluded that accessing hyperion imagery enabled the remote detection of diseased crops possible. This offers other sugarcane industries scope for early detection of orange rust in order to speedily introduce appropriate management systems. Magarey *et al.* (2008, 2009c) found that spore trapping using commercial spore traps ('spore and pollen samplers', Burkard, England) could provide up to 24 months early warning for the presence of sugarcane smut (previous to the finding of field symptoms in a region). The system they used incorporated a specific molecular assay to confirm the presence of *Ustilago scitaminea* spores on trap tapes; similar systems could be developed for orange rust with assays specific to *P. kuehni*.

Discussion

The Australian sugarcane industry suffered very significant financial losses in the midst of the orange rust epidemic in the 2000–2002 period. These losses were greatly contributed to by industry reliance on a single, high yielding variety, Q124. The relative abundance of high-yielding resistant varieties enabled a rapid transition to resistant commercial crops, and now the disease provides no direct financial constraint to sugarcane production.

Following the epidemic, there has been considerable discussion on the benefits / disadvantages of cropping a large proportion of a region to one specific variety. Such a strategy involves considerable risk to both farmers and factories from the potential occurrence of a disease epidemic. Australian experience lends credence to the view that the widespread planting of a single variety **will lead** to a disease epidemic. However, economic analyses suggest if a variety is of unparalleled productivity, the local sugarcane industry may make greater monetary returns by taking the risk and growing individual varieties until no longer possible because of disease-associated yield losses. Discussions on limiting variety production gathered momentum in Australia following the orange rust outbreak, but there has been no implementation. Previous high financial returns don't always provide a viable way forward when a disease epidemic hits. When the orange rust epidemic occurred, factory through-puts were suddenly cut and immediate financial returns were drastically reduced, making it very difficult for some factories, farmers and local businesses to remain viable.

The widespread planting of single varieties may also lead to fundamental changes in pathogen populations and the predominance of hitherto minor pathogens. The longer-term costs and implications of this are not always considered. Is it the new strain of *Puccinia kuehnii* that developed in Australian Q124 crops the one that has now invaded Central and North America? Would this have happened if Q124 had not been so widely planted in Australian cropping regions? These and other questions remain to be answered.

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LA ROUILLE ORANGÉE DE LA CANNE À SUCRE

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MOTS CLÉS: Rouille Orangée, *Puccinia kuehnii*,
Résistance Variétale, Alerte Précoce, Fongicides.

Résumé

UNE EPIDEMIE de la rouille orangée (*Puccinia kuehnii*) dans la culture de la canne à sucre en Australie en 2000 a causé des pertes importantes et des difficultés financières aux planteurs de canne, sucreries et communautés locaux qui dépend de cette culture. L'épidémie a été constatée en 2000, mais les analyses ultérieures tendent à montrer que la rouille orangée était présente dans la région centrale (Mackay) depuis au moins 1–2 ans (en 1998-1999) avant sa détection. Cela était évident à partir des données comparatives des sucreries sur la productivité de plusieurs variétés ainsi que de l'analyse des pertes de rendement dans les essais de sélection. Les études de l'effet de la rouille orangée sur le rendement (essais de sélection variétale et de fongicides) ont démontré des pertes d'environ 40%. L'évaluation des variétés pour la résistance a montré un niveau de résistance élevé parmi plusieurs variétés commerciales. La recherche sur les fongicides a conduit à une application des fongicides dans plusieurs plantations industrielles afin de minimiser les pertes de rendement. Une importante quantité de boutures des variétés résistantes a été transportée sur de grandes distances des régions avoisinantes pour aider les fermiers à remplacer les variétés sensibles. Les analyses génétiques n'ont montré que des variations mineures dans le pathogène, avant et après l'épidémie, alors qu'une certaine variabilité a été observée parmi les isolats du pathogène collectés sur son étendue géographique à travers le monde. La possibilité de télédétection de la maladie a été étudiée et des techniques pourraient être mises en place comme moyen d'alerte précoce de la présence de la maladie.

**ENFERMEDAD DE LA ROYA NARANJA
DE LA CAÑA DE AZÚCAR**

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BSES Limitedrmagarey@bses.org.au**PALABRAS CLAVE: Royá Naranja, *Puccinia kuehnii*,
Resistencia, Alarma Temprana, Fungicidas.****Resumen**

EN EL 2000, el brote de roya naranja (*Puccinia kuehnii*) causó severas pérdidas tanto en el campo como en las fábricas y serias dificultades financieras a los cultivadores y todas las comunidades relacionadas con el cultivo de la caña de azúcar. La epidemia se reconoció como tal en el año 2000, pero análisis posteriores demostraron que la roya naranja estaba presente en la regional central (Mackay), al menos durante 1–2 años (en 1998–1999) antes de esta fuera detectada como tal. Esto fue evidente en los análisis comparativos de productividad de varias fábricas y diversas variedades así como por las pérdidas en productividad de las diversas pruebas del proceso de mejoramiento. Los estudios sobre pérdidas en productividad (pruebas del proceso de mejoramiento como con fungicidas) indicaron que éstas fueron alrededor del 40% debido a la roya naranja. Las evaluaciones de resistencia mostraron que existe un alto nivel de resistencia a la enfermedad en muchas variedades comerciales. La evaluación de fungicidas llevó a su aplicación en muchos cultivos comerciales para reducir al mínimo las pérdidas en productividad. Grandes cantidades de semilla de variedades resistentes fueron transportadas a grandes distancias para ayudar a los cultivadores en el replante de cultivos susceptibles. El análisis genético sugiere que existen pequeñas variaciones en el patógeno 'pre-' y 'post-epidemia', puesto que se encontraron ligeras diferencias entre los aislamientos recolectados en diferentes regiones del mundo. Las posibilidades de detección temprana de la enfermedad fueron investigadas así como las técnicas que se podrían utilizar para proporcionar una alerta temprana sobre la presencia de la enfermedad.