L. H. Purdy
University of Florida, Gainesville

Lii-Jang Liu
University of Puerto Rico, Mayaguez

J. L. Dean
ARS, USDA, Sugarcane Field Station, Canal Point, FL

Sugarcane Rust, a

Before 1978, sugarcane (interspecific hybrids of *Saccharum*) had escaped the ravages of rust except for small and infrequent outbreaks in Asia. On 3 July 1978, *Puccinia melanocephala* H. & P. Sydow was observed in the Dominican Republic, marking the beginning of an epidemic in the Americas that resulted in significant losses of sugar.

Comparison of cane production during years with rust vs. years before rust has been the only effort to assess losses resulting from sugarcane rust. Production of a very susceptible cultivar, B 4362, was measured in Mexico at four *ingenios* (mill and production areas supporting the mill) for 1978–1979 (before rust) and 1979–1980 (after rust). Losses due to sugarcane rust ranged from 1.5 to 21.7 t of cane per hectare, with an average loss of 12.6 t. The total production loss on the 10,668 ha harvested during 1979–1980 was 1,344,160 t of cane (6). Scientists in Tabasco, Mexico, indicated that almost 50% of B 4362 cane produced during 1981–1982 was lost because of rust.

Announcements that millions of pesos were lost in Cuba because of sugarcane rust are not surprising. During 1978–1979, 40% of the planted surface of sugarcane was in B 4362; the amount was reduced to 28% during 1979–1980. The performance of B 4362 with rust in Cuba must have been similar to its behavior in Mexico.

Sugarcane rust can be caused by *P. melanocephala* and by *P. kuehnii* Butler, but *P. melanocephala* occurs around the world and is more damaging than *P. kuehnii*. Loss of production to rust caused by *P. kuehnii* seems to have been negligible, perhaps because incidence is low and outbreaks are scattered (4). *P. melanocephala* has been reported from almost all areas where sugarcane grows, with the exception of southern South America, Oceania, and several countries in Asia. *P. kuehnii* is seemingly restricted to Australia, Oceania, and Asia. In many reported occurrences of *P. kuehnii*, the pathogen responsible for sugarcane rust was actually *P. melanocephala* (4).

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Fig. 1. Sugarcane rust (*Puccinia melanocephala*) of cultivar CP 63-588 in Florida. Green of water hyacinth in ditch and weeds on ditch bank contrasts with reddish brown of infected sugarcane.

Fig. 2. Rust symptoms on sugarcane cultivar CP 63-588 include pustules and leaf reddening.

Sugarcane rust in the Americas is caused by *P. melanocephala*, even though the pathogen was misidentified in the first report of the disease in the Dominican Republic (7). (A 1956 report of sugarcane rust in Cuba was withdrawn in 1959.) From the Dominican Republic, *P. melanocephala* moved quickly to almost all countries of the Caribbean, to northern South America and Central America, and to the United States, to Florida, Louisiana, and Texas.

Another epidemic of considerable significance also began in late 1978 in Australia, where *P. melanocephala* had not been observed previously. The Australian epidemic might have been initiated by urediospores carried by wind from the Americas (11) or by northeast monsoonal winds from the Indian Ocean (5).

Thus, *P. melanocephala* is in abundance almost everywhere sugarcane is grown. Certainly its spread has been dramatic and its presence of considerable economic significance.

Symptoms and Identification of the Pathogens

In susceptible cultivars, small, elongated, yellowish spots or flecks on both leaf surfaces grow longer and turn brown to orange-brown or red-brown. Individual lesions often retain a slight but definite chlorotic halo, and the leaf becomes reddish brown (Figs. 1 and 2). Lesions 2–10 mm long and 1–3 mm wide parallel the leaf venation. Pustules that produce urediospores almost always develop on the lower leaf surface (Fig. 3). Urediospores form over a considerable period of time, but eventually the lesions darken, teliospores may develop, and the surrounding leaf tissues become necrotic. In some cultivars, teliospores develop without a uredial stage.

Considerable numbers of lesions and pustules may occur on leaves of the susceptible cultivars B 4362 and CL 41-223. Lesions coalesce to form large, irregularly shaped necrotic areas, and young leaves may die prematurely. Cane diameter, cane length, and the number of canes per stool are reduced. Although rust is regarded as a disease of the leaf only, uredia have been reported on leaf sheaths and occasionally on stalks of some *S. spontaneum* cultivars.

Urediospores of *P. melanocephala* (Figs. 4 and 5) may be distinguished from those of *P. kuehnii* by their smaller average size (28–33 × 18–23 μm vs. 30–43 × 17–26 μm), the absence of a thickened apical wall, and a darker spore wall. Butler observed apically thickened and

Newly Important Disease

nonthickened urediospores in different pustules on the same host plant. Also, urediospores with and without apical wall thickenings were observed in the same pustule.

Teliospores of *P. kuehnii* are oblong to club-shaped, narrow below, two-celled but not constricted or only slightly so at the septum, and pale yellow. Teliospores of *P. melanocephala* are club-shaped and two-celled, but the apical cell is dark and rounded and the lower cell is elongated and slightly constricted at the septum (Fig. 6). Butler may have described immature teliospores, and teliospores of both species probably resemble each other much more closely than the descriptions suggest. Study of sugarcane rust in areas of the world where both *P. kuehnii* and *P. melanocephala* occur, such as India and other parts of Asia, might reveal that both species occur simultaneously on the same plant and that interspecific recombinants may exist.

The question of pathogenic races of the sugarcane rusts is poorly resolved. The presence of pathogenic races has been suggested for *P. kuehnii* in India and for *P. melanocephala* in Puerto Rico and Nicaragua. Indicator cultivars should be monitored for changes in susceptibility that might indicate development of new races in various sugarcane production areas (3).

Hosts of sugarcane rust include species in *Saccharum* and related genera. *S. arundinaceum* Retz., *S. nargenga* Wall., *S. officinarum* L., *S. spontaneum* L., and *Sclerostachya fusca* (Roxb.) A. Camus are hosts of *P. kuehnii*, and *S. officinarum, S. spontaneum, Erianthus ravennae* (L.) Beauv., and *E. rufipilis* (Steud.) Griseb. are hosts of *P. melanocephala*.

How the 1978 Epidemic Began

Plant disease epidemics are of special concern in areas where the pathogen was not present previously (5,7), particularly when the resulting production loss is significant. How did the epidemic of sugarcane rust develop in the Americas? The Standing Committee on Sugar Cane Diseases of the International Society of Sugar Cane Technologists had never reported rust in any American country. But in 1978 an epidemic of sugarcane rust began in the Dominican Republic in a cultivar (B 4362) that had been grown free from rust in the Caribbean region for more than 20 years. The nearest known source of *P. melanocephala* to the Americas in early 1978 was the Cameroons in West Africa.

A reasonable assumption is that rust developed in B 4362 in the Dominican Republic in 1978 because: 1) viable urediospores of *P. melanocephala* were deposited on leaves of the cultivar and 2) conditions for infection were favorable and disease developed. Weather condi-

Fig. 3. Rust pustules on susceptible sugarcane cultivar CP 78-1735.

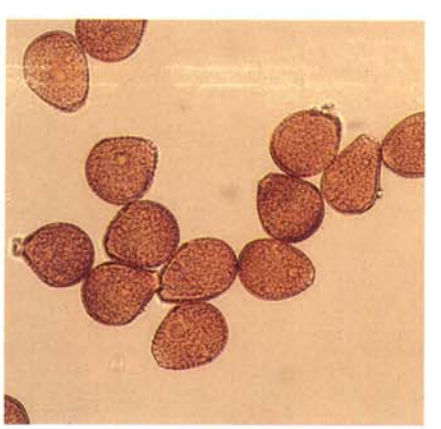

Fig. 4. Urediospores of *Puccinia melanocephala*. Uniform spore wall thickness differentiates *P. melanocephala* from *P. kuehnii*, in which the apical portion of the urediospore wall is thickened. (×400)

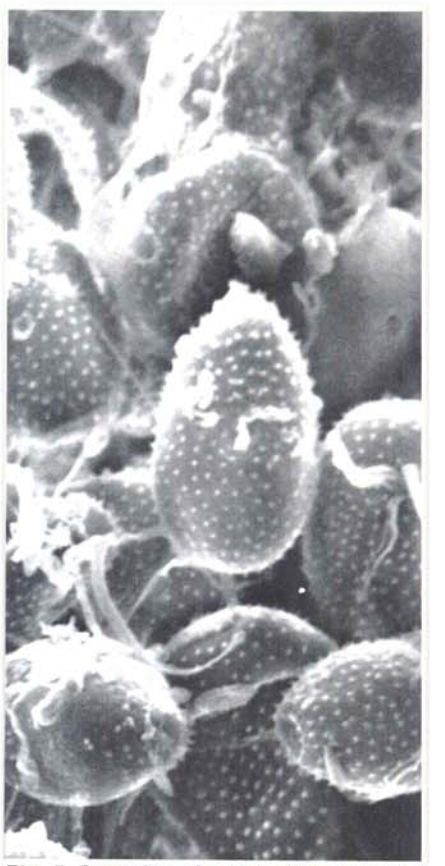

Fig. 5. Scanning electron micrograph of urediospores of *Puccinia melanocephala* showing echinulate nature of outer wall. (×1,000)

Fig. 6. Teliospores of *Puccinia melanocephala* showing constriction at septum and characteristic darker color of terminal cell. (×250)

tions at the time were not unusual, so the most intriguing questions center around the source of inoculum. Where did it come from? There are three possibilities:

1. *P. melanocephala* was always present in the Dominican Republic on other hosts or other cultivars of sugarcane, and rust exploded into a severe problem because of changes in crop management. The demonstrated susceptibility of B 4362 to *P. melanocephala* is ample evidence to reject this option; if rust had been in the Dominican Republic before 1978, its presence would most certainly have been obvious on this very susceptible cultivar. Also, no crop management changes in sugarcane production were made in the Dominican Republic from 1977 to 1978.

2. Rust was introduced into the Dominican Republic by man or by implements employed in sugarcane production or harvest. This option has a low probability for several reasons. For *P. melanocephala* to have been introduced on cane pieces for seed, leaves had to be attached, since pustules are very rare on leaf sheaths. Buds on seed cane might have been infected with rust, but bud infection has not been demonstrated. Buds might have had urediospores on their surfaces, but infection of emerging shoots has also not been demonstrated. Finally, urediospores might have arrived in the Dominican Republic on clothing and cutting equipment brought to the island by imported cane cutters, but this is highly unlikely because there was no transfer of a labor force to the Dominican Republic from areas where rust occurred in 1978 and before, on the African continent and the Mascarene Islands.

3. Viable urediospores were carried by wind from their origin to the infection site in the Dominican Republic. Prevailing winds in the southern hemisphere are east to west, and coffee rust (*Hemileia vastatrix*) probably arrived in Brazil from Africa by wind transport. Intercontinental spread of sigatoka disease of banana (*Mycosphaerella musicola*) into Central America probably resulted from spores carried across the Atlantic Ocean by wind (10). Inoculum production sites for sigatoka, East Africa and the Cameroons, are almost identical to locations where sugarcane rust occurred in early 1978. If sigatoka moved to the Americas on winds from Africa, sugarcane rust could have followed the same pattern of transport.

Prevailing wind direction is the average direction of wind movement, but wind moves in all directions at some time and directional change may be frequent. Cyclonic winds are common in areas where sugarcane grows, and disturbances associated with cyclonic winds may be responsible for vertical movement of urediospores to high altitudes and horizontal movement over long distance in directions not normally touched by prevailing winds. This seems to have been the circumstance that introduced urediospores of *P. melanocephala* into the Dominican Republic from their most likely site of origin, the African continent.

Localized epidemics seem to develop in the direction of surface or near surface winds as well as high-altitude winds, such as coffee rust in Brazil (8). From the Dominican Republic sugarcane rust spread to adjacent islands, southward as far as Venezuela, and westward to Jamaica, Cuba, Central America, Mexico, and Florida, Louisiana, and Texas—all by wind movement of urediospores. Within a year, sugarcane rust had attained a Pan-American distribution.

A common source of inoculum for the outbreaks of sugarcane rust in the Americas and Australia is unlikely, according to Egan and Ryan (5). The Australian epidemic might have originated from urediospores of *P. melanocephala* carried to Australia in mid-1978 on the winds of the 1978 northwest monsoonal inflow from the Indian Ocean (5). Such an origin is possible, as are airborne spores from the Americas arriving in Australia prior to September 1978.

Germination and Infection

Germination occurs in either water drops or liquid water on germination substrates. Sugarcane rust is considered to be a warm temperature–high humidity disease. High humidity per se will not support germination of urediospores. The relationship of high humidity may be indirect in that at temperatures suitable for urediospore germination, the higher the relative humidity, the fewer degrees of temperature drop needed to reach the dew point. Liquid water as dew on plant surfaces most certainly will support urediospore germination.

Urediospores of *P. melanocephala* germinate over a wide temperature range, from 5 to 34 C, with an optimum of 25 C. Urediospores loose viability rapidly during hot weather (above 35 C) but may retain viability for 6 months or longer in closed glass containers stored at 4 C. Urediospores stored for a year and a half in liquid nitrogen remain viable. Teliospore germination occurs at 15–20 C, but basidiospores do not initiate infection in sugarcane. No alternate host is known for either *P. kuehnii* or *P. melanocephala*.

Urediospore content of air is highest during daytime hours because urediospores are produced during periods of leaf wetness, and as the leaves dry and winds increase, more urediospores become airborne. When urediospores are on leaf surfaces, germination and appressorium formation occur over the range of 5 to 30 C if liquid water is on the leaf surface for at least 6 hours (9). The optimal temperature range for appressorium formation is 15–30 C, and the percentages of germ tubes that produce appressoria at 5 and 10 C are low. Almost all appressoria develop over stomata on sugarcane leaves. The role of leaf wetness on infection needs to be evaluated in the field.

In Florida during February–May 1981, the sugarcane cultivar CP 63-588 was growing in a block of fields that included several where vegetables had been grown recently and would be grown again. Preparation of the fields for vegetable production included flooding controlled by pumping water out of or into drainage ditches around and within blocks of fields of muck soil. The high water table, very near or at times above the soil surface, in the sugarcane field provided high atmospheric humidity near the soil surface within the canopy of CP 63-588. Inoculum was abundant, temperature was satisfactory, leaf wetness was adequate—all factors supported an epidemic within that particular field. In another field one mile away, the same grower planted CP 63-588 from the same seed source during the same week that the rusted field was planted. The second field had a much lower water table, conditions not favorable for infection, and very little rust. The only difference between the two fields was in location of the water table, which affected leaf wetness.

Mature plant resistance has been suggested as one means by which seasonal sugarcane rust epidemics subside. However, how to distinguish mature plant resistance from the suppressive influence of increasing seasonal temperature on rust development has not been determined. Mature plant resistance is a likely occurrence. At the outbreak of rust in Jamaica, older fields of B 4362 seemed to escape infection (1). Certain sugarcane cultivars gain mature plant resistance slowly, 5 months after planting, whereas others develop resistance in only 2 months. Among susceptible cultivars planted each month in two different years at Canal Point, Florida, B 4362 seemed to develop mature plant resistance 6 months after planting, while some, such as CP 78-1735, failed to show resistance regardless of age. As summer temperatures increased at Canal Point, rust development was suppressed in all cultivars in both years.

The sequential development of *P. melanocephala* from urediospore germination through establishment of the fungus within the susceptible cultivar CL 41-223 and production of urediospores in urediosori (pustules) has been described (9). Urediospores germinate to produce a germ tube of variable length that contacts a stoma (guard cells) and forms an appressorium over the stomatal aperture. A penetration peg develops that enters the substomatal cavity, where a lobed substomal vesicle develops. Two or more, but usually four, infection hyphae develop. On contact with a mesophyll cell, the tip of an infection hypha is cut off

by a septum to form a haustorial mother cell. The mesophyll cell wall is penetrated, then the haustorium develops.

The haustorium of *P. melanocephala* differs from haustoria of other *Puccinia* spp. by being lobed, with several lobes arranged like fingers on a hand. The rust fungus grows parallel to the venation of the leaf and advances in both directions from the point of penetration. After 7 days, urediospores and paraphyses develop from the sporogenous hyphae, and subsequently the epidermis is ruptured by the developing urediospore mass.

In the rust-resistant cultivar CP 70-1133, a golden fluorescence develops around the point of penetration and in host cells adjacent to fungal structures. This host cell necrosis may be an incompatible interaction between host and pathogen. Rust development in CP 70-1133 is slower and less encompassing than in CL 41-223. Pustules have been observed in CP 70-1133 in controlled environments, but chlorotic flecks with necrotic centers are the common field symptoms of rust in this cultivar.

Developing Host Resistance

Withdrawing or withholding susceptible cultivars of sugarcane from cultivation is the only measure used for rust control (2). Implied, of course, is replacement with resistant cultivars.

The susceptible cultivars B 4362 in the Caribbean basin region, CL 41-223 in Florida, and Q 90 in Australia are being replaced. Rust resistance seems to be available, and resistant selections are being evaluated for agronomic acceptability. This should yield resistant cultivars to replace the susceptible ones. There is, however, a need to establish criteria for selection. For example, if pustules develop, will a cultivar be useful in the field in large populations? Must resistance equal immunity? And so on. It may be that cultivars known to support the development of a small number of pustules, such as CP 70-1133, will contribute to the development of new pathogenic races.

With concomitant introduction of rust-resistant cultivars, rust nurseries may be needed specifically for production of inoculum to maintain sugarcane breeding programs to screen for resistance to rust. Perhaps greater use may be made of detached leaf and shoot techniques to evaluate rust resistance under controlled environmental conditions. With near worldwide distribution of *P. melanocephala,* a vigorous pathogen, continued screening for rust resistance will be essential.

Exchange and use of exotic germ plasm most certainly can significantly advance the progress made in almost all cultivar development programs. The concern about moving seed pieces from one area to another should be alleviated to achieve maximal exchange of germ plasm. Workable quarantine procedures must be established to exchange cane. Perhaps exchange of buds grown in culture that are apparently free from certain diseases offers a potential solution to present restrictions. The future of protoplast isolation and culture holds great promise as a vehicle by which germ plasm might be exchanged. In addition, protoplast culture has tremendous potential to uncover variability and useful disease resistance for cultivar improvement.

To maximize benefits from germ plasm exchange, methods must be developed to exchange observed results in a form that is understood by the participants of the exchange as well as by other interested individuals. The stage of plant development when rust response is recorded offers the means to relate the plant growth stage with mature plant resistance. Whatever system is used for data recording, it must convey information accurately and in a form that can be understood by all.

Sugarcane cultivars grown today are interspecific hybrids of *Saccharum*, including *S. officinarum, S. spontaneum,*

S. robustum, *S. barberi*, and *S. sinensis*. There are many cultivar improvement programs, and released cultivars are designated by one or more letters indicating the improvement program's location, followed by a selection number. For example: B 4362 from Barbados; Co 775 from Coimbatore, India; CP 70-1133 from Canal Point, Florida; CL 41-223 from the U.S. Sugar Co., Clewiston, Florida; L 60-14 from Louisiana State University; Mex 57-473 from Mexico; NCo 310 from Natal Coimbatore; POJ 2878 from Java; PR 1000 from Puerto Rico; Q 90 from Queensland, Australia; and H 62-4671 from Hawaii.

The Future

We believe the greatest impact on production of sugarcane by *P. melanocephala* has already occurred, and as susceptible cultivars are replaced by cultivars possessing adequate resistance, losses caused by sugarcane rust will become negligible. Hybrids with resistance to rust have been developed, and selected crosses will make rust-resistant cultivars available for grower use. Susceptible cultivars, as well as those that support luxurious development of rust for short periods but seem damaged only slightly if at all, are being replaced.

Rust will remain a threat to sugarcane, however, and cane improvement programs must evaluate progeny for rust resistance. There may be a need to grow rust inoculum for progeny evaluation where naturally occurring rust fails to apply adequate disease pressure for satisfactory differentiation of resistance. At present, naturally occurring rust has aided selection of many resistant hybrids in almost all plant improvement programs. The future of sugarcane with respect to rust can be bright if the rust-resistant cultivars developed are planted by growers and if the rust inoculum used in cultivar selection represents the naturally occurring population with its variants.

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L. H. Purdy

Dr. Purdy is a professor in the Plant Pathology Department of the Institute of Food and Agricultural Sciences, University of Florida, Gainesville. He received a B.S. degree from San Diego State College and a Ph.D. degree from the University of California, Davis. He worked with smuts and stripe rust of wheat and other cereals with the USDA at Pullman, Washington; became chairman of the Plant Pathology Department at the University of Florida; and is now doing research with rust, smut, and other diseases of sugarcane, diseases of cacao, and *Sclerotinia sclerotiorum*.

Lii-Jang Liu

Dr. Liu is Director of International Programs in the Office of the Dean, College of Agricultural Sciences, University of Puerto Rico, Mayaguez. He has a B.S. degree from Taiwan Provincial Agricultural College, an M.S. degree from Oklahoma State University, and a Ph.D. degree in mycology and genetics from Purdue University. His research has dealt with diseases of tropical crops, sugarcane ratoon stunting and brown stripe diseases, resistance to *Helminthosporium*, sugarcane mosaic virus, sugarcane rust and smut, and downy mildew of sorghum.

J. L. Dean

Dr. Dean is a research plant pathologist with the USDA's Agricultural Research Service at the U.S. Sugarcane Field Station at Canal Point, Florida. He received his Ph.D. degree at Louisiana State University in 1965. His research deals with aspects of resistance to sugarcane diseases.