THE SUSCEPTIBILITY OF VARIETIES TO MOSAIC AND THE EFFECT OF PLANTING DATE ON MOSAIC INCIDENCE IN SOUTH AFRICA

By R. A. BAILEY and P. H. FOX

South African Sugar Association Experiment Station, Mount Edgecombe

Abstract

The majority of South African sugarcane varieties are resistant to mosaic but two widely grown varieties, NCo 376 and NCo 293, are highly susceptible. Application of Temik (aldicarb) to the soil at planting was not beneficial in controlling mosaic. Mosaic transmission was restricted to January to May but very rapid spread only occurred in mid January to March, i.e. late summer and early autumn. The susceptibility of sugarcane to infection varied considerably with plant development. Young plants of NCo 376 of approximately 6-12 weeks were much more susceptible than older plants. Plants of NCo 376 more than 16-20 weeks old were not severely infected, although very rapid spread occurred concurrently in younger plants. Differences of only 3 - 5 weeks in planting date exerted a marked effect on subsequent mosaic incidence. Planting susceptible varieties in early spring, thus avoiding the coincidence of a young, susceptible stage of growth with maximum vector activity in late summer and autumn, may enable crops to escape severe outbreaks of mosaic.

Introduction

Sugarcane mosaic virus in South Africa has fluctuated in importance since before 1920. The disease was probably introduced in varieties imported from the Argentine in 1914. It rapidly became common in the susceptible varieties then being grown, resulting in widespread planting of the resistant variety, Uba, which comprised 99,8% of production in 1924[13]. After Uba had been replaced by other varieties, mosaic reappeared on variety Co 281 in the Umzinto area on the South Coast in 1943 and by 1946-47 was very widely distributed in this variety, particularly in the area inland from Umzinto and between the Umdloti and Umvoti rivers on the North Coast[7, 9]. By 1952-53 mosaic occurred in all parts of the cane belt, largely through the planting of susceptible but tolerant varieties such as Co 281[7]. Variety NCo 339, also highly susceptible but tolerant, rapidly became widely contaminated after being released in 1952[8].

In 1955-56 mosaic was first recorded in NCo 376 and it was also shown that more than one strain of the virus occurred in South Africa[8]. By 1959-60 mosaic was reported to be spreading in NCo 376 in the by then traditional mosaic-infested area inland from Umzinto[14]. Isolated outbreaks of mosaic in NCo 376 occurred sporadically over the period 1960-1975, such as those at Eshowe in 1965 and Malelane in 1966[4]. However, severe outbreaks were largely restricted to the areas from Glen Rosa to Dumisa and from Mid-Illovo to Eston in the high altitude parts of the South Coast hinterland. Since 1975-76 there has been a noticeable increase in the frequency of severe outbreaks of mosaic occurring in NCo 376 and NCo 293 in a number of new localities. The disease is now an important problem at Melmoth and new severe outbreaks have occurred at Shongweni, Pietermaritzburg, Dalton and Nqabeni. Mosaic also appears to be spreading outside the areas of high mosaic incidence around Dumisa and Eston.

Mosaic is now widespread in NCo 376 and NCo 293 in the South Coast hinterland and Natal Midlands. Thomson[15] showed that these varieties were very intolerant of mosaic in small plot trials. Recent work (unpublished data) indicates that in severe outbreaks yields of NCo 376 can be reduced by more than 50%. The susceptible varieties NCo 376 and NCo 293 presently comprise approximately 75% of the annual crop in the cooler areas of cane production. The evidence that a severe strain of mosaic is spreading and becoming more firmly established indicates that the crop in the cooler production areas will be vulnerable to severe damage if this spread continues. The previous widespread incidence of mosaic in susceptible varieties on the North Coast, indicating that mosaic can also spread in this coastal environment, suggests that NCo 376 may be susceptible to damage from a severe strain of mosaic over a significant proportion of the industry.

It is well known that mosaic can only be controlled by the planting of resistant varieties once the disease reaches serious levels. There was therefore a need to determine accurately the relative susceptibility of NCo 376, NCo 293 and other released varieties to the severe strain(s) of mosaic in outbreak areas.

During surveys of mosaic incidence in recent seasons it was often noticed that the most severe outbreaks tended to occur in fields that had been planted or cut in summer. This indication that the rate of spread of mosaic varied with planting date was supported by variations in spread in different seasons reported from Louisiana and India[1, 3, 12]. In these countries very rapid spread of mosaic occurs during relatively short periods of the year[3, 12] associated with the times when vector activity is greatest[3]. There is, however, little information in the literature on the prospects of controlling mosaic by suitable choice of planting date so as to avoid infection. No information on the relative rates of mosaic transmission in different seasons or on the effect of planting date on mosaic incidence has been reported from South Africa.

Although foliar applications of systemic insecticides are known to have little effect on the spread of mosaic[3, 5, 6, 16], the effect of applications of aldicarb to the soil at planting has not previously been reported. This systemic insecticide, widely used for control of nematodes in South Africa, is probably effective in killing aphids on sugarcane for several months after planting.

Methods

The five field trials reported here were conducted during the period from 1977 to 1979 on two farms in areas where severe outbreaks of mosaic had occurred for many years. High levels of mosaic were known to occur on both farms. One trial site was at Dumisa, 20 kilometres inland from Umzinto on the Natal South Coast, at an altitude of 700 m. The second site was at Eston, approximately 45 km north of Dumisa and 45 kilometres inland from the coast, at an altitude of 900 m. The sites are in the high altitude zone of the

coastal hinterland and have a relatively short, cool growing season. The cropping cycle at both sites is approximately 24 months.

In 1977 a mosaic resistant evaluation trial and a trial to determine the effect of planting date on mosaic incidence were established on each farm. A further time of planting trial was planted at Dumisa in 1978.

The seedcane for all trials was obtained from mosaic-free plots at the Experiment Station, Mount Edgecombe. The percentage of buds germinated in the various treatments in all the trials was estimated approximately two months after planting.

Monthly rainfall totals from 1977 to 1979 were obtained from Dumisa and Eston. Mean monthly temperatures over this period were obtained from Powerscourt, at an altitude of 700 m in the South Coast hinterland.

Evaluation of varietal resistance

Fourteen varieties, NCo 376, N55/805, NCo 293, NCo 310, NCo 339, NCo 382, N52/219, N53/216, N6, N7, N8, N11, CB36/14 and J59/3 were evaluated in each resistance trial. These varieties included all widely grown and newly released varieties in South Africa at the time of planting. Each variety was represented by four single-row plots, 10 metres long, arranged in a randomised block design and with a spacing of 1,4 metres between rows. Each plot was planted with 20, 3-budded setts. Additional rows of NCo 293, NCo 339 and NCo 376 were treated with Temik 15G (15% aldicarb) at a rate of 20 kg/ha at the time of planting, in order to determine whether this systemic nematicide and insecticide had any effects on the incidence of mosaic. The rows treated with Temik were situated among the untreated rows, but were separated from them by guard rows of NCo 376. Guard rows of NCo 376 surrounded the trials.

The trial at Dumisa was planted in early November 1977 and that at Eston in mid December 1977. Counts of plants with mosaic symptoms were made at approximately monthly intervals in the plant crop from the first appearance of symptoms. In estimating mosaic incidence individual plants were regarded as infected with no regard given to the proportion of tillers or stalks with symptoms. Incidence was estimated as the percentage of plants that exhibited symptoms. The recording of mosaic incidence ceased after several months, when little further spread of mosaic was taking place and when it was difficult to distinguish individual plants in the row.

The trial at Dumisa was harvested in early December 1979, 25 months after planting. The Eston trial was not harvested because of severe lodging.

Time of planting trials

Trials to determine the effect of planting date on mosaic incidence in the plant crop were conducted at both farms in 1977-78. The variety used was NCo 376, known to be susceptible to mosaic in these areas as well as being the most widely grown variety. The trials at the two sites were conducted in an identical manner. Plots of cane were planted on each of five dates at intervals of approximately five weeks from late September 1977 to mid February 1978. The dates were September 20th, October 25th, November 29th, January 4th and February 14th, covering the period from spring to late summer.

Four plots were planted on each date. Plots consisted of six, 10 metre rows at a spacing of 1,4 m, with 20 3-budded setts planted in each row. The 20 plots were arranged in a randomised block design. Records of mosaic incidence were made from the centre four rows in each plot at monthly intervals from the first appearance of symptoms, as with the variety trials. Mosaic incidence was estimated as the percentage of plants that developed symptoms, again without regard to the severity of the disease on individual plants.

A second time of planting trial was conducted at Dumisa in 1978-79. Five planting dates were again used : September 6th, October 12th, November 14th, December 21st and January 24th. The trial was planted with 30 single-budded setts per row of 10 metres but in all other respects was conducted in a similar manner to the previous two trials.

Results

Evaluation of varietal resistance

At Dumisa, all varieties germinated satisfactorily, with the percentage of buds germinating varying from 50% to 81% and with a mean of 65%. Mosaic symptoms first appeared in early February 1978, 12 weeks after planting. Disease incidence reached a peak in May but declined slightly in June and July, when recording ceased (Figure 1). This decline was probably partly due to the increasing difficulty of identifying mosaic in the increasingly dense and tall canopy. However, sugarcane can 'recover' from mosaic by the disappearance of symptoms from the growing plant, as reviewed by Abbot[1]. Observations indicated that a real but slight disappearance of symptoms occurred with increasing plant development in four of the five trials (Figures 1 and 3).

At the Eston site a smaller proportion of buds germinated compared with the Dumisa trial, ranging from 30% – 52% buds germinated and with a mean of 40%. At Eston symtoms first appeared in early March, also at 12 weeks after planting and again reached a peak in June (Figure 1). The period of most rapid spread was in February and March at Dumisa and in March and April at Eston.

The highest level of mosaic in the different varieties was estimated from the mean of the maximum proportion of infected plants occurring in individual plots during the trial (Table 1). Susceptibility to mosaic varied considerably

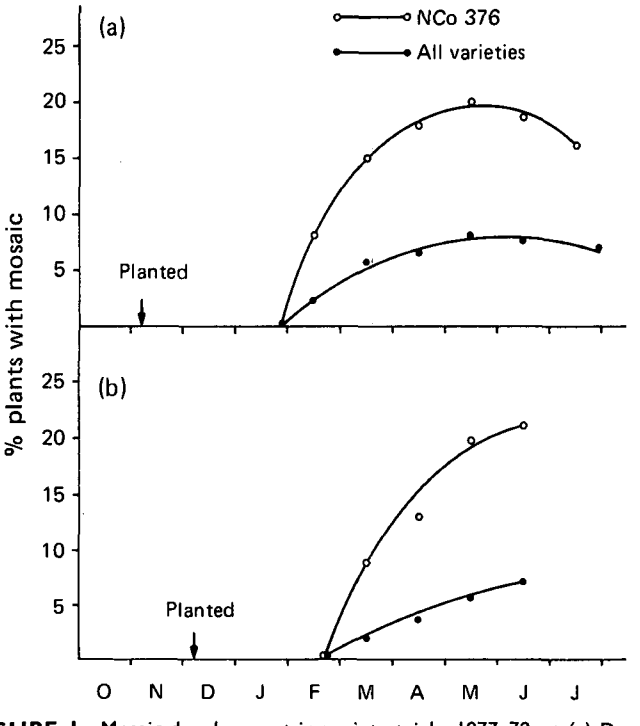

FIGURE 1 Mosaic development in variety trials, 1977–78, at (*a*) Dumisa and (*b*) Eston.

among the varieties. At Dumisa, NCo 293, NCo 376 and NCo 339 contained the highest levels of mosaic, with 22% – 26% of plants having symptoms. The differences in mosaic incidence between these three and all other varieties were highly significant. Varieties NCo 310 and N55/805 contained moderate levels of mosaic, 7% and 12% infected plants respectively, while all other varieties contained no mosaic or less than 3% infected plants. N55/805 contained significantly more mosaic than the nine varieties with low levels of infection.

At Eston the mean incidence of mosaic was slightly lower than at Dumisa (means of 4,8% and 7,0% respectively in the plots without Temik). Varieties NCo 376, NCo 339, and NCo 293, together with NCo 310, contained significantly more mosaic than all other varieties, having from 13% to 18% infected plants (Table 1). In this trial N55/805 again contained an intermediate proportion of infected plants, 4%, but not significantly more than in the varieties with little or no mosaic.

With the exception of NCo 310, the relative severity of mosaic infection in the different varieties was very similar in the two trials.

TABLE 1

Mosaic incidence in 14 South African sugarcane varieties

Variety	Dumisa			Eston	
	Cane yield (tons/ha)	% plants with mosaic	Rank	% plants with mosaic	Rank
NCo 376 . .	144	22,5	13	17,8	14
NCo 339 . .	144	25,5	14	14,3	13
NCo 293 . .	161	22,3	12	12,8	11
NCo 310 . .	136	7,3	10	13,0	12
N55/805 . .	119	11,7	11	4,0	10
N53/216 . .	115	0,5	3	2,5	9
N7 . . .	118	2,5	9	0	1
NCo 382 . .	165	1,3	6	1,3	8
N52/219 . .	114	0,8	4	1,0	7
N6 . . .	117	1,3	6	0	1
N8 . . .	136	1,3	6	0	1
J59/3 . . .	83	0,8	4	0	1
N11 . . .	135	0	1	0	1
CB36/14 . .	171	0	1	0	1
LSD P=0,05 .	37	7,0	—	7,8	—
P=0,01 .	—	9,3	—	10,5	—

The majority of plots of NCo 376, NCo 339 and NCo 293 that were treated with Temik at planting contained slightly more mosaic than the untreated plots, although the mean increase in incidence was not significant at P = 0,05 (Table 2). In both trials the increase in mosaic incidence following treatment with Temik was greatest in NCo 376 and at Eston the difference in the case of NCo 376 was statistically significant.

In the Dumisa trial all plots of NCo 376, NCo 339 and NCo 293 treated with Temik produced higher cane yields than untreated plots, although the differences were not significant at P = 0,05. The soil at the Dumisa site was a light sandy loam, derived from Table Mountain Sandstone and it is possible that the slightly higher yields in the plots treated with Temik were a result of nematode control.

Cane yields at Dumisa varied considerably among the varieties, with a range from 83 to 180 tons cane per hectare (Tables 1 and 2). Both NCo 293 and NCo 376 were among the highest yielding varieties, despite a possible slight loss in yield due to mosaic. Of the resistant varieties, CB36/14 and

TABLE 2

Effect of Temik on cane yield and mosaic incidence

Variety	Dumisa				Eston	
	Yield (tons cane/ha)		% plants with mosaic		% plants with mosaic	
	Temik	No Temik	Temik	No Temik	Temik	No Temik
NCo 376 . . .	147	144	27,3	22,5	28,0	17,8
NCo 339 . . .	171	144	21,5	25,5	15,3	14,3
NCo 293 . . .	180	161	24,3	22,3	9,3	12,8
LSD (P=0,05) .	37		7,0		7,8	
Means . . .	166	150	24,4	23,4	17,5	15,0
LSD (P=0,05) between means	22		4,0		4,5	

NCo 382 produced the highest yields but most of the resistant varieties were inferior to NCo 376 and NCo 293. It should be noted that this trial was not designed to provide accurate information on the yielding ability of varieties.

Time of planting trials

In the trial conducted at Dumisa in 1977-78 the proportion of buds germinated varied with planting date, being lowest (31%) in the February and highest (63%) in the November planted plots, but was generally satisfactory (mean 46%). Mosaic symptoms appeared simultaneously in early February in the plots that had been planted in September, October and November, i.e. approximately 21, 16 and 11 weeks after planting respectively. In the January and February planted plots mosaic first appeared in March and April, at 10 and 8 weeks after planting. The proportion of plants with mosaic symptoms and the rate of symptom appearance increased progressively with planting date from September to late November and then progressively declined in the plots planted later (Figure 2b). The rate of symptom appearance and the proportion of plants infected were much greater in the plots planted in late November than in the other plots. In these most severely infested plots the level of infection increased very rapidly in the period from February to mid April, until 66% of plants were infected. Comparatively little mosaic and a slow rate of spread occurred in the plots planted earliest and latest. The highest levels of mosaic were recorded in May.

In the Eston trial the rate of germination was similar to that in the Dumisa trial, ranging from 39% buds germinated (February planted plots) to 58% (November plots) and with a mean of 48%. At Eston (Figure 2a) the general pattern and severity of mosaic occurrence was very similar to that in the trial at Dumisa. However, symptoms appeared several weeks earlier at Eston, in early January. The intervals between planting and the first appearance of symptoms were approximately 16, 9 and 6 weeks after the first three planting dates respectively and approximately 6 weeks after the last two planting dates. The incidence of mosaic and the rate of symptom appearance again increased progressively with planting date up to late November and again declined in the later planted plots. However, at Eston the period of most rapid spread also occurred somewhat earlier compared with the Dumisa trial, from mid January to late March, following the trend of earlier appearance of the disease. More mosaic also occurred in the plots planted in January in this trial.

Excellent germination occurred in the third time of planting trial, conducted at Dumisa in 1978-79. The proportion of

buds germinated varied from 86% in the September plots to 39% in the January plots, with a mean of 62%.

Symptoms first appeared in late January in the plots planted in early September, mid October and mid November, i.e. 19, 14 and 9 weeks after planting respectively (Figure 2c). As in the other trials, symptoms first appeared simultaneously in the plots planted on the three earliest dates.

Very high levels of mosaic, such as occurred in the plots planted in late November in both trials in 1977-78, did not occur in this third trial. With this exception, the general pattern of mosaic development was similar to that in the previous season's trials and was also similar to the development of mosaic in NCo 376 in the variety trials in 1977-78. In this 1978-79 trial similar proportions of plants were infected compared with the two earlier and two later planting dates in 1977-78 and the slowest rates of symptom appearance and the lowest levels of mosaic again occurred in the first (early September) and last (late January) planted plots. Spread of mosaic again occurred most rapidly in February and March and the highest levels of mosaic occurred in May and June.

Figure 3 illustrates the rates of spread of mosaic (means of the proportion of plants with symptoms in established plots at the various planting dates) in relation to temperature and rainfall. Rapid transmission in both seasons occurred largely from mid January to March, towards the end of the period of maximum mean temperatures and after the months of maximum rainfall.

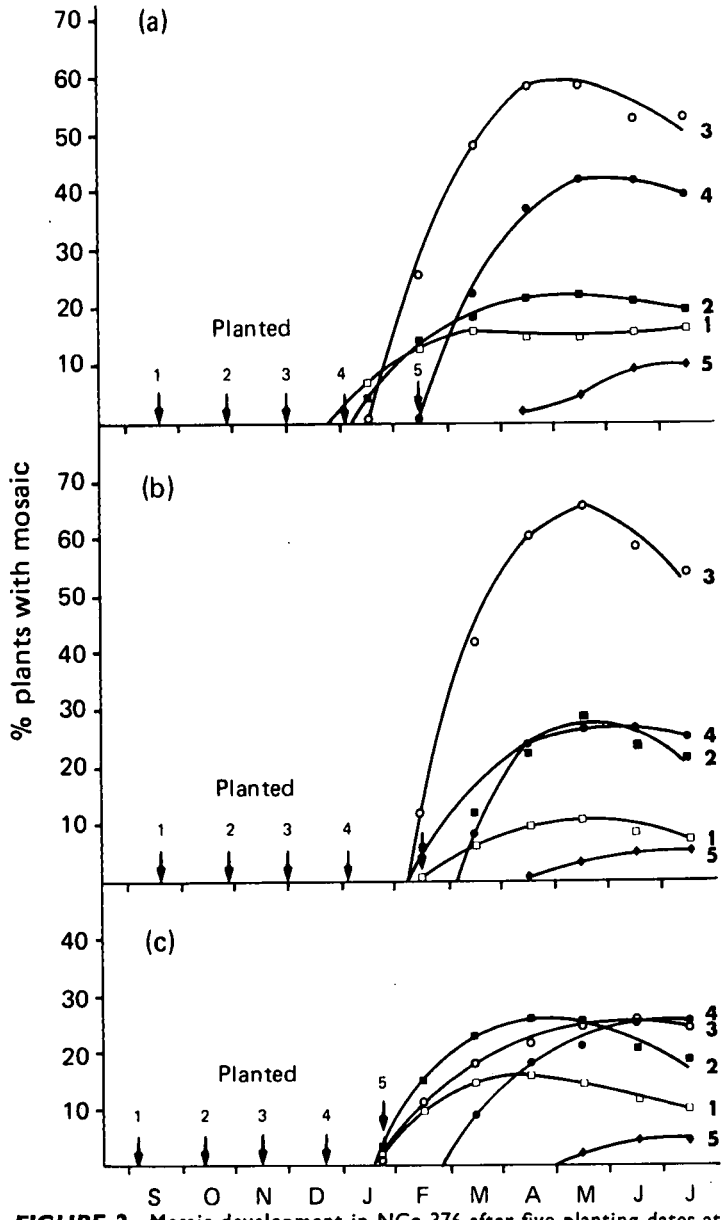

FIGURE 2 Mosaic development in NCo 376 after five planting dates at (*a*) Eston and (*b*) Dumisa in 1977–78, and (c) Dumisa in 1978–79. Planting dates shown by arrows.

Discussion

The ranking of varieties according to susceptibility to mosaic was very similar in both variety trials, the only significant difference being that NCo 310 developed relatively more mosaic in the trial at Eston. This differential reaction of NCo 310 compared with the other varieties could be due to there being a difference in the strain or strains of the virus at the two sites. However, the very similar ranking of all other varieties at both sites is evidence against such a hypothesis.

The variety trials indicate that currently released varieties in South Africa can be grouped into four broad categories of resistance (Table 3). Varieties NCo 376 and NCo 293 are clearly the most susceptible of current released varieties when grown in areas where mosaic occurs, typified by the Dumisa and Eston trial sites. NCo 376 and NCo 293 proved to be as susceptible as NCo 339, this latter variety long having been regarded as highly susceptible in all areas. NCo 339 was recently removed from the list of released varieties that can be grown in South Africa, its susceptibility and the inconspicuous nature of the symptoms often making NCo 339 an unsuspected source of mosaic for the infection of more valuable varieties.

TABLE 3

Resistance of South African sugarcane varieties to mosaic

Variety	% plants infected (means of trials at Dumisa and Eston)	Resistance rating (1=immune, 9=very highly susceptible)	Resistance category
NCo 376	20,2	8	Highly susceptible
NCo 339*	19,9	8	
NCo 293	17,6	8	
NCo 310	10,2	6	Moderately susceptible
N55/805	7,9	5	
N12**	—	4	Resistant
N53/216	1,5	3	
N7	1,3	3	
NCo 382	1,3	3	
N52/219	0,9	2	Highly resistant
N6	0,7	2	
N8	0,7	2	
J59/3	0,4	2	
N11	0	1	
CB 36/14	0	1	

* No longer a released variety in South Africa.
** Rating based on other observations.

The cooler and higher altitude areas that are heavily dependent on NCo 376 and NCo 293 are clearly vulnerable to further spread of mosaic. The increased frequency of severe outbreaks in these susceptible and intolerant varieties in recent seasons indicates that further spread outside the presently affected areas should be expected.

The highly susceptible reaction of NCo 376 in outbreak areas in the higher altitude areas of the coastal hinterland and Midlands differs from its reaction at the coast. In resistance trails at Mount Edgecombe NCo 376 develops little mosaic compared with NCo 339. This differential reaction indicates that there may be different strains of mosaic at the

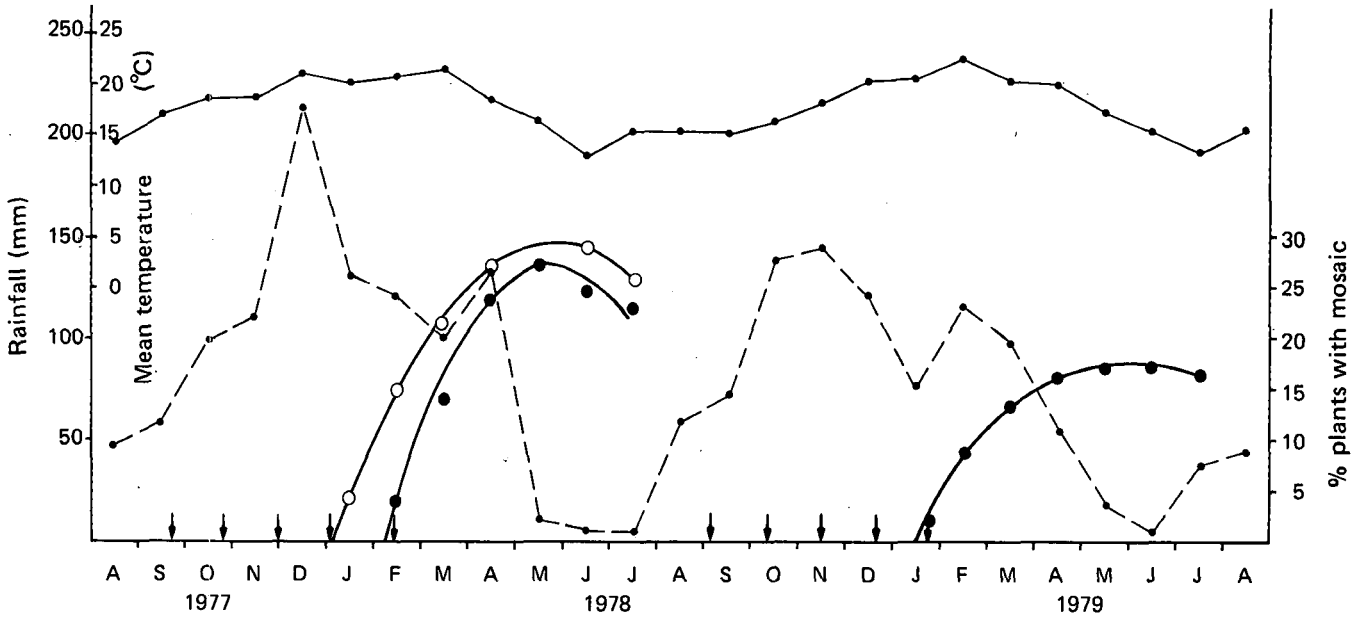

FIGURE 3 Mean incidence of mosaic in NCo 376 after five dates of planting at Dumisa and Eston, in relation to temperature (●——●) and rainfall (●– – –●) in 1977–79 (planting dates shown by arrows; mosaic incidence: ●——● Dumisa, o——o Eston).

coast compared to the high altitude areas. Alternatively, a differential rate of spread in these varieties may occur under coastal conditions.

Although it is generally considered that the warmer areas of the cane belt are not favourable for mosaic development, the extent of the disease in older varieties such as Co 281 on the North Coast must not be forgotten. The spread of a new, more virulent strain of mosaic into the main coastal areas of production could present a very serious hazard to NCo 376. Investigations into the extent to which different strains of mosaic virus occur in South Africa and into the suitability of different areas for the spread of mosaic are in progress.

Although the variety trial at Dumisa was not designed to provide accurate information on the productivity of the different varieties, the yield data illustrate the dilemma faced by growers in areas where mosaic is a problem. Many of the resistant varieties, such as J59/3, N52/219, N6 and N55/805, are either less productive than the favoured but susceptible varieties under these growing conditions or are susceptible to other diseases. However, all varieties released in South Africa in the last decade have a high measure of mosaic resistance. It is in the continuation of this policy of breeding for mosaic resistance that the long term answer to the mosaic problem will be found. Variety N12, released in 1979 as being suited to the high altitude areas, may prove to be a useful alternative to NCo 376 and NCo 293.

The failure of Temik to control mosaic in sugarcane was not unexpected. This systemic insecticide probably kills the aphid vectors that spread mosaic when they feed on highly susceptible young cane. However, mosaic is a non-persistent virus and transmission must occur before the aphids are killed. Temik evidently is as ineffective as foliar applied insecticides in controlling mosaic.

Rather than mosaic being controlled by Temik applications, the data in Table 2 indicate that more mosaic may have developed in NCo 376 when Temik was applied. Such an effect could be explained by the fact that rapidly growing cane can be more susceptible to infection[1], this phenomenon probably being related to a greater attractiveness of such cane to the aphids. The slightly greater (although not significantly different) yields in the Temik-treated plots indicate that a positive growth response from Temik did occur.

That mosaic spreads more rapidly at certain times of the year in South Africa is perhaps to be expected, in view of the reports of such a pattern from other countries[1, 3, 12]. If the time and rate of spread is judged from the appearance of symptoms, the most rapid spread in both 1977-78 and 1978-79 occurred in mid January to March, or late summer to early autumn. The period of most rapid transmission probably included early January, if a latent period of approximately two weeks before the appearance of symptoms following infection is accepted[1].

The period when mosaic was spreading rapidly in is apparently related to a number of factors. As mosaic is spread by aphids, these factors must include changes in the activities of infective vectors. It appears from Figures 2 and 3 that peak populations of vectors occur in summer and autumn, or at least that high numbers of infective vectors do not occur before summer.

The period when mosaic was spreading in sugarcane in both seasons was comparatively short, some 10-12 weeks in late summer and early autumn. This period occurred after the months of maximum rainfall in both seasons (Figure 3). Aphid populations are known to increase in dry weather and it is possible that rainfall distribution in summer is an important factor affecting the transmission of mosaic.

At least some slight spread of mosaic would occur in spring and early summer even if only low numbers of infective vectors were present. That no spread of mosaic occurred in this period indicates that no vectors were present, which seems unlikely[11, 17], or that suitable infected host plants, constituting an effective source of the virus, were rare. Infected maturing cane, which was abundant at both trial sites in both seasons, apparently was not an effective source of the virus from which further spread could take place in spring. The virus is known to be transmitted more readily to and from other hosts, such as maize, than is the case with sugarcane[9, 10]. Very high populations of vectors of mosaic can build up in other species[2]. These other hosts probably include grasses, of which several species in South Africa are known to be hosts of mosaic. It is possible that the lack of spread of

mosaic in spring and early summer was partly due to there being few other infected host plants at a suitable stage on which infective aphid populations could develop. The role of annual grass weeds and maize crops, therefore, may be an important factor in the development of mosaic outbreaks in sugarcane in South Africa, where low temperatures and dry conditions occur in the winter months (Figure 3). A preliminary build up of the virus as well as of the aphids on these annual host plants may be a prerequisite before the annual spread in sugarcane can occur.

The marked effect of slight changes in planting date on mosaic incidence is both interesting and significant. In both seasons the early planted plots did not develop mosaic for many weeks after planting, between 16 and 21 weeks in the case of the plots planted in September. Symptoms appeared more rapidly with successive planting dates, but in no case was the period less than six weeks after planting. The approximately simultaneous appearance of symptoms in the plots planted in September, October and November in all three trials indicates that the first flights of infective vectors into sugarcane occurred in January.

Mosaic incidence in the early planted plots, that escaped infection for several months after planting, always remained low, despite being in close proximity to very high levels of infection in more severely infected, later planted plots and in the surrounding cane. This indicates that sugarcane is most susceptible to infection in the early weeks of crop development, up to approximately 12 weeks after planting, and that older plants tend to escape infection by being less attractive to the vectors. By planting fields early, it appears that very severe outbreaks of mosaic can simply be avoided.

A similar escape of young ratooning crops may occur if fields of susceptible varieties are cut early rather than very late in the milling season. The low level of mosaic in the plots planted late in both seasons, when young plants were in proximity to abundant sources of the virus, was probably a result of a decline in vector populations after approximately mid April.

The maximum levels of mosaic in the worst affected plots differed considerably between the two seasons. The very rapid and severe spread of the disease in the plots planted in late November 1977, more than 60% of plants infected, is similar to that seen in many severe outbreaks in commercial fields of NCo 376 and NCo 293. It seems that very rapid spread of mosaic in susceptible varieties is dependent on the coincidence of a number of factors. These appear to include the presence of young cane, at a stage when it is highly attractive to aphids; rapid growth of this young cane under good growing conditions; high populations of vectors, probably markedly influenced by weather conditions and by the presence of host plants other than sugarcane and, fourthly, adequate sources of the virus, this latter at least initially in the form of infected graminaceous weeds and crops other than sugarcane. The results in 1977-78 indicate that very high populations of vectors in that season occurred for only short periods.

The less severe development of mosaic in NCo 376 in the varietal resistance trials compared with the very severe levels in the plots planted in late November on both farms is further evidence that very rapid transmission of mosaic is highly dependent on a combination of specific circumstances. At Dumisa the two trials were adjacent but the variety trial was planted three weeks before the most severely infected plots in the time of planting trial. In the former trial at Dumisa 23% of plants of NCo 376 developed mosaic compared with 66% in the latter trial. At Eston the two trials were separated by several kilometres and the incidence of mosaic in surrounding commercial cane fields was much lower at the variety trial site.

The marked effect of a difference in planting date of as little as three to five weeks on the subsequent severity of mosaic indicates that young cane plants are highly favourable for aphid colonisation and transmission of the virus for only a limited period.

The high levels of mosaic that occurred in the susceptible varieties in all five trials demonstrate that control measures such as the planting of healthy seedcane and the roguing of infected plants from fields are likely to be ineffective when such varieties are planted in mid-summer in areas where mosaic occurs commonly. Such measures, however, are important in minimising the spread of mosaic into areas that are not yet severely contaminated with the virus.

Conclusions

The two most popular varieties grown in areas where mosaic is spreading, NCo 376 and NCo 293, are highly susceptible to the disease. Many of the varieties that are resistant or only moderately susceptible are not adapted to the conditions in the cool areas where mosaic is often serious, or are susceptible to other diseases.

Temik had no beneficial effect on the control of mosaic in three varieties when grown in small plots.

Transmission of mosaic occurred from January to May, with the most rapid spread taking place in mid January to March, i.e. late summer and early autumn. A high level of vector activity at this time may have been associated with the onset of drier conditions after the main summer rains.

Infected sugarcane appears to be a poor source of mosaic for further infection. Other graminaceous species are probably the most important sources of the virus.

Sugarcane is most susceptible to infection when at a young stage of growth. In this investigation the period of maximum susceptibility occurred between 6 - 12 weeks after planting. The period of high susceptibility appears to be short. Differences of only three to five weeks in planting date had a very marked effect on subsequent mosaic incidence. Very rapid spread of mosaic, with the majority of plants becoming infected in only a few weeks, is probably caused by the coincidence of a highly susceptible stage of growth with maximum vector activity. Plants of NCo 376 older than 16 - 20 weeks did not become severely infected, even during periods of high vector activity. High populations of vectors did not occur in young sugarcane crops in late autumn and winter.

Selection of planting and cutting dates so as to avoid the coincidence of an early, highly susceptible stage of growth of susceptible varieties with high populations of vectors appears to be a useful technique for escaping very severe damage from mosaic. In spring this would mean planting fields before approximately the middle of October and preferably as early as possible, conditional on satisfactory germination.

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