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Inheritance of Resistance in Sugarcane to Mosaic and Red Rot Diseases.

Youssef Elsedik Azab

Louisiana State University and Agricultural & Mechanical College

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INHERITANCE OF RESISTANCE IN SUGARCANE
TO MOSAIC AND RED ROT DISEASES

A Thesis

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

in

The Department of Botany, Bacteriology and
Plant Pathology

by

Youssef Elsedik Azab
B.Sc. Fuad First University, 1944
M.Sc. University of California, 1949
June, 1952
MANUSCRIPT THESSES

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ABSTRACT

The mosaic and red rot diseases have been responsible for crop failures in Louisiana and many sugarcane growing countries. Disease resistance is one of the major objectives in breeding favorable sugarcane varieties in Louisiana. Studies on the inheritance of resistance to these two diseases are reported here. They were conducted with the hope that they would yield information that would be of value to the plant breeder.

Inoculations of 71,769 sugarcane seedlings from 175 crosses with sugarcane mosaic virus were made in the greenhouse. The spindles were rubbed with a mixture of carborundum and the mosaic virus after the seedlings were transplanted from flats to pots. The seedlings were later classified as to whether or not they showed mosaic symptoms. Symptomless plants were transplanted to the field and examined later for mosaic symptoms.

The percentages of plants showing mosaic symptoms in the progeny of crosses involving resistant x resistant, susceptible x susceptible, and resistant x susceptible parents ranged from zero to 87.9, 2.6 to 35.6 and 0.8 to 94.5 per cent respectively. When some of the mosaic resistant varieties were used as parents in different crosses, a high percentage of mosaic-susceptible seedlings was obtained among the progeny; in other varieties a high percentage of resistant seedlings was obtained, and still in other varieties the percentage of mosaic susceptible seedlings among the progeny in a given cross varied according to the other parent in the cross.

Most current sugarcane varieties possess at least a complete complement of *Saccharum officinarum* chromosomes plus a few chromosomes
of *S. spontaneum*. The literature indicates that the genetic factors for
resistance to mosaic and those for resistance to red rot diseases are
carried on the *S. spontaneum* chromosomes. However, the nature and
number of those factors were unknown.

The hypotheses is advanced that inheritance of resistance to
mosaic in sugarcane is conditioned by dominant complementary genes.
A group of minor modifying factors is thought to affect the degree of
expression of the disease. Irregularities in behavior of *S. spontaneum*
chromosomes during meiosis in sugarcane varieties also influence
the transmission of resistance from the parents to their progenies.

The flat inoculation technique, in which the seedlings were
sprayed with the mosaic virus and then clipped off immediately after
spraying was compared with the pot inoculation technique described
earlier. About 20,282 seedlings obtained from 18 crosses were involved.
In 12 crosses a much higher percentage of mosaic-susceptible seedlings
was obtained by using the flat technique than by using the pot technique.
In the other crosses there was almost no difference in the results obtained.
Although the flat technique was more rapid, by this method, many seed-
lings which would be resistant to mosaic in the field were unnecessarily
discarded and potentially desirable seedlings might thus be eliminated.
The data also suggest that among the seedlings there were two types of
resistance to mosaic, and that they were genetically controlled. The
period of incubation of the mosaic disease varied from four days to seven
weeks and perhaps more.

Studies on recovery from mosaic symptoms were made with 1470
seedlings and it was found that 19 per cent of the seedlings recovered.
Recovery ranged from zero in the progeny of a few crosses to 51.6 per
cent in one cross. The results suggested that the recovery from mosaic symptoms in sugarcane was apparently a genetic character.

The red rot studies were made by inoculating the progenies of 14 crosses involving resistant x resistant, resistant x susceptible and susceptible x susceptible parents with spore suspensions of the red rot fungus Physalospora tucumanensis Speg. In all types of crosses, there were plants that varied in their degree of resistance or susceptibility to this disease. In the crosses of susceptible x susceptible parents, the percentages of resistant plants obtained among the progeny were more than those obtained in certain crosses of resistant x resistant parents. In other cases, the percentages of commercially resistant seedlings obtained were almost equal regardless of the type of cross made. It did not appear possible to predict the value of a variety as a source of resistance to red rot from its phenotype.

The hypothesis is advanced that resistance in sugarcane to the red rot disease is governed by one or a few dominant genes for resistance from S. spontaneum plus a dominant inhibitor gene from S. officinarum. This inhibitor gene is thought to mask the effect of the genes for resistance from S. spontaneum. A group of minor modifying factors may also influence the degree of expression of the disease. The irregular chromosomal behavior occurring in sugarcane varieties during meiosis influences the ratios of resistant to susceptible plants obtained in the progenies. The observations made suggest that the genes governing resistance in the nodes are different from those governing the resistance of the internodal tissues.

It is concluded that a sugarcane variety need not be discarded
as a potential parent in breeding because it is susceptible to mosaic
and/or red rot. Resistance to disease in a sugarcane variety is not a
proof that it will be valuable as a source of resistance in a cane breeding
program.
INTRODUCTION

Genetic studies in sugarcane have lagged behind many other phases of study on this crop. Many reasons have been given in the literature to account for this; among those mentioned were the difficulty of controlling flowering in sugarcane. A given variety may flower one year and may not flower the following year. In addition, sterility in many of the varieties prevented selfing or intercrossing of these varieties. However, some investigators have conducted studies on the inheritance of several characters of sugarcane. Unfortunately, they frequently obtained data which did not fit simple Mendelian ratios. This led to pronouncements which later developed into the common belief that the inheritance of characters in sugarcane apparently did not follow known genetic rules. This appears to be one of the major factors responsible for the dearth of investigations, as well as the deemphasis of genetic studies in sugarcane.

Sugarcane is a polyploid plant and the varieties occurring in nature have chromosome numbers varying from 80 (n = 40) to 128 or more. The basic chromosome number of the genus Saccharum is believed to be 10 by some investigators and 5 by others. Further, many of the sugarcane varieties which were used in genetic studies by early investigators were hybrids between two or more species of the genus Saccharum. These two facts together would account to a great extent for the complicated data obtained in the early genetic studies. An awareness of these facts, coupled with a knowledge of the recent developments in the fields of genetics, cytology, and cytogenetics should make it possible
to place some interpretation on the data obtained from studies of inheritance of various characters in sugarcane.

The prevailing belief that genetic rules do not hold in sugarcane studies is no longer valid since we are now aware of the fact that sugarcane is a highly polyploid plant, and that most of the varieties studied are products of interspecific hybridization. These facts led to a demand that extensive studies on the inheritance of economic characters of sugarcane be conducted. Such studies undertaken, with these points in mind, have been conducted on two important diseases of sugarcane; namely, mosaic and red rot. These studies are reported here.

The mosaic disease has caused considerable damage to sugarcane in Louisiana and many other cane growing areas. Red rot is also a major disease of sugarcane. Because these two diseases and the root rot disease were mainly responsible for crop failures in Louisiana, breeding for disease resistance became the major objective in the sugarcane breeding program conducted by the United States Department of Agriculture.

The first step in this program was to inoculate the seedlings obtained annually with sugarcane mosaic virus. The seedlings that showed mosaic symptoms were discarded. Later, the selected seedlings were usually tested for resistance to red rot disease caused by \textit{Physalospora tucumanensis}, Speg.

The Louisiana Agricultural Experiment Station started its breeding program on sugarcane on a large scale in 1949, and among its major objectives was the incorporation of resistance to mosaic and red rot diseases in agronomically good varieties. A knowledge of the inheritance of resistance to these two diseases in sugarcane became a prerequisite to the establishment of such a breeding program.

In breeding for disease resistance many factors have to be
considered among which are: 1) the mode of inheritance of resistance to the disease; 2) the number of genes conditioning resistance and the nature of these genes; 3) the genetics of the pathogen; 4) linkage studies and 5) the history of the material under study.

A large number of seedlings, approximately 50,000 to 80,000, had to be inoculated each year with sugarcane mosaic virus. The method used involved the inoculation of each seedling by rubbing it with the fingers, using carborandum size 220 as an abrasive. It soon became apparent that this method involved the expenditure of a large amount of time and effort, so an investigation was undertaken to develop an inoculation technique which would be more desirable. The results of these studies are reported here.

Recovery from mosaic symptoms in sugarcane is a known phenomenon. The opinion has been expressed by other investigators (347), that such a phenomenon should be incorporated in the sugarcane breeding program, but no work has been done on this subject. Further, there is a belief among some plant pathologists that good quality of sugarcane plants is connected with general susceptibility to disease. The existence of these opinions called for studies on the recovery from mosaic symptoms in the progeny of certain crosses. The studies reported here were made with the hope that they would provide the first step for further genetic studies on this phenomenon, and furnish information that would be of value to the plant breeder.
REVIEW OF LITERATURE

A large number of papers have been published dealing with mosaic and red rot disease of sugarcane. Only the important papers related to the subjects discussed here are reviewed in the following paragraphs:

Considering the history of the mosaic disease of sugarcane, Brandes 1919 (51), and Dastur 1923 (114), state that the disease was first described from Java in 1890. Lyon (229), Brandes and Matz 1935 (64), Hes 1950 (187), Summers et al. 1948 (347), reported that the first publication on the disease was by Van Musschenbrock in 1892 (249), who described the disease in Java under the name "Gele Stipenziekte", or yellow stripe disease. In April 1893, Wakker published further notes on the disease (229), and in July of the same year Arendsen Hein (32) contributed a short article on the subject, which was illustrated by a multicolor plate showing a portion of a sugarcane leaf affected with the disease (229), (64), (243).

In Java, Wakker and Weunt in 1898 (367) as reported by Lyon (229), fully described the disease, and in the following year Kruger briefly described it in his book, "Das Zuckerrohr und seine Kultur". Wilbrink and Ledebor in 1910 (374), gave a thorough treatment of the disease. They carefully described it and presented excellent colored plates (229). Hes 1950 (187) summarized the history of research on mosaic disease of sugarcane in Java.

The disease was then reported from other countries. Brandes (51) pointed out that Dutch investigators reported the presence of yellow
stripe in Egypt in 1909 on cane imported from Java. Lyon (229) indicated that it was first noted in Hawaii in 1908. Lee and Kopke (217) pointed out that it was first observed in the Philippines by Dr. Lyon in 1910 or 1911.

In Puerto Rico, Chardon (87), (89) stated that the mosaic disease of sugarcane was first reported there by Stevenson in 1915 and that by 1919 it covered almost all the island. Dodds 1932 (125), stated that the mosaic originated in the North Coast Hills of Puerto Rico and from there spread rapidly until the establishment of the Uba cane which was found to be immune in 1918, and distributed for general cultivation in 1924.

Stevenson published his first reports on mosaic in Puerto Rico in 1917 (326), (328) and followed it by another detailed publication on the disease (327) in the same year and announced that the disease was known there as the mottling disease of sugarcane. The disease attracted worldwide attention following the publication of Stevenson’s paper (327). Lyon in Hawaii, as reported by Colon (103) (translation) became particularly interested in the subject, and regarded this disease as identical with the disease known as gene strepenziekte, or yellow stripe disease, in Java, Hawaii, and elsewhere. The yellow stripe disease had been studied before by Lyon in Hawaii from 1911 - 1914 (228).

Earle was commissioned by the United States Department of Agriculture to go to Puerto Rico and investigate the disease, where he did remarkable work (89). The identity of the disease causing mottling of sugarcane in Puerto Rico with the gene strepenziekte of Java and the yellow stripe disease of Hawaii was finally recognized by the investigators of this subject in Puerto Rico (51, 103, 133, 241, 329).

In the United States, Edgerton as well as Brandes, announced
the occurrence of mosaic on sugarcane in 1919. Disputes have arisen as to whom the credit should be given for first reporting the disease. According to Edgerton et al 1924 (156) it is clear that Edgerton was the first to report it in June 21, 1919 (142), following the receipt of a letter from Puerto Rico in which it was stated that cane received from Louisiana had developed the disease. This was followed by another publication by Edgerton that came out in September 1919. The bulletin by Brandes (51) was issued on October 29, 1919, in which he recorded also the presence of the disease in Florida, Georgia, Alabama and Mississippi.

Edgerton, 1919 (143) stated that the disease had been in Louisiana for a number of years, but it was not recognized as mosaic. Brandes 1919 (51) inferred that the disease had been present in Louisiana for a number of years prior to 1914. Rands and Abbott 1939 (282), and Summers et al 1948 (347) summarized the results of research on mosaic disease of sugarcane in the United States.

In Cuba, Brumer (79) stated that the mosaic was first observed there in 1915. Johnston (202) stated that the disease was first publicly reported as occurring in Cuba by him in 1919. However, he indicated that it had been noted previously by L. D. Larson who attached little importance to it.

In Trinidad, Williams (375) reported that mosaic was found there on sugarcane in 1920. Nowell (268) stated that in 1921 there were 1700 acres in the infected district, of which at least 1,000 acres had been worked over in an effort to control the disease. In Barbados, Bovell, as reported by Johnston (202), stated that the disease was found in October of 1920, and by 1923, in spite of efforts to control, it has spread over the
whole island. Johnston (202), stated that the disease was present in Surinam, and that it was reported by Stevenson in Santo Domingo. Martyn 1946 (238) stated that mosaic was first reported in Jamaica in 1920 by Dr. Lyon while visiting the island.

In South Africa, Storey, 1923 (332) and 1924 (333) claimed that he was the first to find the mosaic on sugarcane in Natal, upon his arriving there in 1922. He expressed the belief that the disease had been introduced into Natal from Argentina in 1914. Shepperd in 1924 (305), (306), reported the mosaic to be widespread in Réunion.

In Brazil, mosaic was first introduced in 1920 (202). Abbott (2), (3), stated that the disease was first introduced in Peru (where it was first seen around 1928) from Argentina. Fawcett 1924 (166) indicated its presence in the Argentine. Dastur (114) first reported it in Pusa, India in 1923.

Other regions in which mosaic was reported on sugarcane are Australia (178), Central America (110), China (110), (339), Dutch Guiana (110), Fiji (339), Angola, Antigua, Belgian Congo, British Guiana, British Honduras, Cameroon, Costa Rica, Egypt, El Salvador, Formosa, Grenada, Guadeloupe, Haiti, Honduras, Indochina, Ealy, Japan, Kenya, Madeira, Malay States, Martinique, Mexico, New Guinea, Okinawa, Paraguay, Peru, Siam, Sierra Leone, St. Kitts and Nevis, St. Luca, St. Thomas, Tanganyika, Trinidad and Tobago, Turkey, Uganda, and Venezuela (46), (93), (110), (356), (237).

The major sugarcane country where mosaic was not reported is Mauritius (306), (307). Shepperd (306) reported that only once was mosaic discovered there. That outbreak occurred in the nursery at Central Experiment Station on Coimbatore seedlings. The plants were uprooted
and burnt and no further trace of the disease has further been discovered. In 1938, Bouriquet (48) reported that no mosaic occurred on sugarcane in Madagascar. Martin 1951 (237) reported mosaic as occurring on cane in Madagascar.

Considering the origin of mosaic disease of sugarcane, Brandes and Mats 1935 (64) expressed the opinion that no sugarcane had been taken to New Guinea and planted there, perhaps for thousands of years. Mosaic and Fiji diseases have been found there. Neither disease is conspicuous nor abundant there, but assuming one-way traffic of sugarcane outward from New Guinea and not inward, the presence of the disease in any amount would seem to establish the fact that this island is a place of origin of the diseases mentioned. Together with the records tracing the outbreaks of Fiji disease in Fiji, Australia and Philippines, to cane originally brought from New Guinea, the evidence is enough to indicate New Guinea as the place of origin beyond a reasonable doubt. They further stated that in many instances the presence of virus diseases where cane was exotic could be traced to particular importations from the countries where it was indigenous, for example, chlorotic streak in Puerto Rico could be traced to Java, and mosaic in Argentina could be traced also to Java.

Bruner 1923 (79) (translation) pointed out that the exact origin of the disease was unknown. However, it was assumed that it had first originated in New Guinea, occurring first on some wild cane or some related grasses and later attacking the cultivated cane. From there it was introduced into Java, which he considered as the source of infection of the disease. Deer 1921 (119), stated that about 1910, certain approved Java seedlings were imported into Egypt, thence they went to
Argentina, and from there they were brought to Puerto Rico in 1914. In no one of these localities had the mosaic been recorded before, but in each, shortly after the introduction, the disease was recognized. In another paper, Brandes 1935 (61) suggested that mosaic is not a naturally-occurring disease in cane but that it depended upon occasional and transitory infection from some other source, probably a wild grass associated with cane where it was indigenous.

The losses that the mosaic disease causes vary considerably. Chardon 1932 (89) estimated the losses due to mosaic in Puerto Rico to be between $8,000,000 and $10,000,000. Figueroa 1919 (168) in Puerto Rico, reported that the total production of sugar in 1917 was 503,081 tons, while in 1919, there were only 406,002 tons produced. He believed that the greater part of this reduction in sugar production was due to the mosaic disease.

Earle (133) and (136) stated that in a general way when mosaic infection reached an average of 60 - 80 per cent losses of sugar production would be from 30 - 40 per cent. Lyon 1914 in Hawaii found also a reduction of 40 per cent in sugar yield in a field infected practically 100 per cent. The reduction was due to the loss of tonnage, as the disease had no effect on sucrose content of the plants.

Lopes 1919 (227) concluded that the mosaic disease did not affect materially the sugar content of the juice of canes attacked with the disease, except in an indirect way when the stalks became cracked as a result of the drying up of the stalk. In this case the exposure of the inner tissues brought about fermentation, with subsequent increase of acidity, inversion, and loss of sucrose.

Edgerton 1919 (143) stated that the loss from mosaic disease
was mostly caused by the decrease in vigor, with a resulting low
tonnage. Edgerton et al 1924 (156) stated that the mosaic had no in-
fluence on the sucrose content of the juice, and that juice from affected
sugarcane plants would analyse at the mill just as well as the juice from
healthy plants. Lee et al 1923 (218) in the Philippine, reported that in
most cases the loss in sugar production resulted from the loss in cane
tonnage rather than from poorer quality of the juices in the diseased
cane. Faris 1931 (164) found that in two series of tests in Cuba, the
average annual losses from mosaic over a five year and a three year
period, respectively, were 11 and 8 tons per acre (30.82 and 23.42 per
cent of the yield). Brandes 1924 (55) reported that the decrease in yield
of certain mosaic-infected varieties in Hawaii ranged between 36 and 83
per cent, and in Louisiana between 10 and 38 per cent.

Charden 1925 (88) noted that in some cases losses in yield as
high as 60 per cent resulted from mosaic. Bruner 1925 (81), as cited
by Tims et al (360), reported a reduction in yield as high as 62 per cent.
Lee 1929, (216) in the Philippines reported losses varying from 21.5 to
69.3 per cent in the different varieties planted. Summers and Rands
1935 (348) in comparing canes grown from mosaic-free seed and those
from mosaic-infected seed, found that the total loss in yield, when mosaic-
infected seed were used, was 14.8 tons per acre, or 17.6 per cent, and the
total loss of sugar production was 3,277 pounds or 7.2 per cent. They
pointed out that a loss of 3,277 pounds of sugar, if valued at 3 cents per
pound, would represent a total loss of $98.31 per acre for the crops
grown from infected seed.

McRea (259), (260), (261), (262) and Sundaraman (351) in Pusa
compared mosaic-free and mosaic-infected sugarcane in carefully
controlled plot experiments. In the 1931-32 season (260), the diseased Co. 213 was 14.7 per cent less in yield of stripped cane, 8.9 per cent lower in yield of juice, slightly less in brix, and 4 per cent lower in sucrose than healthy cane. McRea and Subramaniam 1934 (265) stated that the loss of weight due to mosaic alone was 11.9 per cent. They further pointed out that this referred to wholly mosaic-infected and mosaic-free canes, but locally, except in experimental plots, no field of wholly infected Co. 213 cane existed. The average amount of infection in 1933 being only 0.57 per cent, a 12 per cent loss in weight of stripped cane in a wholly infected field was equivalent to a loss of only 0.07 per cent on the ordinary crop. They also stated (264) that during a period of four years, the only factor that varied consistently was the calculated juice from the cane. There was a reduction in the quantity of the juice. However, there (265) was no evidence that mosaic affected the quality of the juice.

Tims, Mills and Edgerton 1935 (360) in Louisiana reported on experiments conducted in the period between 1922 and 1934. They concluded that the losses in tonnage due to the mosaic disease had varied to a considerable extent in the tests which had been conducted. The losses in P.O.J. 36, P.O.J. 36-M, and P.O.J. 234 had not been large. The losses in P.O.J. 213 had run from 10 to 18 per cent. The losses in Co. 281 and Co. 290 varied according to the locality. Reductions in yield were more severe when the plants were affected with the yellow type of mosaic than when they were affected with the green type. They also concluded that there had been no significant differences in the sucrose content of juice from healthy and mosaic-infected cane in any variety tested except C. P. 28-70. In canes of this variety affected with
the yellow mosaic, the sucrose content had been considerably reduced. 

Forbes and Mills 1943a (174) and 1945 (175) reported that mosaic 
caused a reduction in the yield of Co. 281, Co. 290 and C. P. 33-243. 
The losses ranged between 8 and 33 per cent, and varied from year to 
year and from one variety to another.

Stevenson 1919 (329) pointed out that the mosaic had an accumu-
lative effect. During the second year of its presence, there was a 
very marked falling off in the yield, and a decrease in the amount of 
juice. The final stage in which no merchantable cane was produced 
might occur in the second or the third year. Edgerton et al 1924 (156) 
disagreed with Stevenson's statement. They stated that the oldest 
mosaic-infected districts in Louisiana, were not the ones showing the 
poorest conditions. Edgerton et al 1937 (153) pointed out that losses 
caused by mosaic to the stubble crop were no greater than those to the 
plant cane.

Stevenson 1919 (329) studied different factors like fertilizers, 
liming, seed treatment, manner of trash disposal, soil moisture, drain-
age, etc. and indicated that this did not have any direct influence on the 
mosaic disease. Chardon 1924 (87) noticed that in alluvial soils, Uba 
variety (a mosaic-resistant variety) gave a poor yield, while some 
mosaic-susceptible varieties gave a very high yield of sugar, and it was 
favorable to plant them in the alluvial regions without taking any pre-
cautions against mosaic.

The germination of cane cuttings may be affected by the mosaic 
disease. Medella and Serrano in (246) the Philippines observed better 
germination with healthy than with mosaic-infected cane. Unite and 
Capinin 1926 (363) concluded that the buds of the nodes within a
mosaic-infected stalk had poor or no germination at all. Martin 1929 (233), in studying varieties H. 109 and H. 8965, observed that every eye germinated in both healthy and diseased cuttings. Every shoot upon germination from the mosaic-infected cuttings was diseased. Ocfnia et al. 1933 (271) and 1939 (270) stated that in some cane varieties, the percentage of eye germination was reduced by mosaic and Fiji diseases, while in others the healthy plants produced fewer eyes than the infected ones, indicating that germination was influenced by some factor independent of the diseases under observation. Shaffer 1939a (304) in Louisiana, observed that selected seed under field conditions gave better germination and better yields than ordinary unselected seed, which had a high percentage of mosaic. McRea 1932 (260), in India, reported that the diseased cuttings of Co. 312 gave 11 per cent less germination than the healthy ones. However, McRea and Subramaniam (264) reported in the following year that the difference in germination of healthy and mosaic-infected plots of sugarcane was not statistically significant.

Tims et al. 1935 (360), pointed out that many of the statements in the literature in regard to the effect of mosaic on the germination of sugarcane buds were based on observation alone and were not entirely convincing. Considering those based on carefully conducted tests, it was evident that under some conditions, the vitality of the cane might be reduced to such an extent by the disease that the germination of the buds was affected to a greater or less degree. Under good growing conditions, however, practically as good germination could be expected from mosaic-infected cane as from healthy cane (360).

Tims et al. 1935 (360) reported on experiments conducted in Louisiana from 1921 to 1934 on the effect of mosaic on germination of
cane buds. They stated (c.f. page 19) that mosaic had not caused an appreciable reduction in stand of P. O. J. 234 and P. O. J. 36. Some rather slight increases in stands had been noted in plots planted with mosaic-free P. O. J. 213, but these differences had not been consistent. The tests with Co. 290 and Co. 281 conducted for two years showed that there was no indication of reduced germination in mosaic-infected canes. Mosaic was responsible for slight reduction in germination of the Louisiana Purple and Louisiana Striped varieties. They also stated that the yellow type of mosaic did seem to cause a reduction in germination of the buds of C. P. 28-70 though differences in the final stands in the field were not as great as the differences in germination might have indicated.

Symptoms of the mosaic disease of sugarcane have been described by several authors, who more or less agreed on the general features of the disease. The one characteristic feature used for diagnoses was the presence of a peculiar "mottled" pattern upon the younger leaves caused by the contrast of light green areas against the normal dark green of the leaf. These light colored areas were irregular in size and shape and had distinct margins (45, 236).

Johnson and Stevenson 1917 (204), in describing the disease, reported the appearance on the leaves of innumerable white or at times yellowish spots and stripes with irregular indefinite margins, and noted that the mottled leaves did not die any sooner than the normal ones nor cling abnormally to the stalk. Stevenson 1919 (329) in reporting on a thorough study of the disease in Puerto Rico, stated that two distinct types of discoloration had been noted. However, he proceeded to say that they were always so grading into one another that it was
not considered that they were other than phases of the same phenomenon. In one type the background or larger portion of the leaf-blade was a light, abnormal yellow-green and scattered about in it were areas of apparently normal color, "green islands". These spots were for the most part linear, but would vary from mere points to irregular blotches several centimeters long by a centimeter wide, always with a decided tendency to greater length than breadth.

In the other type, which Stevenson (329) considered the usual phase of the disease, the background would vary in color from a normal green in plants but recently attacked to a yellowish green in more severe cases. The markings which produced the mottled effect were always much lighter in color, giving a very decided contrast.

Stevenson 1919 (329) pointed out that as a general rule the leaves were uniformly mottled, but cases were not uncommon in which only a portion of a leaf was affected. Such instances were found in which a stalk previously apparently normal commenced to show mottling. The lower leaves, anywhere from two to a dozen in number, seemed normal; then a leaf showed mottling for a few inches at the base of the blade, which in turn was succeeded by several others above which were affected from a half to two-thirds their length; then completely mottled leaves appeared. Stevenson (329) also pointed out that occasionally one-half of a leaf might be affected more than the other half.

Brandes 1919 (51), in describing the primary symptoms of mosaic stated that upon walking between the rows of sugarcane in an affected field, a greater or less number of plants were conspicuous on account of a general color of the leaves. This color was due to irregular light-colored streaks or spots on the leaves. The affected leaf areas,
in so far as color was concerned, were of two distinct types. The most common type presented a merely "washed out" appearance. It was merely a tint of the normal color, in which the blue and yellow were present in the same proportions, but diluted. In the second type, the yellow was predominant, and the affected areas had a decided yellowish-green appearance.

Brandes (51) also pointed out that the normal and affected areas in the leaves were sharply demarcated. In other words, there was no gradual merging of one color into the other. Kunkel 1924 (212) stated that while this was true for young leaves, on the older leaves there was a more or less gradual fading out of the green color in the tissues between normal and light green areas. He concluded that the sharpness of the line of demarkation between normal and light green areas varied with different varieties.

Lyon 1921 (229) pointed out that the primary and critical symptom of the disease was the appearance of pale blotches in the green tissues of the leaves. These blotches were several shades lighter than the normal green tissues which surrounded them. They were not constant in size or shape, even on the same leaf. They were usually irregularly oval or oblong in outline, their longer axes lying parallel to the midrib. However, they were not confined between veins and consequently were not of uniform width throughout any considerable part of their length. Storey 1924 (333) stated that the pale areas were also of irregular shape and spread unevenly laterally over several veins. Kunkel 1924a (213) stated that the mosaic pattern on the upper surface of a leaf was exactly the same as that on the lower surface.

Lyon 1921 (229) stated that after the diseased sugarcane leaf had
unrolled the patches did not change materially in size or shape, but they usually did change considerably in color; in some varieties becoming more distinct, and in others less distinct, as the leaf grew older. On the other hand, Kunkel 1924a (213) stated that in sugarcane, the pattern shown by old diseased leaves usually was not present on young ones. He observed that the chlorotic spots began as small spots scattered about over the leaf, then they grew and finally included much tissue that was at first a normal green color. In their growth, the spots that happened to be close together coalesced and formed chlorotic areas of irregular sizes and shapes.

By marking off portions of very young leaves and carefully drawing to scale the individual spots, using the varieties striped tip and Hawaii 109, Kunkel (213) was able to prove that the spots slowly increased in size and frequently fused together. He further observed that new spots sometimes arose in the normal green tissue as leaves unrolled from the spindle. Cook 1926 (106) in this connection stated that groups of cells so small that they could not be seen with the unaided eye occurred in green areas of young leaves, and it was very probable that groups of this kind enlarged with the growth of the leaf and gave the appearance of new spots.

Cook 1926 (106) stated that his studies showed the chlorotic areas in diseased plants were quite definitely determined before the formation of chlorophyll. These areas increased in size with the growth of the leaves but they did not encroach the surrounding green tissues and they did not coalesce. The increase in size was attributed by Cook to be by cell division and growth and not by infection of surrounding cells.
Storey 1924 (333) indicated that in certain varieties the mosaic pattern tended to become obscured as the leaves became old. Thus, the determining point for diagnostic purposes was the presence of the mosaic pattern upon the youngest, most recently unfolded leaves. He concluded that this afforded a means of differentiating mosaic from the many other types of leaf-markings which were to be seen in a cane-field. Brandes and Matz 1935 (64) also stated that the symptoms were most easily detected on the younger leaves of the cane. They further stated that the symptoms were more difficult to detect on plants which presented a general yellowed appearance due to waterlogging.

The midrib of the diseased leaves, as reported by Stevenson 1919 (329) remained to all external appearances normal. Dastur 1923 (114) in India, reported that in the cane varieties, D99 and Sathi 131, the under surface of the midrib was mottled.

The leaf-sheaths of mosaic diseased sugarcane plants, as reported by Stevenson 1919 (329) presented no abnormal signs, except a faint mottling in early stages of growth. Lyon 1921 (229) stated that the green tissues of the leaf-sheaths were affected in the same manner as those of the leaf-blade but the blotches were always less conspicuous.

A diversity of mosaic patterns in the different varieties of sugarcane was noticed by Brandes (51). He stated that the variation was found in the amount, size and shape of the light-colored areas, but the arrangement was so constant in any particular kind of cane that the character could be used as an aid in determining the varieties.

Secondary symptoms of mosaic disease of sugarcane have been described by several authors (45), (51), (202), (204), (229). Brandes (51), Stevenson (329), and Lee and Kopke (217) expressed the opinion
that the deleterious effects of the disease were accumulative. Brandes 1919 (51) stated that in first ratoons of canes which became infected the previous year, or in plant canes originating from diseased cuttings another quite distinct series of leaf symptoms appeared. It consisted of small white opague spots and streaks in the light-colored areas. These non-pigmented streaks ranged from mere points to several inches in length. The white opague tissue had a dried out appearance, but remained firm and did not become brown or rot out.

Johnston and Stevenson 1917 (204) reported the appearance of cankers on the stalks of mosaic infected plants. These cankers or lesions appeared as linear spots, somewhat sunken and brown in color. They soon became ashen or dull grey and often coalesced to form continuous patches practically covering the internode. However, they were superficial only, never penetrating for more than 1 - 2 mm., and did not pass from one internode to the other.

Brandes 1919 (51) indicated that ordinarily, cankers did not become noticeable in mosaic infected plants until the cane was well developed; yet, by tearing away the leaf sheaths, cankers could be found in the incipient stage. They appeared as discolored or water-soaked patches or longitudinal streaks on the internodes. In severe cases these areas became sunken and the internodes were spindle shaped and attenuated. Longitudinal cracks may appear resulting in the drying out of the cane. Stevenson 1919 (329) indicated that a stool might show mottling through seasons before the cankering appeared, and that cankering was much more marked in some varieties than in others. Edgerton 1919 (143) reported that no lesions or cracks had ever been noticed on diseased canes in Louisiana.
Additional symptoms appearing on the stem of canes infected with mosaic have been reported by Lee and Kopke (217). They stated that on badly affected plants a mottling or streaking with a green or pinkish color was apparent on the stalks of yellow varieties, while on purple varieties a mottling with yellowish or grayish white streaks showed on the stalks. Storey 1924 (333) reported that many of the red-colored canes would fail to develop the red pigment in places, producing a blotching upon the stem and conversely, certain normally pale-colored canes would produce deeply pigmented marks upon the stem as a result of the disease.

Mottling or marbling of the stem was considered by Lyon (229) to be the one secondary symptom most frequently displayed by diseased canes in Hawaii. He stated that in some varieties of cane no mottling of the rind could at any time be distinguished; in other varieties it was very distinct, disappearing entirely shortly after the internode was exposed to the light; in still other varieties it became apparent only after the internode was exposed to strong light.

Discoloration and necrosis in mature inner stalk tissues was considered by Kunkel (213) to be a constant symptom of mosaic disease of sugarcane. Cross sections through a stalk of the variety Hawaii 109, showed circles of necrotic tissue. Kunkel expressed the belief that this form of necrosis in the stalk corresponded to the zonation shown by the chlorotic tissue in the light green colored spots on certain diseased leaves.

Shortening of the internodes of the individual stalks of diseased plants as well as stunting or dwarfing of the stools, have been reported (51), (329), (114). Lee and Kopke (217) pointed out that the
length of cane as well as the diameter of cane was much reduced, yet the effect of mosaic differed according to the variety.

Faris 1929 (163) in Cuba, stated that the mosaic cane had a very high proportion of dead stalks with dry tops, in marked contrast to the healthy cane, which was on exactly similar soil. As the tops and leaf areas in the mosaic cane were much less than in healthy cane it was suspected that the mosaic disease was also dwarfing the root systems. He concluded that it was very dangerous to have mosaic fields, even though under normal rainfall conditions they gave adequate yields.

Faris (163) also noted that the root disease was most prevalent in mosaic areas. There are no other reports known to the writer which confirm Faris's opinion on the prevalence of root rot in mosaic infected plants, and some of the symptoms described by him for mosaic are usually those of root rot disease of sugarcane. Brandes (51) for example, stated that mosaic was never fatal during the first year, and rarely terminated in death, even in diseased plants that had been allowed to ratoon for years.

The first studies on the histology of mosaic of sugarcane were made by Matz 1919 (241) who noted the occurrence of distinct single spherical, dark colored protoplasmic bodies in the parenchyma and collenchyma cells of the cankered stem tissues. In a comparative study of healthy and diseased canes Matz (241) also noted the occurrence of a foreign plasmodium-like substance in the cells of diseased leaves and stems, and that the microscopic sections of the discolored areas in the diseased stalks showed that some parenchyma cells were full of a hardened or compact but finely granulated and slightly browned plasma.
Furthermore, he stated that the deterioration and breaking down of cells in the interior of the cane (an advanced stage of the disease) was due to the destructive action of the infective substance of the mosaic disease, as there was no connection between these interior cells and outside agencies. Mats concluded that this substance resembling a "Plasmodium" was found to be constantly associated with mosaic-diseased cane in an advanced stage of the disease.

Kunkel (210) found intracellular bodies in mosaic-diseased corn plants. These bodies were variable in shape and amoeboïd but never spherical, and were closely associated with the host nucleus which became enlarged. He expressed the belief that these bodies were living organisms and were similar to those associated with certain virus diseases of man and animals. In a later paper, Kunkel 1924 (212) reported these amoeboïd bodies in mosaic-diseased plants of sugarcane, tobacco and Chinese cabbage. Cook 1925 (105) reported that the intracellular bodies were difficult to find in diseased cane plants, and they were more readily found in young rapidly-growing plants than in old plants. They were found in the light areas of the leaves and near the growing points in the stems of the canes and sometimes in other places, but never in healthy canes and never in the roots of diseased canes.

Mats 1922 (242) pointed out that Iwanowiski in his studies of tobacco mosaic published in 1903 reported intracellular bodies similar to those reported by Kunkel in 1921 (210), but Iwanowiski disregarded these as a possible cause of the mosaic disease on the grounds that these bodies were altogether too large to pass through the fine pores of filters. Kunkel in 1923 (212) observed small bodies resembling protozoa which
were associated with the mosaic disease. He expressed the belief that the transmission of the disease by aphids was caused by the transfer of these bodies from diseased plants to healthy plants.

Dastur 1932 (115) reported that in mosaic diseased leaves foreign bodies had been observed in large numbers. These bodies were totally absent from healthy sugarcane leaves. They were found in all parts of diseased leaves, including xylem vessels. In sclerenchymatous fibers and xylem vessels the bodies were elongated and might have a flagellum-like appendage at one extremity, while in those in the young parenchymatous cells then resembled amoeba in shape. He further stated that these bodies appeared to divide by fission, had one or more of what appeared to be nuclei and could pass from one cell to another. They were also observed in stem and "eyes" of diseased canes, but were less frequent there.

The chloroplasts in diseased cane leaves were found by Matz 1922 (242) to be fewer in number than those in leaves of healthy plants. Matz further stated that there was evident destruction of chloroplasts in the diseased portion of the leaf tissue, the chloroplasts being misshaped and broken up. In the early stages of infection the breaking up of chloroplasts began with a reduction in their size. Sundaraman 1928 (349) in India confirmed Matz's observations.

Cook 1925 (105) and 1926 (106) pointed out that he was unable to detect any differences between the tissues from green areas of mosaic plants and the tissues from apparently healthy sugarcane plants. In the chlorotic areas of diseased leaves there was reduction of the chloroplasts in size and number, which was accompanied by enlargement and deformity of the nuclei and occasionally by intracellular bodies. He also
noted that the chloroplasts in the chlorotic areas in the older leaves of the diseased plant were very nearly or quite normal in appearance.

Cook 1925 (105) stated that there was no evidence of disintegration of the chloroplasts in the diseased leaves and that they were undeveloped rather than disintegrated. He further stated (105), (108) that in the light green areas the formation of the chloroplasts was inhibited but with the exposure to sunlight this inhibition was gradually overcome, resulting (106) in increase in number and size of chloroplasts in the old leaves. This could not possibly be the case if the chloroplasts were undergoing disintegration.

The theory of inhibition of the chloroplasts advanced by Cook 1925 (105) was well received by some workers, and was not accepted by others. Cook 1930 (109) reviewed the literature on this point and reported his investigations on mosaic and healthy plants of sugarcane, tobacco, tomato and cowpea. In the summary of his paper, Cook (109) stated that the active agent inhibited the differentiation of the cell structure from the time that the two came in contact. If the active agent came in contact with the meristematic tissue at an early period in its development, the structure remained undifferentiated; if at a later period, the tissues remained in the stage of development at time of attack. Considering the plastids he stated that their development was inhibited in both size and number. In very young, growing leaves, it was difficult to locate them in either fresh or stained material, but they developed rapidly with age and exposure to sunlight.

Among the histological studies on mosaic disease of sugarcane is that by Dufrenoy 1929 (127). Working with Louisiana Purple and a resistant P. C. J. variety of cane, he reported that the first symptoms
of the mosaic was the breaking up of the large central vacuole, which
was usually found in healthy cells of the leaves, into a large number of
small vacuoles. In cases where a large vacuole did develop in a cell
within infected tissue, the cytoplasm of the neighboring cells was always
pitted with small vacuoles, this giving it a spongy aspect. This pheno­
menon, which considerably increased the contact surface between the
cytoplasm and the vacuolar contents, was believed to have heightened
activity of the cell. In mosaic diseased cells photosynthetic activity
was reduced, and the osmotic pressure of the vacuolar liquid was less
than in normal green cells; further, they always contained a proteolytic
focus (127).

Forbes and Dufrenoy 1943 (171) reported on an internal break­
down and collapse of the central pith cell in rather definite elongate
areas in the internodes in the variety C. F. 33-243. The necrotic areas
were distributed in various internodes from the base to the top of the
stalk, but usually did not occur in all internodes. These areas were
usually present in mosaic-infected stalks. The writers concluded
that the internal breakdown was a symptom of mosaic in this variety.
They further stated that the vascular contents in these lesions leaked
out into the intercellular spaces. The material left in the cells floccu­
culated into a brown sediment and settled to the bottom of the cells.

Cook 1925 (105) and 1930 (109) stated that the light areas of
a diseased leaf were slightly thinner than the green areas of the same
leaf or corresponding leaves of a healthy plant of the same age.
Ciferri 1932 (101) made 17,620 micrometric measurements with gal­
vanometric control of the thickness of mottled and healthy leaves.
Fifteen leaves of four mosaic susceptible sugarcane varieties were
examined. The mottled leaves were found to be generally very slightly thicker than the healthy ones. The difference approached the limits of error and was variable in relation to many distinct factors. Ciferri (101) expressed the belief that the greater thickness of mottled leaves was caused by a different turgidity, during the period of the activity of the leaf, while the dry substance contained in the healthy leaves might be greater than the substance contained in mottled leaves.

Cook 1926a (107) studied the relationship of the mosaic disease to photosynthesis in sugarcane plants. He found that normal healthy plants and the green areas of the diseased plants performed photosynthesis normally, and showed a large amount of starch in the afternoon and very little in the morning (5 - 6 a.m.), while the white and light green or yellowish areas showed a small amount of starch in the afternoon and practically none in the early morning. He further stated that it was evident that the starch-forming power of the mosaic cane was reduced in proportion to the amount of infection while the power of translocation was practically unimpaired, and that the old leaves of cane infected with mosaic produced more starch than the young leaves of the same plant.

Yamafugi et al 1943 (377) in Japan reported the catalase activity of mosaic diseased sugarcane leaves to be much weaker than that of healthy ones. For instance, reckoning the catalase activity of a sound leaf at 100, the corresponding figure for an infected one was 61. Evidence was secured pointing to the enclosure of the host cell catalase in the high-molecular virus protein during the process of virus multiplication, so that the activity of the enzyme could only be exerted under appropriate conditions after the splitting up of the virus.
Considering the cause of the mosaic disease of sugarcane, Johnston 1919 stated that its cause was unknown, but that it was not a simple chlorosis. It was transmitted by cuttings, but the soil was incapable of transmitting it, and that it was injurious and incurable. Grey 1919 (183) stated that mosaic was not incurable and that he had eliminated it by a culture method. Earle 1919 (133) suggested that Grey confused true mottling with the often similar attacks caused by insect punctures and fungi. Grey 1919a (184) denied that he had made such a mistake, and that an authority on mosaic sent by the United States Department of Agriculture identified the mosaic he had referred to in his earlier paper (183). Furthermore he (184) cured the mosaic-diseased plants by "aid of a good soil and proper treatment". He also stated that climatic conditions had a direct influence on the appearance, diminution and self-eradication of the disease in the field. Barber 1921 (41) presented a short resume of the different opinions on the cause of mosaic. He also stated that the very complete study to which mosaic had been subjected had knocked away these props one by one, until it was generally conceded that to no single one or combination of them could the disease by any chance be attributed, and there was nothing left to fall back upon but hypothesis. Rosenfelt 1924a (295) suggested that the organism that caused the disease might be seen and the ultra-visible or filterable virus term attached to it passed out of fashion.

Cook 1924 (104) reviewed the theories advanced on the cause of mosaic disease and mentioned the following: Bud variation, soil, bacteria, enzymes, protozoa, physiological, and virus.

Brandes 1919 (51), 1920 (52), (53), (62) started experiments which gave proof of the infectiousness of the mosaic disease of
sugarcane. The disease is now considered to be caused by a virus. Summer et al. 1948, stated that "The furtive virus that had managed to conceal most of its strategy for so long a time was now fairly out in the open."

The physical properties of the sugarcane mosaic virus have been studied in recent years. Bruner 1922 (78), stated that by passing the juice expressed from mosaic infected plants through a Chamberland-Pasteur filter, no infection was obtained. Then he pointed out that whether the failures were due to the exclusion of the infection agent, or simply to the oxidation of the liquid consequent upon the lengthy process of filtration was not known.

Carpenter as reported by Martin 1934 (235) kept mosaic infected leaves of Ba. 11569 cane for ten minutes at temperatures ranging from 52 degrees to 56 degrees centigrades and then attempted to transmit the disease from the treated to healthy Ba. 11569 leaves by Scll's needle prick method. The results indicated that the thermal death point of the virus was between 53-54 degrees centigrade.

Rafay 1935 (277), using a standard method of inoculation by means of 20 entomological pins No. 10 mounted in a cork pricked through the mosaic juice into the leaf, found that the sugarcane mosaic virus tolerated a dilution of 1 in 10 but not 1:100. He also found that the virus lost its potency in two hours when kept at a temperature of 20 degrees centigrades. Filtration experiments showed that it was retained even by filter paper, the filtrate being non-infective. In testing the reaction of the virus to chemicals he found that copper sulphate, 1 in 1500, hydrochloric acid 1 in 1000, nitric acid 1 in 800, hydrogen peroxide, 1 in 25, and formalin 1 in 50, inactivated the virus; while zinc powder
and manganese dioxides did not retard its activity. Evidence was obtained that the dilute concentrations of the reagent might increase the virulence of the virus (277).

Brandes 1935 (61) found out that sugarcane mosaic virus when heated at temperatures ranging from 60 to 80 degrees centigrade failed to cause infection when used to inoculate D-74 cane plants. He further stated that preliminary experiments of Dr. Julius Matz indicated that the thermal death point of the virus might be as low as temperatures often encountered in the field.

Chona 1937 (99) reported that the thermal death point of Saretha sugarcane mosaic virus was little over 50 degrees centigrade, but heating to this temperature did not appreciably reduce the infective power of M. 16 and Co. 313 sugarcane mosaic virus. Further, heating at 60 degrees centigrade inactivated the latter, but did not completely inactivate the former. The thermal death points of Co. 213 and Red Mauritius mosaic viruses were found to lie between 40 and 45 degrees centigrades, and 40 and 50 degrees centigrades respectively. Clear brown L3 filtrates of mosaic juice were found to be non-infective. With both Co. 213 and Saretha mosaic the infective power appeared to fall rapidly when the juices were diluted to 1 in 10 or more.

Aduar 1950 (24) used one month old sorghum seedlings (Holcus sorghum L.) as test plants for studying the physical properties of sugarcane mosaic virus. The leaves were dusted with carborundum and rubbed with a cotton swab soaked in the infectious liquid. The juice was obtained from the leaves of mosaic-infected plants of sugarcane variety BH 10/12. He found the longevity of the virus was seven hours at room temperature (82 degrees F.); that the thermal inactivation
temperature was 55 degrees centigrades for ten minutes, and that the
dilution end point was 1 - 1000. Filtration was done by passing pressed
juice through Coors porcelain filter cylinders of four different porosi-
ties; P₂, P₃, P₅, P₇; porosities increasing in fineness from P₂ to P₇,
and the virus failed to pass the Coors P₂.

Costa and Penteado 1951 (112) used corn seedlings of inbred
lines and standard commercial varieties for studying the physical pro-
PERTIES of sugarcane mosaic virus. The seedlings were grown in pots
and when they were about 5 inches tall they were inoculated by juice ob-
tained from mosaic infected plants of P. O. J. 213, using carborandum
powder (600 mesh) as an abrasive. The virus was active in inoculum
aged for 24 hours at room temperature. At dilutions of 1:10 and 1:100,
the percentages of plants infected were 95.0 and 66.0 respectively, while
at a dilution of 1:1000 the infectivity of the virus was greatly reduced,
only 26.7 per cent of the plants being infected. At dilutions of 1:10,000
and 1:100,000 the percentages of infected plants were 6.6 and 3.3 re-
spectively, while 93.3 per cent of these plants were infected by using
undiluted inoculum, the diluent used was an 0.02M solution of sodium
sulphate.

In tests on thermal inactivation, the sugarcane mosaic virus
used by Costa and Penteado was obtained from corn infected plants.
After heating the plant extract at 50 degrees and at 55 degrees centi-
grade for 10 minutes the percentage of infected plants was 16.7 and 3.3
respectively, while no infection was obtained after heating at 60 degrees
centigrade.

Costa and Penteado (112) pointed out that their results pre-
sented above were slightly different from those obtained by Adsuar
(24) and by Rafay (277). They interpreted that as being due to the higher susceptibility of corn seedlings to the virus in comparison with sorghum seedlings and sugarcane plants which were used by the other authors (24), (277) as test plants.

In serological tests with anti-sugarcane mosaic and anti-healthy juice sera, in which rabbits were immunized by intravenous inoculations with Chamberland candle filtrates of mosaic and healthy leaf juices, the anti-mosaic serum inactivated the mosaic leaf juice while the anti-healthy serum had no effect, this result being confirmed by the fact that all the plants inoculated with the anti-mosaic serum and the mosaic leaf juice mixture remained healthy while those inoculated with the mixture with anti-healthy serum became infected. Precipitation tests with the anti-mosaic serum gave a positive reaction with mosaic leaf juice and a negative one with healthy leaf juice, the anti-healthy serum being faintly positive to both (121).

The host range of sugarcane mosaic virus is very wide. Summers et al reported its occurrence on varieties of *Saccharum officinarum* L., *S. robustum* Brandes and Jeswiet ex Grasai, *S. edule* Hassk., *S. barberi* Jeswiet, *S. sinense* Roxb, *S. spontaneum* L., *Erianthus* sp., and on varieties resulting from hybridization between different species of the genus *Saccharum*. They also reported it on varieties resulting from hybridization between *Saccharum* and sorghum. They concluded that sugarcane mosaic was probably the most cosmopolitan of all viruses that caused disease of grasses.

Several grasses have been reported to be susceptible to sugarcane mosaic virus. Brandes 1919 (51), 1920 (52), (53) pointed out that sorghum, corn, and certain grasses were susceptible to the so called
sugarcane mosaic virus. Brandes and Klaphaak 1923 (62) demonstrated by cross inoculations that corn, pearl millet, sorghum and a number of even distantly related grasses were probably as congenial hosts for this virus as sugarcane itself, and they suggested that the disease be called the grass mosaic. Since then other reports (3), (113), (122), (138), (161), (179), (185), (186), (213), (215), (256), (333), (369), (347), have indicated that the disease might occur in a number of additional hosts that are in most of the countries where sugarcane is grown.

The susceptibility of these different grasses to mosaic has long been recognized and considered important in dissemination of the disease. Brandes and Klaphaak 1923 (62) stated that the disease might be carried over winter in perennial grasses and might reinfect the susceptible annual grass crops in the spring. Summers et al 1948 (347), pointed out that the susceptible annual wild grasses could contribute to mosaic spread only after they became infected from mosaic cane or other perennial grasses in the field at the beginning of each growing season.

Crabgrass (*Digitaria sanguinalis*) probably accounts for a considerable proportion of secondary mosaic spread on sugarcane. Because of its general occurrence in Louisiana, its extreme susceptibility to mosaic, and the fact that it is freely colonized by *Aphis maidis*, crabgrass is probably a greater factor in mosaic dissemination than any wild grass (347).

Johnson grass (*Sorghum halepense*) was reported by Kunkel 1924 (213) to be susceptible to sugarcane mosaic in Hawaii. Summers et al 1948 (347) stated that this was not true in the United States. However, they pointed out that even though Johnson grass may not
actually harbor the disease, it is a very important factor in mosaic dissemination. As it is abundant in and around cane fields in Louisiana and is one of the best host plants for multiplication of *Aphis maidis*, whose populations later migrate to infected hosts and institute mosaic epidemics in cane. They concluded that the presence of obviously mosaic-infected hosts contiguous to the aphid-infected Johnson grass seems to be essential for this grass to play any part in the spread of mosaic.

Corn was first reported to be severely injured by sugarcane mosaic by Brandes 1920a (53) and Brandes and Klap haak (62). As it is one of the favorite host plants of *Aphis maidis*, Ingram et al 1939 (193) conducted experiments to determine the effect on aphid abundance and on mosaic spread of growing corn adjacent to sugarcane. They reported that there was practically no difference in vector abundance between the fields adjoining corn and those at some distance from corn. Their data indicated that the presence of growing corn did not increase mosaic spread in adjoining sugarcane to any considerable extent. The average increase in sugarcane adjoining corn was 10.34 per cent, while in sugarcane more distant from corn was 11.18 per cent.

However, Summers et al (347) working also in Louisiana pointed out that since corn is the favored host of *Aphis maidis* the spread of mosaic to sugarcane is apt to be rapid. They indicated that individual stalks of corn affected by mosaic have been observed harboring countless thousands of these aphids. The production of such large numbers of viruliferous aphids on corn will make them available for instigating a mosaic epidemic in adjacent cane fields when other contributing factors are favorable.
McMartin 1947 (257) in South Africa, reported on experiments, in which in the first plot corn was interplanted with cane on the 9th of November, and in the second plot short lines of healthy Co. 281 alternating with the same variety from infected stools. By the following 10th of January, the disease had begun to spread in the first plot, and on the 22nd, fourteen cases of mosaic infection were found on the cane, while 40 per cent of the cane showed mosaic symptoms. He noted that A. maidis was prevalent on the corn. On the second plot no spread of mosaic had taken place by the 22nd of January. He concluded that this experimental demonstration of the risk of growing corn in proximity to sugarcane gave further weight to the recommendation made for the avoidance of the practice.

Dodds 1947 (126), gave more reports on McMartin's (257) experiment described above, stating that the number of new cases of infection on sugarcane in the plot containing maize had risen to 26.44 and 67 on the 3rd, 10th and 17th of February, respectively. In the plot without corn the cane remained healthy until the 10th of February, when two cases were observed, and on the 7th an additional case was noticed.

Insect transmission of sugarcane mosaic was first announced by Brandes in 1920 (52). He announced that Aphis maidis was able to transmit the disease to healthy sugarcane plants after a previous feeding on diseased plants. Brandes (52) further pointed out that A. maidis had been reported on sugarcane from practically all sugarcane countries and assumed that a certain amount of natural transmission in the field could be traced to this insect. The belief was expressed, however, that natural transmission was not restricted to A. maidis.
Smyth carried on a large number of experiments on insect transmission of mosaic in Puerto Rico. In 1919 he reported (309) that out of 1817 trials only four successful inoculations resulted and in 1920 (310) out of 310 additional experiments, only nine gave positive results.

Chewing insects did not transmit the disease in Smyth's experiments (309). On the other hand he reported (309) that the four successful inoculations resulted were with the sucking insects, the yellow sugarcane aphid (*Sipha flava* Forbes, the mealy bug (*Pseudococcus sacchari* Ckll and *Pn. calceolariae* Mask) and the cane leaf scale.

Smyth 1919 (309) concluded that in view of the small number of successful inoculations secured as compared with the large number of tests made, the question of insect transmission of mosaic diseases could not be looked upon as settled, and that other factors not visible to him may have entered into the success of inoculations.

Brandes 1923 (54) also questioned the successful results reported by Smyth (309) since in some cases his test plants were transplanted to an open field and left for periods greater than the minimum inoculation period for mosaic before showing symptoms.

Tower 1920 (361), working in Puerto Rico, got only negative results in experiments in which he used sucking and chewing insects.

Ledebor 1920, in Java, as reported by Brandes 1923 (54), announced that *Aphis sacchari* as well as *A. maidis* could transmit the disease. Later in 1921 (54) he stated that while his experiments with *A. maidis* had confirmed the work of Brandes (52), his preliminary experiments with *A. sacchari* were not considered conclusive. Ledebor 1922 (214) announced that *Aphis adusta* has been proven to be an active
agent in transmitting the mosaic, and that it occurred more generally in sugarcane plantations than was usually supposed, especially on *Panicum coloratum* and *Paspalum (Panicum) sanguinale*. These grasses are subject to sugarcane mosaic, and are capable not only of infecting the cane in their vicinity but also serve as new sources of infection when the diseased canes have been removed.

Ledebo 1922 (214) further stated that there was no actual evidence that *A. sacchari* was implicated, though at one stage of the investigations, this seemed probable.

Walcott 1921 (376), published a long account of the minor sugarcane insects of Puerto Rico. He pointed out that there were certain essential characteristics which an insect must possess to be considered as a possible vector of mosaic disease. First, it must occur in abundance on sugarcane in all parts of the Island where, and at the times when, the disease is spreading. Second, it must be capable of more or less rapid spread or dispersion, either by itself or by other insects or agencies. Third, presumably it should be an insect with sucking mouth parts. However, he pointed out that insects with biting mouth parts were not tested. Fourth, it should live on the part of the cane plant in which the virus which causes the disease occurs, and to which the virus applied in healthy plants causes the disease, and he considered that to be the central whorl of tender, growing leaves of the cane plant.

Walcott 1921 (376) also pointed out that corn aphid had not been found on sugarcane in Puerto Rico. It could not in any way be considered responsible for the transmission of mosaic disease of sugarcane under field conditions in Puerto Rico.

Bruner 1922 (78) conducted careful experiments in Cuba and was
able to bring about infection of healthy plants in a low percentage of his trials by means of the aphid employed by Brandes in his trials \textit{(Aphis maida)}s, but he stated that as this insect did not normally attack sugarcane under field conditions, it could not be taken seriously into account as an active agent in the spread of mosaic. He also tested different insects belonging to several genera and stated that they proved non-carriers or else their activity was thought to be negligible, with the exception of \textit{Phaciocephalus} sp., which he suspected to be active carriers of infection.

Kunkel 1922 (211) in Hawaii conducted eleven different experiments for studying insect transmission of mosaic. He demonstrated the ability of \textit{Aphis maida} to transmit the disease, but was unable to demonstrate this ability on the part of \textit{A. sacchari}.

The material presented in the preceding paragraphs showed that although most of the investigators agreed that \textit{Aphis maida} could transmit the mosaic disease under experimental conditions, yet they doubted this aphid's ability in playing an active role in transmitting the disease in the field. Chardon and Veve 1922 (90), 1923 (91), working in Puerto Rico, threw light upon this problem. They indicated that \textit{Aphid Maida} occurred in every cane field in Puerto Rico, and it was not living on cane but occurred on various common grasses. They further reported the observation that after the weeding of a field the aphids passed to the cane plants and lived in the central whorl of the leaves. This obligate change of host was followed by a sudden spread of mosaic.

Experiments were then conducted in the field by Chardon and Veve (91) to test their observation. Mosaic and healthy sugarcane plants in the field were covered with cheese cloth and \textit{A. maida} was introduced
In the cages thus formed. The weeds were then cut down in one evening, and early in the next morning the aphids were seen feeding on cane plants. They stayed on the cane, but as soon as weeds started to come up, they were seen on their favorite hosts. Two weeks later, secondary infection of the sugarcane plants with mosaic appeared and about 63 percent of the healthy plants became infected at the end of two months.

In conclusion, Chardon and Veve (91) stated that *A. maidis*, after the weeding, passed to cane plants for a period of time. During the short time that it remained on cane, it would transmit the infective substance of cane mosaic. They further stated that dissemination of the disease in the field was done by the aphids that carried the disease from diseased to healthy plants.

Kunkel 1922 (211), working at the same time in Hawaii, reported observations confirming those of Chardon and Veve. He observed that a rapid spread of mosaic in sugarcane followed the weeding of a field. The diseased grass was infested with *A. maidis* previous to weeding and the insects probably migrated to the cane when the weeds were cut. However, Kunkel (211) expressed the belief that *A. maidis* could not invade cane fields to such an extent as to be of any importance in the spread of mosaic from diseased to healthy plants, as these aphids did not thrive or maintain a colony on the sugarcane plant. Then he proceeded to state that yet if a suitable host plant subject to the same mosaic disease was present in or near cane fields, this aphid might become a serious means of spreading the disease.

Brandes 1927 (56) expressed the opinion that weeding had no more than an infinitesimal effect in increasing the number of new cases of mosaic in the field, as weeding did not synchronize with conditions
that led to natural migrations of the insect in tremendous numbers. He then proceeded to state that weeding might play a small part in the spread of mosaic, but that it had been overemphasized and that he had reported the abundant occurrence of *A. maidis* on sugarcane plants in fields that were never weeded. This showed that such a thing as depriving the aphids of their food was not an essential factor in their migration.

Ingram et al 1951 (192) pointed out that the corn leaf aphid was normally present on sugarcane only in the winter and early spring, and then only in the central whorl of leaves. It was seldom found on this plant later in the season when the wild grasses growing in and around sugarcane fields were available. After the new growth of its favorite grasses appeared in the spring, the aphids moved to it. If the grasses were susceptible to mosaic, the aphids might transfer the disease from mosaic-infected cane to them. Later the mosaic-infected grasses were killed by plowing or hoeing, or became mature and unattractive to the aphids. The aphids might then leave the grasses and move to uninfested sugarcane, taking the mosaic with them.

Hadden 1928 (185) in Hawaii found that wingless forms of *A. maidis* had great difficulty in crawling any distance, except on a flat, smooth, dustless surface, and from this he inferred that it seemed most improbable that wingless aphids might crawl from mosaic grasses which had been cut down onto cane and infect it.

Winged forms of *A. maidis*, as indicated by Hadden (185) were probably the main vector of mosaic, as they were produced by the drying of the host, and by the ripening of the host as the seeds were produced. Thus in the field, grasses which were weeds were generally left until they had gone to seed before they were cut down. By this time the aphids
on these grasses had developed into winged forms and deserted the
drying host, flying to sugarcane and other grasses. He concluded
that winged aphids might fly with the wind for miles and therefore
were probably the main agent in the spread of mosaic.

Van Bremen, as reported by Brandes 1927 (56), and by Ingram
et al, 1939 (193), found that winged individuals could occur in all seasons
in Java, and that flights of *Aphis maidis* coincided with periods of heavy
mosaic spread. Ingram et al 1939 (193) conducted experiments to de-
termine whether similar flights occurred in Louisiana. They concluded
that there was no mass flight of any aphid species in the sugarcane
section.

Summers et al 1948 (347), in studies conducted near Houma,
La., observed that the intensity of secondary spread of mosaic naturally
varied from year to year with the population and relative efficiency of
the aphids and with weather conditions. Furthermore, there was a very
close correlation between the number and proximity of infection foci and
the extent of mosaic spread to nearby healthy plants of sugarcane. The
movement of viruliferous vectors was decidedly limited in scope and
proximity of infection foci was the controlling factor in the rate of
spread. They further stated that possible occurrence of mass flights
of viruliferous vectors (not, however, found in their study) might also
tend to minimize the significance of this local plant-to-plant spread.
They suggested that in the absence of direct information on vector move-
ments, the extensive local spread observed by them must have been
caused largely by non-viruliferous aphids, whether from a local or a
distant source, first colonizing the plants, and then, after becoming
infected, transferring the disease from the mosaic cane to nearby
healthy cane.

Ingram et al 1951 (192) stated that the rate of mosaic spread in a given field depended on the number and species of the various carrier insects present, the species and abundance of alternate hosts of both the insects and the disease, the strain or strains of mosaic present, and the susceptibility of the sugarcane as governed by the variety, rapidity of growth, and age.

Difficulty was obtained in transmitting the disease by the aphids to cane plants over two months old. In this case transmission could be accomplished only after the cane had been heavily cut back, exposing the tender inner leaves of the spindle to the proboscis of the aphid (185). As it was usually in younger canes that weeding was necessary, and as some of the cane was always cut at the same time, ideal conditions for the transmission of mosaic by aphids were produced (185).

Hadden (185), working with corn aphids, indicated that in the warmer months it was difficult to transmit the disease, but in the cooler winter months mosaic was readily transmitted. The maximum temperature for the transmission of infection was probably near the average temperature of the three coolest months in Hawaii.

The mechanics of inoculation with sugarcane virus by *Aphis maidis* was described by Brandes 1923 (54). He pointed out that the beak of the aphid was usually placed on the cuticle covering a stomate guard cell at the point where the cuticle was the thinnest and the seta was thrust into the latter by pressure. During this process a copious secretion was excreted at the end of the seta from the salivary glands. This secretion continued to pour from the tip of the seta as the latter passed into the deeper tissues and formed the sheath described by
Bütschen (54) in his work on honey dew. The secretion usually poured into the practically uninjured and growing tissues of the leaf (54). This secretion was the actual medium by which the virus was introduced into the plant, (54), (57), (42).

The phloem was invariably sought by the insect for food (54). In every instance a dark saliva penetrated to the sieve tubes and companion cells of the phloem. The evidence indicated this part of the fibro-vascular bundle as the path along which the virus was transmitted through the plant, (42), (58).

Morphological characters of leaves might prevent the aphid from inserting the beak and thus transmitting the mosaic disease virus. These characters, as indicated by Hadden (185), include the number, size (thickness) and hardness of leaf hairs; distance between the vascular bundles and their corresponding stone and sclerenchyma cells; the size of the groups of stone and sclerenchyma cells; the distance between the phloem and epidermis; and the thickness and hardness of the epidermis.

Hadden 1928 (185) thus indicated that H. 109 was one of the varieties of cane which was more resistant to mosaic, and the fact that the leaves (especially the sheath) were heavily covered with stiff, large hairs, might be the reason that it was so resistant; for the aphid became helplessly lost and were unable to approach near enough to the epidermis to sink their beaks into the phloem.

Other insects beside the corn aphids have been reported to transmit the mosaic of sugarcane. In addition to Smyth's work reported above (309), the corn leaf hopper *Peregrinus maidis* was found by Kunkel 1922 (211) to be able to transmit the mosaic from corn to corn, but not
from corn to sugarcane or from sugarcane to corn. Kunkel (211), also stated that the cane leaf hopper, *Perkinsiella saccharicida* Kirk, did not carry the virus.

Ingram and Summers 1936 (194) proved that the rusty plum aphid *Hysteroneura setariae* Thomas, was capable of transmitting the disease.

In a series of experiments it was markedly less effective in transmission of the disease, as only 24 out of 419 healthy sugarcane plants exposed to viruliferous *H. setariae* became infected (5.2 per cent) as against 17 out of 69 (23.3 per cent) healthy plants for *A. maidis*. The fact that *H. setariae* was generally distributed on plums throughout most of the United States, with grasses as alternate hosts, and that it fed on the sugarcane (usually at the collar lobe at the junction of the leaf blade and the sheath) throughout the year might account for the specific instances of sugarcane mosaic spread in early summer in the absence of *A. maidis* which was present only in the winter and in early spring (194).

*Toxoptera graminum* Rand was reported by Ingram and Summers 1936 (194) to transfer the mosaic in two cases. In 1938 they (195) reported additional evidence which established this green bug as a vector of sugarcane mosaic. On the basis of insect-population counts and of observations they stated (195) that *T. graminum* was of less importance in field transfer of sugarcane mosaic than the other two vectors (*A. maidis* and *H. setariae*). However, Ingram and Summers called attention to the fact that under certain weather, host-plant, or other conditions it might become the most important vector in some fields.

Tate and VandenJberg 1939 (353) in Puerto Rico reported that sugarcane mosaic was successfully transmitted by *Carolinaea cyperi* and *Hysteroneura setariae*. The percentages of inoculated plants which
became infected were 31.3 and 8 respectively, while the *Aphis maidis*
used as control showed 34.5 per cent infected plants. In most cases a
considerably higher percentage of transmission was obtained on plants
grown from seeds than on those grown from cuttings.

Ingram et al 1939 (193) reported that *Hysteroecura setariae*
showed a consistent preference for the variety Co. 290 over Co. 281.
This preference apparently was due to the larger collar lobe borne by
Co. 290, which afforded a large food area and greater protection from
winds for this aphid. No varietal preference has been observed among
other known and possible vectors. Ingram et al 1951 (192) stated that
the sedge aphid carried the sugarcane mosaic disease from infected to
uninfected plants. Although the sedge aphid has not been observed on
sugarcane in Louisiana or elsewhere in continental United States, it
does occur on a sedge that grows in sugarcane fields, and undoubtedly
moves to sugarcane plants from this host (192).

In concluding this review on the vectors of mosaic, the point is
to be emphasized as to which vector is the most important one. Ingram
et al 1939 (193) and 1951 (192) conducted studies on the known and
possible vectors, and indicated that the corn leaf aphid was the most
efficient insect vector of mosaic in Louisiana, owing to its habit of
feeding mostly in the tender and disease-susceptible leaves of the central
whorl of its host plant. The rusty plum aphid was the least efficient among
the three species of aphids that seemed to be chiefly responsible for
transferring mosaic to sugarcane, but its greater numbers, especially
on variety Co. 290, probably made it the most important species in many
fields. Mosaic spread by this aphid was thought to be mainly from cane
to cane, although there was undoubtedly some transfer from wild grasses
to cane. The green bug, *Tetramyris graminum*, was a more efficient vector of mosaic than was the rusty plum aphid, but was less so than the corn leaf aphid. Mosaic transfer was both from cane to cane and from grass to cane. Since crabgrass was one of its favorite host plants its transfer of mosaic from this grass to cane was considered to be of much importance (193).

Transmitting the disease to healthy plants by methods other than insects was attempted as early as 1903 by Kamerling in Java. Brandes 1920 (52) had secured infection by inoculating healthy plants with juice expressed from diseased plants. Matz 1933 (243) indicated that the injection was presumably done by a hollow-needle syringe, since Kamerling stated that he had followed the technique employed by Beijerinck (44) in transmitting tobacco mosaic. However, Kamerling in his publication admitted that some of the control plants contracted the disease (243), (301). Van der Stok, 1907 (331), Kobus 1908 (207) and Wilbrink and Ledeboer 1910 (374) working also in Java, were unable to produce the disease by repeating Kamerling’s experiments (52).

Stevenson 1919 (329) conducted extensive studies on inoculation methods. He expressed the juice used in inoculations by grinding the stalks in a food chopper. Hypodermal injections were made in the buds, in the growing point and at the internodes. He inoculated stalks of various ages from 6 inches high to mature cane, but he got negative results. He was also unsuccessful in transferring the disease by using other methods, as inserting small bits of diseased tissue into various parts of normal canes; or by rubbing growing tips of a number of healthy stalks after a diseased tip had been crushed in the fingers.

Tower 1919 (361) attempted to produce the disease by crushing
and rubbing mealy bugs from diseased cane on buds and shoots of healthy cane, and by forcing the crushed bugs into punctures made in the buds and shoots. His results were negative.

Matz 1919 (241) split the seed pieces in half, tied a diseased half and a healthy one together and planted them, but the disease was not transmitted to the healthy cane. He was also unsuccessful in transmitting the disease by watering healthy seed pieces with water in which diseased cane was allowed to stay for some time, and by inserting buds from healthy seed pieces in diseased seed pieces and vice versa.

Hypodermic injection was attempted by Matz (241), using juice exposed to the air. Cane stalks grown in three pots were cut back, leaving stumps about four inches above ground. There were shoots about six inches high emerging from the base of stumps. The juice was injected by a hypodermic needle into the stumps near the surface of the ground. The three shoots in one pot developed mosaic. He conducted other similar experiments but the percentage of infection was very low.

Matz 1919 (241) tried another method of inoculation. Seed pieces of mature cane were cut to one or two eyes and were inoculated near the base of the bud by boring holes into the seed piece three-fourths inch deep and into them was put juice from diseased cane. Also, stools in a field that had just been cut were inoculated with juice in the stubble near the bases of sprouting buds. In both cases no mosaic developed. He finally concluded that the exact method to insure "takes" was not known.

Earle 1919 (133) conducted twenty-one experiments on inoculation methods. He tried rubbing or otherwise lacerating healthy leaves with diseased tissue, but no mosaic developed. Four experiments
consisted in binding pieces of diseased tissue in contact with cut surfaces of healthy stalks. Out of eleven such attempts one was successful. Diseased tissues were dropped into the inrolled terminal leaf spindle so as to lie in contact with unwounded young tissue. Out of sixty-five attempts seven positive cases resulted.

A hypodermic needle was thrust into the soft tissue near the terminal bud of a diseased cane and was immediately inserted near the base of the inrolled leaf spindle of a healthy cane. There was no transmission in fifty attempts.

Exposure of the cane juice to the air was considered as a possible factor affecting the vitality of the mosaic virus. Earle (133) noticed that mosaic was spreading in the fields by secondary infection, and that insect carriers seemed to be the only logical cause, while only a few successful cases were obtained by artificial inoculation; thus Earle suggested that a sucking insect flying from a diseased to a healthy plant and again feeding might regurgitate a minute quantity of the diseased juice without having exposed it to the air. So the juice was extracted from diseased cane under oil. In this connection, Sein 1930 (301) stated that upon the suggestion made by Mr. F. A. Lopez Dominguez, Prof. Earle and Mr. E. D. Colon extracted the juice from the tender parts of mosaic cane plants by grinding with a paste under a layer of mineral oil in a porcelain mortar. The juice was then taken up with a syringe, the needle of which was inserted through the oil layer, and injected immediately into healthy cane plants. With this method Earle (133) obtained five infections out of ten inoculated plants. The experiment was repeated twice, but no infection occurred.

Juice exposed in the open air was also used by Earle (133) to
inoculate healthy plants, using a hypodermic needle and thrusting it into the leaf spindle above the terminal bud. Unexpectedly he obtained mosaic infection in two cases out of seven attempts. He concluded that although successful artificial transfers of the disease had been made by various methods, the results had not been uniform and complete failure often resulted. Sein 1932 (302) in discussing the effects of exposure of the juice to the air concluded that there was no evidence that the air directly destroyed the virus, but that it was likely that the oxidation of the medium, that is the juice, directly destroyed the virus.

Smyth 1920 (310) crushed the juice of diseased plants into the leaf tissues of very young vigorous plants, and in other experiments he spread finely-cut diseased tissue, either in juicy or dry condition, over the healthy plants and over the soil surrounding them. In all cases no mosaic developed in the healthy plants.

Brandes 1920 (52) succeeded in transmitting the mosaic disease directly from diseased to healthy sugarcane without the aid of insects. The inoculum was prepared by grinding young tightly rolled leaves of diseased Rayada cane in a food chopper and straining through several thicknesses of cheese cloth. Undiluted juice was used for inoculation immediately after being prepared. One plant out of ten developed mosaic when the youngest leaves were inoculated by numerous needle pricks. Also, two plants out of ten developed mosaic when the plants were inoculated by injecting 0.5 cc. of the sap into the growing point with a hypodermic syringe.

Brandes 1920 (52) prepared another inoculum by extracting the juice from the youngest joints, and to prevent oxidation it was pressed out under a cover of mineral oil and was also used for
inoculation immediately after it had been obtained. He was able to obtain mosaic in eight out of ten plants by injecting 0.5 cc. of extract into the growing point with a hypodermic syringe. No infection of cane was obtained when the juice (inoculum) was rubbed on the unbroken surface of young leaves with fingers into either unbroken or needle-scarified surfaces of young leaves. Brandes 1920 (52) concluded that the sugar-cane mosaic virus was highly infectious only when exacting demands in the matter of favorable conditions were satisfied, and that the cell sap of diseased plants was infectious when introduced in the proper manner.

Lyon 1921 (229) in Hawaii used juices extracted by pressure from various parts of affected canes, but more particularly from tissues adjacent to the growing point of the stem. Inoculation of healthy plants was attempted by applying these juices externally to all parts, and introducing them internally at various points in the stem, eyes and spindle with a needle-syringe. The experiments were conducted in the open under field conditions and mosaic appeared on such a number of untreated shoots as to invalidate the evidence of artificial infection. In the few experiments in which canes grown in tubes and carefully isolated in a glass house were employed, only negative results were obtained.

Kunkel 1921 (210), in Hawaii rubbed the inoculum into wounded leaves of healthy sugarcane plants. The leaves were wounded by crushing them between finger and thumb. The wounded tissue was inoculated by rubbing it with a small piece of absorbent cotton saturated with the juice of diseased plants. He conducted three experiments, each with six plants, but reported no infection except from the third experiment, after a lapse of about three months, when five of the plants showed mosaic symptoms.
Bruner 1922 (78) in Cuba conducted several inoculation experiments. In needle inoculations eight out of one hundred succeeded when the needle was rapidly inserted into the midrib of the leaf after passing through a living affected leaf. Inoculations of the growing point with the expressed juice of the top of the cane taken into a hypodermic syringe without exposure to air mostly failed, but two out of thirteen succeeded in one series and three out of ten in another. When no precautions were taken to avoid contact with air, three out of fifteen succeeded.

Bruner 1922 (78) further found out that when no precautions were taken to avoid contact with air, three out of fifteen succeeded in one series, which was exactly the same number of successes as in a set of fifteen inoculated simultaneously without exposing the juice to the air. Provided the inoculations were made rapidly there seemed to be no advantage in excluding air.

Bonazzi 1926 (47), in Cuba, tried a new method of inoculation. Mature canes from plants known to be healthy were divided into as many pieces as there were internodes with uninjured buds. From each cane the terminal and basal portions were separated and planted immediately to serve as controls. In the remaining pieces a hole was bored with a sterile 3 mm. cork-borer diagonally into the node, immediately above the leaf scar and as close as possible to the bud, care being taken not to injure the latter. A small piece of fresh apical bud tissue from an infected cane was introduced into the hole thus obtained which was then closed by the disk of tissue excised by the cork borer and hermetically sealed with a small cotton plug soaked in melted paraffin. Planting was carried out soon after inoculation.

Bonazzi (47) further stated that the infected tissue for inoculation
should be removed from a freshly exposed apical bud only a few seconds before use, the cut being renewed for each new inoculum in order to avoid undue aeration. Bonazzi (47) obtained a high percentage of infection in the treated seed pieces and none in the checks.

Dr. M. T. Cook as reported by Sein, 1930 (302), working independently in Puerto Rico, devised a method similar to that of Bonazzi (47). A one-bud seed piece was cut out of a healthy stalk leaving the bud in the center and part of the internodes at each end. One end was then hollowed out by removing with a knife the spongy tissues down as far as the bud or close to it. A plug was prepared from the uppermost joints of the mosaic cane stalk by removing the hard outer covering and this plug was then forced into the healthy cylinder. The grafted seed piece was then planted. A high percentage of infection in susceptible varieties was obtained by using this method.

Fawcett 1928 (167), in Argentina, as reported by Sein 1930 (301), working with extracts of diseased tissues, succeeded in transmitting the disease by wetting the leaves of healthy cane shoots ten to twenty centimeters high, while he was unable to produce the disease in shoots fifty centimeters high. In another experiment he was also successful in transmitting the disease by the inoculation of between one and two cubic centimeters of virulent juices into the tender leaves of the spindle a short distance above the growing point of shoots 20 to 30 centimeters high.

McRae and Subramaniam 1928 (263) in India, transmitted the disease successfully by obtaining juice from crushed diseased leaves which was then immediately pricked into leaf sheaths and stems of healthy plants. The disease passed from a sugarcane variety into other sugarcane
varieties, from sugarcane into corn and sorghum and also passed from corn infected from Co. 210 to other corn.

Sundaraman 1928a (350), in India, reported that sugarcane mosaic virus was successfully transmitted by hypodermal injection and by placing plugs of diseased cane tissue in setts used for planting.

Wilbrink 1929 (373), in Java, stated that mechanical transmission of sugarcane mosaic would take place on the cutting knife. Thirty healthy plants of E. K. 28 growing in ten tubs were cut down when about three months old, a sterile knife being used on the cane in half the tubs and one contaminated with mosaic on the remainder. The knife was contaminated by splitting a mosaic top of P.O.J. 36 with it. The tubs were then kept in an isolated place in the open. Later, the sprouts of the ratoons in two of the tubs containing canes cut with the infected knives developed ten and twelve diseased sprouts respectively, the stools in the other tubs remaining healthy.

In a further experiment Wilbrink (373) used the variety D. L. 52. Five adjacent rows each containing ten healthy stools of six-months-old plants were cut down, the cut canes being used as seed pieces; a sterile knife was used in three of the rows, and a contaminated knife on the remaining rows. Adjacent plots were then planted with the seed pieces, each with the cutting of one row. About 15 per cent of the plants in the plots planted with seed-pieces cut with the infected knife showed infection.

In a third experiment, in which the seed pieces were planted in an insect-proof stage, some of the stools from the canes cut with the contaminated knife showed mosaic. This was considered by Wilbrink (373) as evidence that sugarcane mosaic could be transmitted on the cutting knife. Sein 1930 (301), pointed out that he had made a few unsuccessful tests of this
method.

Sein 1930 (301), applying the principle used by Earle (133) and Bruner (78) announced a new mechanical method for artificially transmitting sugarcane mosaic. The leaf-slip method could be used for inoculating plants two feet or more in height. The expanded leaves were bent down to expose the central spindle. The outermost leaf, the basal part of which was still tightly wrapped round the spindle, was stripped off, exposing the whitish cylinder of tender leaves. A slip about one inch wide and eight long was stripped from a mosaic leaf, placed like a band around the base of the exposed spindle, and held tightly with the thumb and forefinger of the left hand. A pin was thrust rapidly in and out through different parts of the mosaic band into the healthy spindle. The pin was thrust in a slanting position and was not made to go clear through the spindle. After the pin had been thrust in about twenty times, a fresh part of the mosaic band was used and the pricking repeated at four or five points along the spindle. Using a bunch of pins tied up into a brush made the inoculation work progress more rapidly.

Sein (301) pointed out that ordinary pins had proved worthless and suggested that black number 0 and white number 2 Asta insect pins be used for ordinary routine work, although a finer pin than black number 0 would produce higher percentages of infection. Sein (301) stated that by using this method the diseased material was carried almost directly into healthy tissue without any considerable exposure of the material to the air. Mats 1933 (243) pointed out that if exposure to air was really detrimental to this virus, the air between the two leaf surfaces might be sufficient to affect the virulence of the minute quantity of infected juice carried by the needle, no matter how firmly the band of diseased
leaf was held by the fingers against the leaf cylinder. Matz (243) further indicated that the advantage of using a very fine needle by Sein would come from the fact that the very small stab lesion resulting from the puncture would cause very little injury, and that the virus would thus be introduced directly into an environment of living cells.

A modification of the Sein's leaf-slip method was used in Hawaii. Carpenter, as reported by Martin 1933 (234), used an electrically operated needle to speed up the inoculation process. He succeeded in transmitting mosaic to different varieties. In British West Indies another modification of Sein's method was tested (291). A long strip of diseased leaf was cut, doubled in half, placed around the central unrolled leaf spindle of the healthy plant, and held between the thumb and forefinger and then pinched, after which inoculation was effected, in the usual manner, with a bunch of No. 000 insect pins thrust through a cork and projecting about 3 in. Preliminary tests (291) demonstrated the satisfactory nature of this technique.

Matz 1933 (243) described a new inoculation method devised by him, in which a drop of inoculum obtained by crushing young mosaic infected suckers of P.O.J. 234 was deposited with a pipette in the wedge shaped opening on healthy canes between the youngest expanded leaf blade and the next younger leaf of the same side, which was still rolled. A fine needle point, No. 216, special Minuten Nadeln, set into a glass rod, was then passed horizontally, or somewhat obliquely downward through the liquid and into the submerged area of the still rolled leaf. In addition several vertical cuts with the needle were made through the leaf tissue in order to allow the contact of the virus with the severed transverse connections of the vascular bundles. Matz (243) further stated that the
stabbing operation was repeated rapidly about five or six times with gentle pressure; each needle thrust formed a new puncture, but all punctures were close together and were covered by the inoculum juice. With fresh or properly stored inoculum, Matz was able to obtain a high percentage of infection, although the juice left remaining at the point of inoculation was never protected from the air. Matz (243) concluded that this method was suitable for young sorghum and Digitaria plants as well as for sugarcane.

Matz 1934 (244) using his method, found that in a given shoot the juices from infected expanded lamella of young and mature green leaves, midribs, leaf sheaths, and true stems, separately, showed an equal degree of infectiousness (80 to 100 per cent), whereas in a parallel series of inoculations a relatively low measure of virulence (0 to 50 per cent) characterized the juices from the rolled inner white and brittle portion of the leaf bases in the lower region of the pseudo-stem. There was no reduction in virulence on adding equal portions of the juice from the latter region or from the healthy green foliage to virulent green leaf juice.

Rafay 1935 (277), employed a modification of the Matz method. Twenty black entomological Pins No. 10 were mounted in a cork, which served as a handle. With a piece of cotton three to four drops of the inoculum were rubbed on the first open leaf and the spindle, the rest dropped into the hollow between the spindle and the open leaf. Inoculation was made by pricking with the sterile pins through the juice dropped on the leaf. They were pricked ten to twenty times so that each plant received about 200 pricks.

Matz 1935 (249) observed that within the sugarcane plant and
immediately above the apex of the stem the white, brittle portion of the inner leaf bases was less receptive to artificial infection with active virus extract than surrounding tissues and in infected plants the former possessed comparatively weak virus potency if used as a source of inoculum.

Abrasives were used in virus investigations as an aid in mechanical transmission by Fajardo in 1930 (162). Some viruses that were difficult to transmit by rubbing leaves with infective sap were readily transmitted if a suitable abrasive was incorporated in the inoculum (43).

Sand was the first abrasive to be used (162). Fajardo 1930 (162) found that the most satisfactory method of obtaining a high percentage of successful transmissions of bean mosaic was by rubbing the surfaces of the leaves with muslin dipped in inoculum containing sand. Samuel and Bald 1933 (298) found that the presence of fine sand (120 mesh) in the inoculum enormously increased the number of successful primary infections on the smooth leaves of *Nicotiana glauca*. The presence of sand also considerably increased the number of infections on the hair-covered leaves of tobacco and *N. glutinosa*.

Carborundum was used as an abrasive by Rawlins and Tompkins 1934 (287) who found that sap from lettuce infected with tomato spotted wilt virus, and from beans, celery and cabbage infected with various viruses, rarely gave infections with ordinary inoculation methods but readily did so when carborundum was present.

Histological studies conducted by Rawlins and Tompkins 1936 (288) demonstrated that in leaves inoculated by the carborundum method the epidermal cells frequently were pierced by the carborundum crystals and that the points of the crystals were small relative to the size of the
cells. They concluded that the evidence favored the hypothesis that the carborundum crystals pierced leaf cells and allowed the virus particles to enter but in certain cases did not injure the pierced cells sufficiently to prevent virus multiplication in these cells. Costa 1944 (111), working on Nicotiana, found that carborundum had no effect on the virus and that the action of carborundum was on the test plant and that the particle size of the carborundum was important.

Bain 1944 (37) conducted experiments for using abrasives to inoculate sugarcane seedlings with sugarcane mosaic virus. Finely ground, unwashed, white sea-island sand of 100-mesh grade to which inoculum was added was picked up between the thumb and index finger and rubbed on the spindle. He also used 100-mesh carborundum in the same way. Enough pressure was exerted by the fingers to rupture the tissues to the extent that they appeared to be water-soaked. No effort was made to proportion the amounts of inoculum and abrasives, but enough of the former was added to make a mixture of thin consistency. The seedlings used were two months old and 3-5 inches tall of the cross Co. 231 x U.S. 1694. Fifty plants were inoculated by using the sand and another fifty plants using the carborundum as abrasives, while 100 plants were inoculated by the needle-prick method.

Bain (37) found that comparatively high percentages of infection were obtained with both sand and carborundum as abrasives, the percentages of plants in each instance being more than three times as great as by the needle-prick method. However, he called attention to the fact that the seedlings used differed genetically in their susceptibility to mosaic and strictly comparative data for the methods could not be obtained. Bain (37) also pointed out that one man could inoculate by the
abrasion method approximately as many plants in a day as four men could
by the needle prick method.

Plants grown from cuttings (two months old) were inoculated
with the abrasion method. Bain (37) stated that the results were not
consistent. Infection was obtained in some tests but not in others. He
pointed out that apparently the maturity of tissue and possibly the particle
size and cutting quality of the abrasives influenced the results. Abbott
1949 (12), also found that as plants increased in age and size before
inoculation the abrasion method became less effective, and that with
plants several months old, higher infection was obtained with the needle-
prick technique.

Seedling inoculation with sugarcane mosaic virus in the germi-
nating flats prior to potting was attempted by Abbott 1949 (12). The un-
folding leaflets of the plants in the flats were crushed between small,
flat pieces of wood covered with fine sandpaper (which was more rapid
than rubbing with fingers) and then atomised with the inoculum. Infection
comparable with that obtained by other methods was obtained. Abbott
(12) concluded that this technique was not suitable as a routine procedure
because it was difficult to avoid uprooting some seedlings, and practically
impossible to give uniform treatment to all plants.

Saint 1945 (297) reported on experiments for the development of
a satisfactory technique for the artificial transmission of infection, and
on experiments carried out in an atmosphere of high humidity during and
after inoculation, which had given promising results. He concluded that
the maintenance of high humidity for 12 hours before and six after inocu-
lation was sufficient to insure the development of a reasonable incidence
of infection.
A comparison of the different methods of artificial inoculation has been carried out by certain investigators. Matz 1933 (243) using the method of inoculation developed by him, obtained 6 infections out of 11 inoculations of Louisiana Purple cane, whereas, 10 similar inoculations without puncturing the leaf tissue resulted in no infection. Employing a very fine needle gave a higher percentage of successful infections than did a coarse dissecting needle. Similar results were obtained by inoculating Black Amber sorgo with sugarcane mosaic virus. In another experiment with sorgo, out of 12 tests, the syringe method of inoculation gave only two infections while Matz's method gave eight. By merely rubbing the inoculum into young leaves of sorgo by means of a glass rod without puncturing the tissue, out of 12 tests, 8 were successful. However, Matz (243) stated that this rubbing method was not usually so efficient in transmitting the disease.

Stevenson 1939 (322) in Barbados compared the efficiency of four methods of inoculation, the Sein leaf-slip method, the Matz method, the hypodermic needle method in which the inoculum was injected into the shoot near the growing point, and the plug method with cuttings. In the first three methods the plants were inoculated when they were between one and two feet in height. In the plug method, a single eye cutting of a healthy cane was used, and a plug of tissue removed with a sterile 3/8 inch cork borer as near as possible to the bud. Into the cavity thus made a plug cut from the leaf spindle of a diseased shoot with the same cork borer was introduced, and crushed with a sterile glass rod. The original plug was then replaced, sealed with paraffin wax, and the cutting planted and allowed to grow.

Stevenson 1939 (322) found that the Sein method was the most
successful (44 per cent infection); with the plug method only moderate success was obtained (20 per cent infection), and no infection was obtained by the other two methods. Matz, in the discussion that followed the presentation of Stevenson's paper (322), emphasized that the size and quality of the needle used in inoculation was very important, pointing out that large needles broke down too many cells, thus preventing the virus from entering living cells.

Saint 1945 (297) in Barbados, reported on experiments for testing the efficiency of Bain's method for the inoculation of sugarcane seedlings with the aid of abrasives and found that none of the plants on which these were used contracted mosaic, whereas 80 per cent of those inoculated with the needle had become diseased. While Saint did not get any infection by using the abrasion methods, other investigators in the United States obtained successful results.

Chilton et al 1950 (95), Dunckelman et al 1951 (129) and Azab and Dunckelman 1951 (36) using the abrasion method in inoculating sugarcane seedlings from different crosses obtained in 1949, 5,814 infected seedlings out of 45,824 inoculated seedlings, and in 1950, 16,624 infected seedlings out of 51,969 inoculated ones.

Liu 1949, in China, (244) compared the various techniques for the inoculation of sugarcane with sugar cane mosaic virus, and found that a modification of Matz's method was the most satisfactory. About four drops of extracted viruliferous juice were dropped into the axil of the youngest open leaf, and the tight roll of young leaves was pricked several times through the inoculum with a steel needle (0.2 mm. in diameter). The incubation period was 19.8 days and the average infection 95 per cent.

Abbott 1949 (12) compared the efficiency of the abrasion method
and Matz's needle-prick method, using three sugarcane clones: Louisiana Purple, Co. 281 and Co. 290. Washed, 80-mesh, sea-island sand was used as an abrasive, and the plants were inoculated when they were 4 to 6 inches tall. All the plants of Louisiana Purple inoculated by the abrasion method showed mosaic symptoms, while 89 per cent of infection was obtained by using the needle-prick method. In both Co. 281 and Co. 290, 89 per cent and 86 per cent infection respectively was obtained by using the abrasion method, while 60 per cent and 44 per cent infection respectively was obtained by the Matz method. Thus, the abrasion method gave a higher percentage of infection and the differences between methods for each variety was significant at the 5 per cent level.

Abbott (12) further found that some clones became infected by the abrasion technique which would be resistant to the needle-prick method or to exposure to natural sources of the virus. He concluded that this fact, however, was not a valid objection to the practice of abrasion, since the annual surplus production of seedlings in the breeding program necessitated a certain number of rejections in any case.

Extracts from corn plants recently infected with the sugarcane mosaic virus were found to be slightly more infectious to sugarcane seedlings than that obtained from diseased sugarcane plants (112). Costa and Penteado 1951 (112) pointed out that corn plants gave more juice than sugarcane leaves, and that corn tissues were easier to grind. They concluded that the use of inoculum from corn plants was advantageous in inoculating sugarcane seedlings in breeding projects.

Strains of the sugarcane mosaic virus have been under study in recent years. Brandes and Klapaak 1923 (62), suggested the possible existence of more than one type of the mosaic disease affecting the
numerous representatives of the grass family.

Brandes 1919 (51) and Kunkel 1924a (213) described several symptom patterns of mosaic of sugarcane that appeared on the different varieties. Many variations and types of mosaic were then reported in the literature, but these were always explained as being due to differences in varietal reactions to a single virus, or to environmental conditions influencing the manifestation of a single causative agent (340), (341).

Yoder 1926 (378) discovered that some popular varieties long thought to be highly resistant, if not immune to mosaic were not, and he suggested that "a more virulent form of the disease" might have been responsible for that as well as for the wave of mosaic spread that occurred in 1925 at Cairo, Georgia.

Storey 1927 (336), and 1929 (336), reported the occurrence in Transvaal of a virus that produced symptoms in corn and sorghum spp. indistinguishable from that caused by the sugarcane mosaic virus in these hosts. Leaf-cage experiments showed that *Aphis maidis* Fitch was capable of transmitting the virus in corn. However, sugarcane failed to contract the disease, both in field and caged experiments. Storey 1929 (336) concluded that the virus occurring in the Transvaal was not virulent to sugarcane and was therefore different from the common sugarcane mosaic virus. However, Storey 1929 (336), also stated that the mosaic he had studied was not a different kind but possessed a different degree of virulence in relation to a range of host plants. Storey 1936 (337) reported that the Agaul sugarcane variety in South and East Africa was affected by a mosaic disease, the virus of which was not transmitted by needle inoculations or *Aphis maidis*; further, that there was no
evidence that spread of this mosaic disease to other sugarcane varieties had occurred. He concluded that the virus attacking this variety was exceptional in regard to its transmission, not conforming in its behavior in this respect to the other viruses of the common sugarcane mosaic group.

Tims and Edgerton 1932 (358) reported that mosaic-infected P.O.J. 213 and P.O.J. 228 cane, from Baton Rouge produced some healthy shoots. However, the proportion of such healthy shoots from mosaic-infected seed was less in 1931 than in previous years. On the other hand, mosaic-infected stalks of P.O.J. 213 and Co. 281 from Reserve, 50 miles distant, when planted at Baton Rouge produced only diseased shoots, all of which remained infected throughout 1931. They also reported that the percentage of mosaic infection in field plantings of P.O.J. 213 at Baton Rouge was between 2-5 per cent, while the natural infection in Reserve was 50 to 60 per cent. Furthermore, there was considerable infection in Co. 281 and Co. 290 at Reserve, which were practically disease free at Baton Rouge. They concluded that this difference in behavior in the two different sections suggested that there might be 2 strains of the sugarcane mosaic virus present in Louisiana.

Summers 1934 (340), reported that in 1932 he found two widely divergent types of mosaic occurring naturally on adjacent stools of a single seedling variety C. P. 28-60. In 1933, two additional types were collected in commercial field of Co. 281. All four types have persisted without apparent change through successive vegetative generations of the infected plants. These types were then inoculated separately into parallel groups of healthy plants of Louisiana Purple and four other sugarcane varieties. On these varieties, types 1, 2, and 4 were not differentiated,
while type 3 gave distinctive yellowish-white stripes. Inoculum extracted separately from the three groups of Louisiana Purple showing identical symptoms as well as from the type 3 plants and introduced into healthy groups of C. P. 23-60 invariably reproduced the typical pattern types 1, 2, and 3. Summers 1934 (340) concluded that from the consistency with which the four types had been reproduced in these experiments, a difference in causative agent, or agents would seem to have been indicated as nearly as could be determined on symptomatology. Possibly these types were the expression of individual strains, or a temporary modification of a single virus, or distinct virus diseases.

Tims 1935 (355) reported the occurrence of two types of mosaic on C. P. 28-70, a yellow or severe type causing a reduction of tonnage of 32 per cent and in sucrose of 20 to 30 per cent, and a green or mild type which apparently caused little or no reduction in growth and only a slight reduction in sucrose. He also reported that the yellow type virus produced the ordinary green type of symptom when transmitted to other cane varieties.

Definite proof of the existence of distinct strains of sugarcane mosaic virus was presented for the first time by Summers (341) at a meeting of the fifth congress of the International Society of Sugarcane Technologists, at which time he announced the occurrence of four strains of sugarcane mosaic virus in Louisiana, which were differentiated by symptom expression on 1 to 4 month old plants of C. P. 28-60 and Louisiana Purple.

Brandes 1935b (61) reported that Summers' strain 3 of sugarcane mosaic virus was readily transmissible by Aphid maidis from
diseased to healthy sugarcane. He also demonstrated that Creole corn was susceptible to the same strain but developed different symptoms from those of sugarcane, and the corn plants finally wilted and died. Needle-prick inoculations from corn leaves, before wilting, into sugarcane leaves resulted in very pronounced symptoms of strain 3 mosaic in 19 days.

Additional strains of the mosaic virus were further reported by Summers. In 1936 (342) he reported that more than four strains occurred in Louisiana and that C. P. 807, a variety long considered immune to mosaic showed appreciable infection, which suggested that another strain might be present. In 1939 (343) he divided the sugarcane mosaic virus into ten strains and substrains based chiefly on the symptoms produced upon three host varieties, C. P. 31-294, Co. 281, and C. P. 29-291 and letters (A to D) were substituted for numbers in designating these strains.

A comprehensive and thorough treatment of the studies conducted on the strains of sugarcane mosaic virus was presented by Summers et al 1948 (341). They also described the symptoms produced by the presence of certain strains on the differential cane varieties. They also gave the distribution of these strains in Louisiana and included their indices of infectivity.

The origin of new strains of sugarcane mosaic virus was suggested by Summer (341), (342) to be locally, possibly within the cane plant or by passage through other gramineous hosts. These less-favored hosts, or the insect vector (A. maidis) or even some new vector, might be fractionating a compound virus that had hitherto existed as a mixture. This would not be without precedent among virus diseases of other plants.
Mutations of a single original virus, assuming it to be a living entity, offered a further possible explanation (342).

The presence of more than one strain of mosaic virus in one plant of sugarcane was studied by Brandes (61), who attempted to superimpose Summers' strain "3" on strain "1" experimentally, using D-74 test plants infected with strain "1" six weeks previously. Insects were used as agents of inoculation and the results were negative.

Forbes et al 1937 (177) and Forbes 1939 (169), conducted two kinds of experiments, using viruses which produced yellow and green mosaic symptoms on C. P. 28-70. In the first series of experiments, sugarcane plants showing yellow mosaic were inoculated with green mosaic, and the plants continued to show symptoms of yellow mosaic. Also, plants showing green mosaic were inoculated with yellow mosaic, and the plants continued to show symptoms of green mosaic. In the second series of experiments, the plants were inoculated first with one mosaic virus either green or yellow, and 4 or 5 days later the plants were inoculated with the other type of virus, and the type of mosaic that developed in each plant was determined.

Forbes (177) and (169) concluded that the two types of mosaic did not occur together in one plant. If one of these was present, the plant was apparently immune to the other. He (169) further stated that of the two mosaics, the green seemed to be the stronger, though it produced less severe symptoms on the host. A higher percentage of infection occurred in inoculation experiments with the green virus, and the period of inoculation was shorter than in similar experiments with the yellow virus. However, another phenomenon occurred in Louisiana Purple. Plants of this variety which showed a very mild mosaic readily
became infected when inoculated with a severe strain which occurred on that and other cane varieties.

The relation of the existence of several strains of the sugarcane mosaic virus to breeding mosaic resistant varieties of cane, and to the distribution of the commercial varieties of cane have been under careful consideration in recent years. Abbott 1951 (13) stated that there have been several instances of the occurrence in Louisiana of mosaic in varieties rated as resistant to this disease. In addition to the unreleased varieties, a fairly high percentage of mosaic (20-30 per cent) had been found in the two important commercial varieties C. P. 34-120 and C. P. 36-105. Strain A of the sugarcane mosaic virus was identified as responsible for this "unusual" occurrence of mosaic. Furthermore, the collections made indicated the increasing prevalence of this strain in Louisiana. Abbott 1951 (13) also stated that the mosaic strain survey made during the past year also indicated that strain D of the virus, which was the most prevalent one in Louisiana when Co. 281 was a leading commercial variety had been superceded by strain B, which had been and still was common on Co. 290. He concluded that thus a change in varieties might be accompanied by changes in strains of disease organisms present.

Recovery from mosaic has attracted the attention of the investigators connected with sugarcane diseases. Grey 1919 (183) in Cuba, stated that repeated experiments had proven that mosaic-affected cuttings of the best field canes, when planted often produced normal plants to the extent of 100 per cent, in "the first bud shoot", and they remained free from the disease until infected by other fungus. He also stated that if the diseased plants were provided with good soil and cuttings, they would resume their normal color in two or three months, each succeeding leaf becoming less mottled. Earle 1919b (134) stated that Grey (183) was
confusing mosaic with cane root-rot and the leaf-spotting caused by insect punctures, as recovery was true for the last two diseases but not for mosaic. Earle concluded that no case had been recorded in which a cane attacked by the yellow stripe disease had ever recovered. Grey 1919a (184) published a note denying Earle's accusation. He further stated that some of the badly infected canes were cured of the mottling through the aid of good soil and proper treatment, in 115 days. Other plants equally affected returned to normal health in 59 days.

Stevenson 1919 (329) added more to the controversy of the recovery from mosaic symptoms. He stated that there had been reports of apparent recovery from the mottling condition, but such cases were considered by Stevenson to be "very doubtful at best".

Brandes 1920a (33), observed numerous cases of apparent recovery in corn, and also in crabgrass, and yellow bristle grass. The three hosts were infected with sugarcane mosaic virus. He further reported that some mosaic-infected stools of sugarcane and sorghum produced tillers with no signs of mosaic.

Lyon 1921 (229) in Hawaii, reported that mosaic-infected plants of Lihuea variety threw off the malady and produced leaves on which no mottling occurred. Cuttings from five of these plants which had recovered were planted. All top cuttings from the portions carrying healthy leaves gave rise to healthy shoots, but cuttings from the older portions which had carried diseased leaves gave rise to diseased shoots. Lyon, also noted that healthy plants were observed to develop from stubble pieces of cane that had mosaic the previous season.

Kunkel 1924a (213) observed that diseased stools of certain varieties frequently recovered. He pointed out that careful studies
showed that this might come about in either of two different ways. The diseased shoots of a stool might at times begin to produce healthy leaves. Later, the old diseased leaves died and fell off. Such stools might grow to maturity without showing any further signs of the disease on the leaves. These stools, according to Kunkel, became healthy through the recovery of the terminal buds of their diseased shoots. In the other way by which the disease might be overcome, none of the diseased shoots actually recovered, but the new shoots produced might be healthy. The diseased shoots remained small and were overgrown by the healthy ones. After a time they died and the stool might then remain healthy to maturity. Kunkel further stated that in another instance, a stool might be diseased or partly diseased in the plant crop or in one of the ratoon crops, but after this crop was harvested, it might produce only healthy shoots. Such stools might remain healthy through the next subsequent crop yields. This was the manner in which many stools were observed to recover in the experiments conducted by Kunkel (213) to test the effect of mosaic on yield. Kunkel also stated that when seed cane was taken from shoots that had been observed to recover, and planted, the young shoots remained healthy for a time, though later some developed mosaic. He considered that this was probably the result of a new infection.

Brandes 1927a (57) stated that as early as 1922 he observed the recovery from mosaic symptoms on the leaves growing on the upper joints of the variety B adila. Cuttings from these upper joints gave rise to healthy new plants. Unite and Capinpin 1926 (363), stated that within an infected stalk the buds invariably produced mosaic-infected shoots, regardless of whether their corresponding leaves showed the
Stahl and Faria 1929 (314) reported that in P.O.J. 2714 there was a drop in the number of plants showing mosaic symptoms from 16.3 per cent at the sixth month to 7 per cent at the end of the seventh month. This was attributed by him to be due to recovery. On the other hand, plants of P.O.J. 2725 showed well marked symptoms of mosaic until the cane was cut. However, the first, second and third ratoon crops did not show any symptoms.

Krishnaswami 1936 (208) in India, reported that Co. 360 and Co. 361 appeared to show genuine recovery from infection as the plants grew older and inoculation tests indicated the development of a kind of immunity.

Brandes and Matz 1935 (64) stated that in addition to examples of individual recovery they had seen mass recovery from mosaic where the plants of entire fields known to have been one hundred per cent affected later became, to all appearances one hundred per cent healthy and remained so for several years. When protected from reinfection, individual plants from such fields remained healthy indefinitely.

Several investigators attempted to explain the phenomenon of recovery from mosaic diseases. In the following paragraphs, the important contributions on this subject in relation to sugarcane mosaic are summarized.

East 1931 (137) in Cuba, reported that there might be recovery from mosaic at any time between 3 and 24 months after infection. After recovery, a plant remained healthy for several years. He also reported that a sugarcane plant might recover and be reinfected as many as three times. He set forth two hypotheses that might explain this (137), (138)

The host might kill the infective agent, throw off the symptoms of the
disease, remain in a partially immune condition for a period and then become infected. It was equally probable that the host merely reduced the virulence of mosaic virus until the latter was unable to produce the usual mosaic symptom, although the virus continued to live within the tissues. In this case the symptoms of the disease could reappear without reinfection if the resistance of the plant was lowered by unfavorable conditions. East 1931 (137) favored the second hypothesis as an explanation to what he considered the first critical evidence of the establishment of an acquired immunity to a definite infective agent in a flowering plant. He concluded that it was perhaps more probable that cane plants gained an apparent immunity by reducing the virulence of the mosaic virus than by throwing it off entirely.

Oclemia 1932 (269) received a sugarcane stalk showing mosaic symptoms. He cut four lateral shoots free from mottling, cut the main stalk into five parts, and planted all parts. Oclemia observed that the plants developing from the apparently healthy lateral shoots were free from mosaic symptoms and altogether more vigorous than those from the main stalk, which showed typical mottling, but mechanical transmission experiments from the former carried out by Sein's method on young, healthy, shoots of variety Mauritius 1900, gave a high percentage of infection. Oclemia inferred that this was an indication that the virus was present in a latent form in the lateral shoots.

Rands and Summers 1932 (286) pointed out that occasionally whole stools known to have originated from diseased seed cuttings showed no leaf symptoms whatsoever. They indicated that this might have been due to: (1) unequal distribution of the virus among the cutting buds, (2) failure of transmission, i.e. failure of the virus to spread from
the bud into the sprout, (3) temporary suppression of symptoms, or
(4) recovery of the entire stool sometime prior to the date of observa-
tion.

Randa and Summers 1932 (286) reported that Ribbon cane stools
on June 2 were fully infected, but on July 21 had from 1 to 3 healthy stalks
each. These were usually the largest stalks of the stools and therefore,
could not have been recent suckers emerging healthy from diseased
mother stalks. These were shipped to Washington, and single-node
cuttings were planted. Both healthy and mosaic plants were obtained
from the upper (presumably recovered) portion of every stalk. There
were propagated through several vegetative generations, and without
exceptions the diseased plants remained diseased, and the healthy
plants remained free from mosaic. Thus, according to the authors, at
least part of the stalk had evidently recovered from mosaic. The same
tendency occurred on three other varieties. They concluded that these
observations indicated the pronounced tendency to reduce the incidence
of mosaic brought about possibly by unequal distribution in, or incomplete
transmission of the virus by cuttings and also apparent recovery of
obviously diseased stalks. Considering the possibility of unequal distri-
bution of the virus in the stalks, Stahl and Faris 1929 (314) conducted
germination experiments on stalks of varieties P.O.J. 2725 and P.O.J.
2714, and concluded that mosaic was not distributed throughout the whole
stalk. However, they noted that their canes had very poor germination.

Brandes and Matz 1935 (64) stated that there was no relation
between recovered and diseased buds in their positions on the stalk.
In a single stalk, the recovered portions might be discontinuous or inter-
rupted, with portions intervening that gave rise to diseased plants.
Forbes and Mills 1945 (175) inoculated young plants of varieties Co. 281 and C. P. 28-19 with sugarcane mosaic virus and observed that the virus spread from an infected shoot through the old seed piece to other shoots. The virus also spread through the seed piece from the point of inoculation either toward the top or base of the seed piece. Furthermore, the virus could also pass by a node and infect other shoots further removed from the point of infection.

Sorenson 1939 (311) attempted to give an explanation of recovery from mosaic. He treated some sugarcane plants with small amounts of manganese, copper, zinc, boron, iodine, and chromium. A month later the mosaic symptoms disappeared from several of the varieties. He was of the opinion that during the first period of growth the roots were few, and present only in the upper part of the soil which might be exhausted of an original quantity of these elements, and as soon as the disease was transmitted by a puncture of a carrier the plant transmitted it easily. Later, when the roots penetrated to deeper and less exhausted layers, the elements were again absorbed and the mosaic pattern on the newly formed leaves did not show up, and the stool after a while "throws off the mosaic".

Edgerton and his associates in Louisiana conducted several experiments for studying recovery from mosaic in sugarcane. Tims and Edgerton 1931 (357) reported on the results obtained in the period between 1926 and 1930. Stalks of the varieties P.O.J. 213 and 228, which showed typical mosaic symptoms, were cut and planted. During the following year the plants developing from these stalks showed a high percentage of mosaic during the summer, but during the fall there was a material decrease. In October, only six plants out of 366 of the variety
P.O.J. 213 showed mosaic symptoms and 71 out of 221 P.O.J. 228. These diseased stalks were replanted, as well as a large number of those that showed apparent recovery. From the disease-free stalks, no mosaic plants developed, while a considerable percentage of the plants developing from mosaic stalks were free of the disease. The work was repeated and the tests showed that mosaic infected plants of P.O.J. 213 and 228 were constantly producing disease-free shoots, even after being infected for four years. Studies on the behavior of individual plants showed that a number of stalks of P.O.J. 234 and P.O.J. 36, which were free of mosaic symptoms at the time of planting, produced diseased shoots. This was attributed by the authors to the occurrence of masking of mosaic symptoms in these varieties. It was also noted that no recovery occurred in variety LSII. Tims and Edgerton concluded that the relative resistance of P.O.J. 213 and P.O.J. 234 to mosaic was due to a considerable extent to the ability of the plants to throw off the disease and to produce buds which were free of the virus.

Tims, Mills and Edgerton 1935 (360), reported on studies on recovery from 1930-1934. They observed that the P.O.J. 213 plants from Baton Rouge continued to recover, while those from Reserve apparently did not recover. Other varieties showed more or less the same behavior. They also noted that mosaic began to spread rapidly in P.O.J. 213 and other varieties which were resistant. They believed that a new or a more virulent strain of the mosaic virus became established in the district around Reserve. Thus P.O.J. 213 became more susceptible and finally ceased to throw off the disease.

Tims et al 1935 (360) also stated that the infective virus was not
always absent from cane plants which had apparently recovered from mosaic. In some cases the disease had been produced by inoculating juice from such plants into healthy plants. The percentage of infection, however, when such juice had been used, was not high.

Forbes and Mills 1943 (173) inoculated healthy plants of six sugarcane varieties with juices from: (1) mosaic plants, (2) symptomless plants grown from diseased seed pieces and (3) plants from which mosaic symptoms disappeared during the growing season. Inoculations from juice of the last two sources were not followed by a development of mosaic symptoms, while inoculations from mosaic canes were followed by a development of mosaic symptoms. They concluded that the results obtained appeared to establish the fact that no infective virus was present in the symptomless plants growing from sugarcane seed stalks originally exhibiting mosaic symptoms, nor in plants from which symptoms disappeared during the current season. They further stated that mosaic free plants grown from diseased seed pieces were not immune from reinfection by the mosaic virus.

Summers et al 1948 (347) presented a full account of the studies conducted at the Division of Sugar Plant Investigations of the United States Department of Agriculture on the recovery from mosaic disease of sugarcane. They stated that the recovery from mosaic observed during the period between 1930-32 in Louisiana was brought about in two main ways: 1) The production of healthy plants by the germination of eyes, either from planted cuttings or from stubbles in ratooning, both of which during the previous crop had supported continuously diseased foliage; and 2) the production of new foliage without mosaic symptoms by diseased plants, which, by elongation of the stalk and natural death of the
older leaves, presently showed no further signs of the disease. In the case of P.O.J. 36-M, in which the original infection dated back to about 1925, it was observed that germination recovery to the extent of about 50 per cent could be expected when pedigreed mosaic cuttings were used and that, under conditions of light secondary spread at least, an appreciable amount of foliage recovery would occur. P.O.J. 234 gave similar results, but to a much lower degree.

By following the behavior of individual stalks of P.O.J. 36-M and P.O.J. 234, Summers et al. (347) classified the stalks in respect to their mosaic history into four different categories. Group A - pedigreed mosaic stalks that usually produced both diseased and healthy plants upon germination (pedigreed plants were those grown from cuttings or stubble that had been observed regularly throughout at least one previous growing season without any change in disease or disease-free status).

Group B - Recovered stalks, stalks that showed foliage recovery. These usually produced mostly healthy plants, but frequently produced a number that were diseased. When these healthy plants were planted, they (C) produced only healthy plants, while the mosaic progeny of recovered stalks (L) behaved like the original pedigreed mosaic material.

A positive correlation between foliage recovery and germination recovery was obtained by Summers et al (347). The comparative figures for the varieties P.O.J. 36-M, P.O.J. 234, P.O.J. 213, and Co. 281 revealed that the latter two showed no foliage recovery and practically no recovery at germination, while P.O.J. 234 showed a small degree of foliage recovery and considerable recovery (up to 25 per cent) at germination. P.O.J. 36-M showed both the greatest recovery of foliage and the greatest from germination.
Considering the control of mosaic disease of sugarcane, Reyes 1927 (293) discussed the following items in relation to control of the disease: 1) quarantine (59), 2) selection of seed, 3) planting resistant and immune varieties, 4) eradication, 5) ratooning, 6) disinfection, 7) use of fertilizers, 8) elimination of weeds, 9) disease carriers, and 10) breeding work. A large amount of literature has been written on this subject. Highlights of the important bibliographies were written by Reyes 1927 (293) and by Chilton (93).

Seed selection, as a means of controlling the mosaic, was practiced early by the Javanese. Brandes 1919 (51) noted that in Java long experience had demonstrated that the disease could best be held in check by careful selection of healthy plants for seed and by replanting fields with cuttings taken from the same field. He also stated that in Hawaii the disease was controlled by selection of clean seed and the use of resistant varieties.

Stevenson 1917a (327) in Puerto Rico indicated that cuttings from mosaic infected stalks invariably gave rise to infected plants. In this connection Brandes 1919 (51) stated that the use of such infected stalks for propagation resulted in spreading of the disease to new regions and in wider distribution of diseased plants on the same plantation from year to year.

Edgerton 1920 (144) emphasized the importance of using disease-free seed. He stated that at the end of the season there was less mosaic infection than in non-selected seed. Edgerton and Taggart 1924 (155) reported greatly increased yields in plots of D. 74 and Purple cane planted with selected cane. Edgerton 1920 (144) recommended growing seed plots each fall for large scale plantings.
Edgerton and Taggart 1924 (155) stated that tests with the D. 74 and the Purple canes showed that in fields with 100 per cent infection plants could be selected that showed a marked tolerance to the disease. They anticipated that by selecting only the most tolerant canes for seed, the susceptible commercial varieties could be developed to a tolerant condition. Edgerton et al (157), (158) stated that with continuous seed selection in Louisiana Purple cane, strains were developed which showed decided tolerance to the disease. The selected strains showed mild symptoms of the disease. They also stated that by continuous seed selection increased yields of cane had been obtained, and that the stubble cane, as well as the plant cane, was improved by selection.

Reyes (293) in the Philippines stated that there was always danger of selecting some plants which appeared to be healthy, but which later, when planted, would develop the disease. He suggested that in such cases it was necessary to plant the cuttings first in an isolated nursery. He also advised the use of seed of known origin and seed selection in fields in which insect transmission and secondary infection were light.

Martin 1929 (233) reported good results in Hawaii in 1929 from selecting mosaic-free cuttings. Unite and Capinpin 1926 (363) observed that selection of healthy cuttings would lessen the spread of mosaic in the following planting season and in the ratoon plants. Pritchet 1924 (275) showed that selecting cane for planting was a promising measure for minimizing the dangerous effects of this disease.

Roguing and eradication of mosaic-infected sugarcane plants had been suggested as a means for controlling the disease. Earle 1919 (135) stated that the idea that mosaic could be controlled by a campaign
of eradication was first suggested in print by Stevenson in 1918 (Revista de Agricultura 1:23, May 1918). However, he further indicated that Mr. Enrique Landron, a cane grower in Puerto Rico, had been practicing the method for some time with considerable success, and that he should therefore have the credit of being the first to suggest it.

Earle 1919c (135) described a method of eradication, which consisted in doing two things: first, planting only healthy seed; second, when the young cane was a foot high, the field should be carefully inspected and all the plants showing signs of the disease should be pulled out. This inspection should be repeated at intervals of about ten days until no more cases were found. When a small cane was pulled the vacancy might be filled by planting a new seed piece. When the cane became larger, replanting was to be done by dividing the stools. Earle (135) further indicated that in regions of complete infection the establishment of healthy seed was necessary before a campaign of eradication could be undertaken. He also reported success in reducing mosaic in Puerto Rico by careful and systematic roguing. Earle concluded that the disease could be controlled by this method. However, he also stated (133) that complete eradication of the disease by this process was very difficult. Rosenfeld 1924 (294) stated that Earle's process of roguing became very effective in controlling the disease in the lighter infected areas in Puerto Rico. Johnston (202) pointed out that where a field was free from outside infection that this method had been a complete success.

Brandes 1919 (51) suggested that roguing be practiced in the spring, just as soon as all the plants had sprouted. A second inspection should be made after 25 or 30 days, and an additional inspection could be made if necessary. He also stated that roguing was not recommended
for fields in which the number of infected plants exceeded 5 per cent in half-grown to mature cane or 20 per cent in young plants just sprouting. In large fields where the proportion of diseased plants was greater than 20 per cent, roguing was impracticable. In such cases, plants should be allowed to mature, but no cane from such fields should be saved for seed.

Ramos 1927 (280) stated that mosaic disease could be economically controlled in sugarcane by seed selection and roguing. His observations were made in Cuba, Santo Domingo and Puerto Rico. Storey 1926 (334) mentioned eradicating mosaic in a large area in Natal by roguing. Faris 1931 (164) reported good results in Cuba by using roguing for controlling mosaic. Martyn 1946 (238) reported successful roguing in Jamaica. Laws were enacted in Jamaica in 1936 and 1942 (85) making the roguing of young cane (less than 4 months old) compulsory, and forbidding the use of planting material from fields with more than 10 per cent infection. McClean 1932 (250) and 1932a (251) pointed out that legislation to eradicate all cane varieties other than Uba was introduced in 1927 in South Africa. This failed in its main purpose, and there had been annually sporadic outbreaks of mosaic in certain susceptible types. Infection might have been derived from wild grasses or from susceptible canes which survived when planted with Uba. He concluded that eradication had been instrumental in reducing considerably the sources of mosaic infection and had created a situation permitting of complete control of cane varieties which might be grown in Natal.

Steel in 1939, as reported by Tims 1939, (356) stated that in Natal systematic roguing reduced the infection with mosaic to a very low point.
Johnston 1923 (202) stated that other sources of infection were frequently present around cane fields, thus rendering imperative the necessity for constant vigilance and repeated inspections, which increased the expense. Johnston thought that perhaps it was for this reason that some planters in Puerto Rico had given up the system of roguing, and that it was the general tendency in all cane countries where this disease occurred to supplement control measures with the substitution of resistant if not immune varieties.

Reyes 1927 (293) stated that in some countries roguing proved disappointing because of secondary infection. If this happened, the insects should be destroyed by chemicals. He concluded that if insects could not be controlled the roguing became futile.

Brandes and Mats 1935 (64) stated that the use of roguing was obviously indicated where areas were newly invaded and the numbers of infected plants were small, and in areas where epiphytotic conditions were temporarily halted, offering the chance of effective work in reducing the danger of massive spread, if subsequently conditions became favorable for rapid secondary infection. He concluded that during a fulminating epidemic it was practically useless to attempt control by roguing.

Denely 1937 (120) was considered by Shaffer as the first to report extensive roguing tests in Louisiana. He reported that roguing could be successfully done in sections where the natural spread of the disease was slow, but that in fields with a high percentage of mosaic the cost was too high. Marier 1937 (232) in Louisiana gave results obtained from roguing, but his data was inconclusive. Abbott et al 1937 (15) mentioned that great benefits had been obtained from roguing and seed selection of Co. 281 on several of the large plantations in Louisiana.
Edgerton et al 1937 (153) classified cane varieties into three groups. First, these varieties which became infected readily with mosaic and did not throw off the disease after becoming infected. Such varieties showed the most unsatisfactory behavior towards mosaic. Second, those varieties which did not become infected readily but did not throw off the disease after becoming infected. The mosaic could probably be controlled very readily in such varieties by roguing (153). Third, those varieties which did not become infected readily and also throw off the disease after becoming infected. Roguing should control the disease readily in these varieties and probably even would not be necessary (153).

Shaffer 1939 (303) and 1939a (304) pointed out that the amount of mosaic spread in the field was proportional to the amount of infection in the cane used for seed. Seed selection alone reduced the percentage of mosaic in non-isolated plots to one-third or one-fourth that present in the field run cane. He also observed that cane varieties differed in the amount of roguing required. The less mosaic-susceptible varieties required very little roguing. Variety Co. 281 required the most roguing among the varieties tested. He concluded that a combination of seed selection and roguing seemed to be a practical measure of mosaic control. He further stated that the practicability of selecting seed plots and roguing for mosaic control depended largely upon the cost of such operations. The cost of such roguing would depend on the amount of mosaic in the fields, the rapidity of spread, and the cost of labor.

Disinfection as a method of controlling the mosaic was tried by Brandes 1919 (51). Seed pieces of cane were soaked in strong Bordeaux mixture or corrosive sublimate previous to planting, but all the shoots
that came out were diseased (51). Reyes (293) stated that hot water at 50–55 degrees centigrade for 30 minutes, and at varying degrees of temperature and length of immersion had been tried in some countries against mosaic, but the treatment failed to cure the diseased cuttings.

Introduction of cane varieties resistant to mosaic has been tried as a measure for controlling the disease. Reyes (293) summarized the work done and the success obtained in this field in different countries of the world. Stevenson 1949 (324) pointed out that mosaic was thus responsible for major varietal changes in Louisiana, Puerto Rico, Natal, and several other countries.

Breeding cane varieties resistant to mosaic was used as a method of controlling this disease. As the literature in this subject deals in some of its phases with breeding cane varieties resistant to the red-rot disease, the literature on breeding resistant varieties for both diseases will be reviewed simultaneously, after the following review on red-rot disease.

Red rot disease of sugarcane caused by *Physalospora tucumanensis* Speg., is one of the major diseases of sugarcane in the world as well as in Louisiana. It attacks and invades the stalks, stubble rhizomes, leaf blades, leaf sheaths, leaf midribs and roots.

The red rot was first reported as a disease of sugarcane by Went in Java in 1893 (371). Went 1896 (372) stated that the disease was first observed during the grinding season of 1892 on the Tjomal estate in Java. In the following year Went (371) published a description of the symptoms of the disease, and of the causal organism which he called *Colletotrichum falcatum* Went. He produced the disease by infecting healthy canes with pure cultures of the fungus and called the disease "Rood Snot".
Masses 1893 (239) working at the Botanical Garden at Kew, England, isolated *Colletotrichum falcatum* Went from sugarcane stalks sent to him from Barbados. He considered this fungus as the causal organism of the so-called root disease in the West Indies. Furthermore, Masses 1893a (240) reported *Trichosphaeria sacchari* Masses as causing the stalk rotting in the West Indies. He also described a *Melanconium* stage as the first stage in the life cycle of *Trichosphaeria* and that the conidia of *Melanconium* produced the so-called macro- and microconidia which in turn characterized the ascigerous stage of *Trichosphaeria*.

Went 1896 (372) published the results of his studies in English and called the "Rood Snot" disease "Red Smut". He stated that the "red smut" attacked the interior of the stems of the sugarcane. Externally no trace of its existence could be discovered, except when the canes were badly attacked, in which case the leaves died. But as soon as the stems were split longitudinally red spots were to be seen on the sections. The true symptoms characteristic of the disease were white blotches extending mostly in the direction transverse to the stalk. He further cited Masses as attributing *Colletotrichum falcatum* Went as the agent which caused the "root disease" of cane in the West Indies. Went proceeded to state that he had received the fungus from the West Indies and identified it as the same fungus, *Colletotrichum falcatum*, found in Java. Went expressed the belief that the so-called root-disease - a disease which looked like the "sereh" in Java - was caused by *Colletotrichum falcatum*.

Thiselton-Dyer 1900 (354) considered that the "root disease" and the "rind disease" in the West Indies were due to one organism,
Trichosphaeria sacchari Masse. Howard 1900 (189) published his first report in his attempts to repeat Massee's work under tropical conditions, and in 1903 he concluded (190, 191) that the red smut disease of Java was identical with the rind disease of the West Indies and was caused by the fungus Colletotrichum falcum Went. He also stated that the invasion by Melanconium sacchari Masse. was secondary following the injury by Colletotrichum falcum.

Tryon 1901 (362) was of the opinion that what had been described as a severe-rotting of cane in Queensland as early as 1895 was probably red rot.

Barber 1901 (39) reported the disease in India. He (39), (40) observed that it was severe in regions where borers were almost absent. He also noted that the thin varieties of cane were more resistant to red rot than the noble varieties. Furthermore, he believed that the apparent immunity of certain cane varieties could be broken down by bad cultivation.

Cobb 1906 (102) in Hawaii, reported the "rind disease" as attacking the cane in Hawaii, but he could not isolate Colletotrichum falcum from any diseased canes in Hawaii. Thus he considered this fungus to have no role in the "rind disease" in Hawaii. Lewton-Brain 1907 (219) in Hawaii, stated that the rind disease was caused by Melanconium sacchari Masse., and that it was distinct from red rot caused by Colletotrichum falcum Went.

Butler 1906 (86) reported on his extensive studies on the disease in India and proposed the name "red rot", which was adopted by other investigators throughout the world. He also stated that in India most of the infection of the growing plants was due to direct mycelial connection between the stalks and diseased cuttings. This was in accordance with Raciborski's work in Java (276). Butler further found that the diseased
stalks were lower in sucrose than the healthy ones. He indicated that the fungus was responsible for inverting the sucrose into glucose, while the actual consumption of sugar by the fungus was relatively slight. He also observed the disease to occur on the leaves and the roots. However, he stated that on the leaves the fungus did not pass back through the leaf sheath to the stem.

Lewton-Brain 1908 (220) gave a full account of the disease, reporting that it was of little importance in Hawaii.

Edgerton 1910 (139), 1910a (140), discovered the red-rot disease in the United States in 1908. In 1911 (141) he reported on his extensive studies on the disease. He did not agree with Butler's observations that a direct mycelial connection existed between the growing plant and seed cuttings. This statement lead Butler and Haflz Kahn 1913 (84) to make an extensive study and they confirmed the earlier statement of Butler. Kulkarni 1911 (209) in India confirmed Butler's results.

South and Dunlap 1913 (313) in the West Indies confirmed Edgerton's observations. Edgerton and Moreland 1920 (154) attributed the different behavior of the fungus in the U.S.A. and in India to the possibility of different strains of the fungus or to varietal susceptibility or to climatic conditions. McMartin 1943 (255) in South Africa confirmed Edgerton and stated that there had been no case in which the fungus penetrated from the infected cuttings to the inside of the stems arising from these cuttings. He further stated that the young stems were surrounded at their base by disintegrating infected material which might prove a source of infection for penetrating the stem from outside when conditions were suitable. Steib 1949a (318) confirmed this opinion. He observed that healthy shoots attached to the seed piece developed disease in
storage. Careful studies showed that infection occurred at the base of the shoot at the point of contact with the old bud scales.

The red rot disease began to get world wide attention and reports of its occurrence in different countries included those of Johnston 1911 and Johnston and Stevenson 1917 (204) in Puerto Rico, South 1911 (312), Ballou 1913 (38), Johnston et al 1917 (203) in the West Indies, Stockdale 1915 (16) in Mauritius and Averna Sacca 1916 (35) in Brazil (cited by Johnston and Stevenson 1917).

Martin 1951 (237), and Stevenson and Rands 1938 (330) stated that the red rot disease of sugarcane had been reported from the following countries: Angola, Antigua, Argentina, Australia, Barbados, Brazil, British Guinea, British Honduras, China, Columbia, Cuba, Dominican Republic, Egypt, El Salvador, Guam, Fiji, Formosa, Guadeloupe, Haiti, India, Indochina, Jamaica, Japan, Java, Madagascar, Madeira, Malay States, Mauritius, Mexico, Mazambique, Okinawa, Peru, Philippines, Puerto Rico, Reunion, Samoa, Siam, St. Kitts and Nevis, St. Lucia, Trinidad and Tobago, Union of South Africa, United States of America.

Edgerton 1951 (149) pointed out that in the early years following 1909, the research in Louisiana with red rot followed the pattern which was characteristic of pathological research of that time. The life history of the causal organism was studied and attention was paid to the losses caused by the disease and to the environmental factors which seemed to be favoring or retarding its development. In later years, investigations were intensified on problems concerned with host-parasite relationships and with certain known phases of the life history of the fungus. Edgerton concluded that it was believed that additional information in regard to how the fungus entered the sugarcane plant and how it spread in the
tissues after entering, as well as more information in regard to the fungus itself might be useful in formulating more satisfactory control measures. The following paragraphs contain a brief résumé on the points outlined by Edgerton.

The perfect stage of the red rot fungus was first reported in Louisiana by Edgerton and Carvajal in 1943 (150), and described in detail by Carvajal and Edgerton 1944 (86). They found the perithecial stage in the field on dead leaf blades and leaf sheaths that were beginning to dry. The perithecia were abundant on leaf sheaths, on the underside of midribs, and on both surfaces of the rest of the leaf blade. The perithecial stage was identical with a fungus described by Spegazzini in Argentina in 1896. They concluded that the perfect stage of the red rot fungus should be named *Physalospora tucumanensis* Speg. Furthermore, the perfect stage was also developed from a single conidium as well as from single ascospore cultures. The pathogenicity of the fungus was proved by the use of single ascospore cultures as well as by several conidium cultures.

Ling and Ma 1951 (223) reported the occurrence of the perfect stage of the red rot fungus in Taiwan, China. They found that conidia could be produced from germ tubes of ascospores of *Physalospora tucumanensis* Speg., when enough nutrient was supplied. They considered this as one of the best proofs of the connection between the imperfect and perfect stages of the organism. They found the perithecia on the dead and drying leaves in the field from November to May. In laboratory tests, using spores of *Colletotrichum falcatum* Went, they observed that dead and drying leaf blades were favorable to the production of perithecia, but there were less perithecia on the midrib, leaf sheath, green leaf blades,
and bagasse, and the perithecia were not formed on the spindle, rind, fresh part of the stem, or the root. Many perithecia were formed on the dry leaf blades but there were less on the green leaf blades, where an abundance of slimy pink masses of conidia formed. They concluded that the difference in behavior on the green leaf blade could be related to the different nutritive value. The imperfect stage of the fungus grew vigorously on the better substrate, but perithecia developed on the media which were less nutritive. They further stated that this conclusion was confirmed by their findings that the perithecial development was more profuse on the green leaf blades from which the water soluble matter had been removed by boiling in water and less on the dry leaf blades with sucrose solution added.

Ling and Ma 1951 (223) using different light filters of hyaline (check), white, black, blue and red, found that the production of perithecia did not show any significant differences under various light filters.

Wang 1951 (390) in Taiwan (China) reported on his studies on the perfect stage of the red rot fungus. He stated that some varieties might yield an abundance of ascospores, while others might produce little or no perfect stage material. In addition the size of the perithecia might vary in different varieties.

Tims and Edgerton 1932a (359) suggested the possibility of the presence of specialized or physiologic races of *Colletotrichum falcatum* in Louisiana. Abbott 1933 (6) reported the presence of such forms in Louisiana. He distinguished them on the basis of their pathogenicity. He developed an index by calculating the ratios of lateral and longitudinal spread of the lesion to varying dimensions of the inoculated internodes.
By multiplying the ratio for length by that for width a figure was obtained which was called "index of virulence". Abbott 1935a (8) reported on two morphological groups of *Colletotrichum falcum* which were distinguished on the basis of the color and texture of the mycelium. The light type was more virulent on the resistant variety Co. 281 than the dark one. Abbott 1938 (10) reported on extensive studies on this subject. He reported that the comparative virulence of both light and the dark cultural races of the fungus was determined on a resistant host Co. 281, and a very susceptible one P.O.J. 213. On these hosts, the isolates of the light race from Louisiana were more virulent, in general, than those of the dark, and the latter were more virulent than either the light - or dark - race isolates from the sirup - producing states. The representative of the two cultural races tested from the sirup-producing states were of about equal virulence. He concluded that there was a relation between virulence and the geographic origin of the isolates with respect to the principal sugarcane growing areas of the United States.

After the work on the physiologic forms of the fungus in Louisiana was published, other reports came from different countries. Among those reporting were Rafay and Padmanabhan 1941 (278), Ramakrishan 1941 (271), Mundkur 1946 (248) and Lo 1947 (225).

Red rot causes considerable injury to the cane, reduction in yield, losses in stand, injury to leaves and losses in sucrose. Edgerton et al 1924 (156) reported that the red rot epidemic in 1923 caused disastrous losses in the crop. Chilton and Mills 1947 (96) reported that Co. 290 and C. P. 29-320 canes inoculated with red rot in 1946 gave 1.4 and 9.9 tons per acre respectively while the check plots gave 27.1 and 20.3 tons per acre. In the period of 1943-1946, the average reduction in yield of
inoculated plants was 64 per cent for Co. 290 (a susceptible variety) and 2 per cent for C. P. 36-13 (a resistant variety).

Germination of buds was reported to be affected by red rot. Edgerton and Moreland 1920 (154) stated that germination of the buds was reduced nearly 50 per cent when seed cane was inoculated with spores of the fungus. The importance of red rot as a disease of cane has been considered also by Edgerton 1928 (145), 1928a (146), Edgerton and Flor 1928 (152), Edgerton et al 1924 (156), Edgerton and Tims 1927 (158), Abbott (4, 5, 7).

Went 1896 (372), stated that the serious damage of red rot was due to the deterioration of canes and reduction in sugar content. Edgerton 1910 (134) stated that this disease affected the stands by rotting the seed cuttings. Tims and Edgerton 1932a (359) estimated that the stands of cane in 1924 were reduced nearly 50 per cent by the disease; Edgerton et al 1934 (159) and Edgerton 1938 (147) considered red rot as one of the factors causing stubble deterioration. They pointed out that the fungus grew in the stubble pieces and caused a decay. Abbott 1935 (7) pointed out that decimation of stands due to red rot caused reduction in sucrose content and purity of the cane juice. This was explained by the more abundant tillering induced by the gappy stands, resulting in later maturity of the cane.

Sucrose was reported by Went 1896 (372) to decrease in plants affected by red rot. In the epidemic year of 1927 in Louisiana, the reduction in sucrose in juices of the noble varieties was as high as 33 per cent (289). Bourne 1934 (49) reported on an epidemic of red rot in P.O.J. 2714 in Florida in 1934 which caused a 30 per cent loss in tonnage and a reduction of about 50 per cent in the sucrose of the harvested cane.
Inversion of sucrose in cane stalks infected with red rot was reported by Butler 1906 (82). Lewton-Brain 1908 (220) grew the fungus *Colletotrichum falcatum* Went in sugar solutions. He concluded that the fungus produced the enzyme invertase which caused the inversion of sucrose. Further, the enzyme was contained in the mycelium, and the fungus itself excreted the enzyme into the medium. Edgerton 1911 (141) stated that the sucrose content of the diseased internodes was reduced as well as that of the joints above the diseased ones.

Edgerton 1910 (139) pointed out that the possible methods of infection of sugarcane by red rot fungus were as follows: (1) direct infection without the aid of injury, (2) infection through burrows made by borers or through punctures by other insects, (3) infection by the fungus growing directly into the stalk from the diseased leaves, and (4) the growth of the fungus from the diseased seed pieces up into the plant. Considering the first point, Went in 1893, (371) tried to inoculate the unwounded rind, but no infection was obtained except when very young internodes were selected. Later, Went in 1896 (372) stated that *Colletotrichum falcatum* was only a wound parasite and that sound canes which had not been damaged could not be attacked by the fungus with the exception of the very young parts of the stem. However, he pointed out that these parts were usually protected by the surrounding sheaths of the leaves. Prinsen Geerligs 1898 (274) pointed out that wound infection would not sufficiently explain every case of attack. Cited by Butler and Hafez Khan 1913 (84). Lewton-Brain concluded that the fungus was essentially a wound parasite and that there had been no record of it being found able to penetrate the unbroken rind of the cane.

Edgerton 1911 (141) pointed out that as the rind tissue was hard and thick it was hardly possible that the fungus infected the internodes
without the aid of injury. He further suggested that at the node, where
the rind tissue was broken by leaf traces and rootlet buds, there was a
possibility that the fungus entered without the aid of injury, but he did
not get complete evidence. South and Dunlop 1913 (313) stated that the
fungus was a facultative wound parasite and infective chiefly through
wounds.

Butler and Hafiz Khan 1913 (84) stated that the fungus pene-
trated the "uninjured" feeding roots and extended freely in the tissues
of the root. The hyphae also penetrated the adventitious root eyes on the
stalk. They also stated that the forms of the fungus that attack the leaves
and those that attack the stems penetrated the uninjured leaves. Penetra-
tion occurred through the motor cells of the midrib, while no penetration
was observed through the stomata. When the leaf was first wounded,
Butler and Hafiz Khan (84) found that penetration was not confined to the
thin-walled motor cells but occurred freely into the epidermis of the
midrib just beyond the wounded parts.

Edgerton and Carvajal 1944 (151) gave a detailed account on the
host-parasite relations in red rot. They found that the conidia when
placed on an uninjured epidermis germinated and produced a single-
celled brown appressorium and became cemented to the substrate. Few
lesions were observed on the upper surface of midribs, but no mycelium
was found in the cells and no infection threads were found in the thickened
walls of the epidermal cells.

Spore suspensions were dropped behind the leaf sheaths of young
active leaves. The appressoria sent infection threads that penetrated the
epidermal walls of the leaf sheaths. Edgerton and Carvajal 1944 (151) did
not observe any erosion in the cellulose wall around the infection thread
and thus there was no evidence that the thread went through a pore dissolved out by an enzyme. The infection threads penetrated the walls in a straight line, and generally either perpendicular to the leaf surface or parallel to the cross walls of the subepidermal layers. Direct penetration by pressure on the cell wall did not seem reasonable without twisting and binding of these straight infection threads. The authors suggested that the demonstration of pores, pits, or at least lines of cleavage perpendicular to the surface would be helpful in explaining these straight infection threads.

Borer holes were suggested as the chief channels through which the red rot fungus gained entrance to the plant. Went 1893 (371), 1896 (372) pointed out that natural infection occurred chiefly through holes made by boring insects. Butler 1906 (82), and Butler and Hafez Khan 1913 (84) doubted whether borers played any important part in introducing the fungus into the cane in India, although agreeing to the possibility of wound infection and that in nature borers were the only means by which the wounds penetrating the pith were made.

Lewton-Brain (220), differed from Butler and Hafez Khan. He stated that the fungus had to be introduced artificially or naturally into a wound that went through the rind into the softer tissues of the stalk. He considered that the borers were the ones that caused these wounds and concluded that three factors were necessary for a successful infection: 1) The presence of a wound, 2) the presence of spores or mycelium of Colletotrichum falcatum in the wound, 3) A susceptible condition of the cane. He further emphasized the importance of undertaking measures for controlling the borers. He was of the opinion that control of borers meant control of external attacks of red rot, just as seed selection would control the internal attack.
Edgerton 1911 (141) stated that the cane borer *Diatraea saccharalis* F. made a large opening in cane stalks and thus the fungus could enter. Further, as the borer passed up and down through the stalk it carried along the fungus and produced a general infection in the whole stalk. Abbott 1938, was in agreement with Edgerton that in Louisiana and Florida infection of the growing stalk occurred through the tunnels of the moth borer. South and Dunlop 1913 (313) found that in Barbados and St. Kitts, the fungus gained entrance largely through borer holes.

Abbott 1938 (10) stated that the underground portions of the stalk might become infected through injuries made by the sugarcane weevils (Anacentrus sp.). Infection from this source seldom extended into the above-ground internodes. The principal effect of infection of the rhizome was that it might lead to stubble deterioration.

Root primordia have been described as a point of entrance of the fungus. As early as 1898, Prinsen Gearlgs (274) pointed out that such infection would not sufficiently explain every case of attack cited by Butler and Khan 1913 (84). Edgerton 1910a (140) found that in the canes received from Georgia, infection had occurred without borer injury. He suggested 1911 (141), that infection might have taken place at the nodes where rind tissue was broken by leaf traces and rootlet buds. Butler and Hafes Khan 1913 (84) found that the root primordia admitted the fungus and that they were more readily infected than the buds.

Abbott 1938 (10) confirmed this opinion and stated that this type of infection was limited largely to certain very susceptible varieties, as P.O.J. 213 in Louisiana and P.O.J. 2714 in Florida. In the sirup-producing areas infection through root primordia and subsequent spread in the stalks usually did not occur early enough in the season to lower seriously the
quality of the cane for sirup. Padwick 1940 (272) confirmed Abbott's
results and reported that in the epidemic of 1939 in India, the type of
root primordia infection had been found on P.C.J. 213. Chona and
Padwick 1942 (100) found that a considerable amount of secondary infec-
tion took place through the upper portion of the cane. They found no
correlation between borers and secondary infection. They suggested
that the infection was through the nodal region. Nesom 1939 (266) found
that in planted seed cane infection was obtained through injured buds and
root primordia, but no evidence was obtained to indicate the penetration
of the fungus through the uninjured buds and root primordia. Steib 1947
(316), 1948 (317), and Steib and Chilton 1951 (320), 1951a (321) isolated
the fungus from the root band region, even after 24 hour immersion in a
solution of bichloride of mercury. Nevertheless, he concluded that the
initial point of entry was through the bud and leaf scar and not through
the root band region.

Infection through the bud was reported by Butler and Hafez Khan
1913 (84). They inoculated the bud with spores by rubbing them with the
fingers and kept the buds moist by moist cotton. After three days the
hyphae were found to have penetrated the bud and to be growing vigorously.
However, they reported that the uninjured buds were less readily infected.
Steib 1947 (316) found that the penetration of the epidermis of young bud
scales by infection threads occurred 33 hours after inoculation with
conidial suspensions of the fungus. In C. P. 34-120 appressoriums were
formed on the surface of bud scales and from the surface small pegs were
found penetrating the wall of the epidermal cells. In cases in which the
pegs had entered the inside of a cell, a cap or overgrowth had formed
around the peg. Penetration was limited to the surface cells in areas in
which the cell walls were thick and extended to four or five cells below
the epidermis in areas without thick-walled cells. Steib 1948 (317),
stated that the peg and overgrowth were observed in both resistant and
susceptible varieties.

The seed pieces are liable to be infected through the cut ends
(10). Edgerton and Moreland 1920 (154) pointed out that the ends of the
seed cuttings soon became overgrown with yeasts and other organisms.
Tims and Edgerton 1932a (359) pointed out that cut ends of cane stubbles
were exposed to invasion of fungi, bacteria and yeast in winter. Most of
these organisms penetrate only the portion of the internode left exposed,
but the red rot fungus, once established in the tissues, might spread
through the nodes and kill the entire stubble piece. However, Tims and
Edgerton stated that evidence indicated that much of the infection in stubble
piece occurred through the root buds. Abbott 1938 (10) conducted labora-
tory inoculations on cuttings of P.O.J. 213 and Co. 281 and observed that
the fungus spread through the nodes from the cut ends and invaded the
internodes. In similar experiments conducted by him in the field, only
a small percentage of the cuttings showed penetration of the fungus from
the cut ends through the nodes and into adjacent internodes. Abbott (10)
concluded that extensive examination of seed cane of several commercial
varieties in plantation fields over a period of several years indicated that
this source of infection was of relatively little practical significance.

Butler 1907 (83) pointed out that when the red rot had been
severe, a large number of the stools left in the ground after cutting the
crop would contain the fungus. In the second crop grown from these
stools the fungus might remain dormant for some months, but, as the
cane matured, the parasite resumed activity. Chilton et al 1947 (97)
stated that many stalks which were apparently healthy were already infected before harvesting. This occurred even when canes had been treated with various fungicides. Steib 1949 (316), 1949 (318), confirmed this and concluded that infection remained in a latent form during the growing season until dry conditions or other environmental factors reduced the vitality of the stalk, allowing further invasion by the organism.

The leaf sheath plays an important role in the red rot problem. McMartin 1943 (255) reported in South Africa that infection had been found in the leaf sheath, where it produced lesions not unlike large eye-spot markings. These lesions have been proved to be of *Colletotrichum falcatum*. This discolored area extended to the young soft rind of the young internodes.

As the leaf sheath pulled away from the stalk the spores of the fungus could find their way in the space between the leaf sheath and the stalk. This region was a moist chamber which provided ideal condition for infection of the sheath. Edgerton and Carvajal 1944 (151) found that spores introduced behind actively growing leaf sheaths produced appressoria and infection threads. When the sheaths pulled away from the stalk the fungus could work down to the buds, root band and sheath tissue, where it joined the stalk. Thus, if the leaf sheaths were tight to the stalk no infection occurred. This was confirmed by Chilton and Steib 1947 (98) as they could not isolate the red rot organism from buds and root bands of varieties Co. 290 and C. P. 34-120 when the leaf sheaths were tight to the stalk.

Leaf sheaths also have their importance as the spores of the red rot fungus produced on them could serve as a source of inoculum throughout the growing season. This was confirmed by Abbott 1938 (10)
and Steib 1947 (316). The latter stated 1949 (318) that removal of the leaf sheaths from the stalks before they became infected greatly reduced the amount of latent infection in the nodal region. However, this was not true for the buds of Co. 290. Out of 20 buds planted in oat agar, 22 per cent gave red rot fungus. He attributed that to the protrusion of the buds from stalks after the leaf sheaths had been removed. The spores of the fungus could lodge between the bud and stalk, and when they germinated they could infect the young buds.

The leaf scar was suggested by Went 1893 (371), to be permeable to red rot infection. South and Dunlop 1913 (313) reported that inoculations on the leaf scars and between the leaf sheaths and the stem failed. Butler and Hafez Khan 1913 (84) reported that old leaf scars were not readily infected. They reported on Howard's results 1903 (190) in obtaining infection through the leaf bases and they considered that this difference probably depended upon the degree to which abscission had progressed at the time of inoculation. They concluded that under normal conditions leaf scars were not exposed until the leaves had completely withered, and such scars were not readily infected. However, less completely withered leaves were sometimes torn away and the scars left were a source of danger.

Abbott 1938 (10), pointed out that leaf scar infection had not been reported in the United States. Padwick 1940 (272) suggested that infection might take place through leaf scars. Steib 1949 (318) stated that in all the varieties tested, the initial points of infection could be traced to the buds and leaf scars.

Leaves of sugarcane were reported to be infected by the red rot organism (82), (267). Butler and Hafez Khan 1913 (84) and later Abbott 1938 (10) found that infection might take place through the apparently
uninjured epidermis. It was suggested earlier that infection of stalks might take place by the fungus growing directly into the stalk from the diseased leaf. Nesom 1934 (266) found that the organism migrated from one part of the leaf to the other through the ligular region, but no such migration was obtained between the leaf and the stalk. The migration probably took place by means of spores which were carried through the vascular bundles.

However, Abbott 1938 (10), stated that the disease on the leaves probably had little effect on the growth of the plants, although it sometimes caused premature drying of the lower leaves of some varieties. He emphasized the importance of leaf lesions as a principal source of inoculum for stalk infections and for disseminating the disease during the growing season.

Padwick 1940 (272) summarized the sources of the fungus as follows: (1) mycelium in the mother sets, (2) spores from diseased sets, (3) spores from leaf spots, (4) spores from old decayed diseased canes, (5) spores or mycelium in the soil, (6) spores from alternate host, if any.

It has been shown that mycelium in the plants stays in a latent form until conditions favor the vitality of the cane (318). The effect of the mycelium in the mother sets will be on the seed piece itself. For example, it will lower the percentage of germinating buds (10), and some varieties which possess resistance in the field become susceptible to red rot in seed cuttings. Abbott 1935 (7) noticed that C. P. 807 cuttings became very susceptible to red rot in the semi-dormant condition, and the infection resulted in lowering their germination.

The common practice in Louisiana is that the seed pieces are laid down with many of the leaves and sheaths adhering to the stalks. This
will furnish the soil with an abundant supply of *Colletotrichum falcatum*. Further infection may then take place from this inoculum through borer holes or through the nodes.

Leaf infections have their importance as they furnish spores for disseminating the disease throughout the growing season (10). Fruiting begins 10 to 14 days after the leaves have been inoculated (10). Carvajal and Edgerton (86) discovered the perithecial stage of *Colletotrichum falcatum* on the dead leaf blades. Leaves on shoots killed by too much crowding were found covered with perithecia. Generally as the tissues lost their chlorophyll and died, the perithecia developed and matured. The isolates of the fungus obtained from leaf lesions have been proved by Butler 1906 (82) and Abbott 1938 (10) to cause red rot infection in the stalks. However, the presence of the disease on the leaves did not indicate its presence in the stalk or the susceptibility of the stalk to the disease (10). Abbott 1938 (10) stated that the multiplication of the fungus on the leaves might be of considerable importance when a variety very susceptible to leaf infection was introduced to a section where another variety might be very susceptible to the disease in the stalk, but had normally little leaf infection.

Presence of the spores on the mycelium of *Physalospora tucumanensis* speg (Colletotrichum falcatum Went) and its role in furnishing inoculum has been discussed by Butler 1906 (82). He stated that the fungus could be present in the soil for 3 to 4 months. However, Abbott 1926 (1) could not isolate the fungus directly from the soil. This source of inoculum was considered unimportant in Louisiana because the seed pieces were subjected to a mass inoculum when they were in the field as growing cane and because the leaves on leaf sheaths adhered to them
after planting. In India the case was different, as Chona and Padwick 1942 (100) concluded that soil infection could play a definite role in the spread and perpetuation of the disease.

Resistance of cane varieties to the red rot disease has been studied under Louisiana conditions by several investigators. Abbott 1933 (6) stated that there were two types of resistance shown by cane varieties to this disease. First, functional resistance, possessed by varieties germinating quickly and forming vigorous roots, thus preventing the entrance of the fungus through the root primordia. Second, physiologic resistance, in which the development of the fungus within the tissues of the stalk was retarded, once infection had taken place.

Abbott 1935 (7) conducted inoculation experiments on several varieties of cane and concluded that the results of the field experiments were in agreement with those obtained in the laboratory with regard to the relative susceptibility of the varieties and their actual field performance, except for C. P. 807 and Co. 290 which were more resistant to red rot when actively growing than when in a dormant condition. He also stated that C. P. 807 was able to resist infection through the root rings to a much greater degree than P.O. J. 213. Invasion of the stalks of C.P. 807 through the root primordia occurred if the young rootlets were injured, or if the cuttings were subjected to conditions that induced dormancy and prevented root development over a prolonged period. Abbott 1935 (7) concluded that this type of resistance, which inhibited or prevented infection at the nodes, or by quick germination permitted the plant to escape the disease, was of considerable significance, even though the same variety was unable to resist the spread of the fungus when entrance had been gained. Abbott 1938 (10) stated that the laboratory
results, demonstrating that this type of resistance (or escape from infection) might be broken down under certain conditions, were confirmed by actual field experience with C. P. 807 in 1935 and 1936, when prolonged dormancy of the planted seed cuttings in heavy soil on several plantations was followed by red rot invasion of the stalks through the nodes. Abbott 1938 (10) concluded that while such functional resistance or escape from infection was influenced by environmental conditions and could not be depended upon to protect the plant against the disease under all conditions, it might be of considerable importance in the protective mechanism of some varieties. He further stated that commercial experience with sugarcane varieties made it evident that resistance of the tissue to invasion as well as to development of the fungus after infection was of primary importance in combating red rot.

Atkinson and Edgerton 1937 (34) stated that when cane plants were inoculated in the field in summer, it required 3-4 months for infection to show for more than 2-3 internodes away from the point of inoculation. When stalks were cut at planting time in the fall and inoculated the disease spread in 3-5 days through the stalk. The spread was faster than the expected growth of the mycelium. Atkinson and Edgerton (34) and Atkinson 1939 (33) demonstrated that the spores of the fungus could be carried through the vessels by the transpiration system. Further, Atkinson pointed out that the spore migration was found to be the factor responsible for extra nodal longitudinal spread.

Atkinson 1939 (33) stated that cane varieties showed two types of resistance to spread of red rot in the stalks, namely, resistance to longitudinal spread and resistance to lateral spread. He stated that the vessels became discontinuous primarily at the base of the node. In the
inoculated stalks, the node below the point of inoculations had many lesions, due to the blocking of spore passage at the base. The node above the point of inoculation, however, had lesions only along vessels that were continuous through the node. He observed that in some varieties more of the fibrovascular bundles were continuous as they passed through the nodes than in other varieties. Atkinson concluded that resistance to longitudinal spread was a type of morphological resistance, while resistance to lateral spread was a type of physiological resistance. He expressed the belief that the first type of resistance was more stable than the second one, which might vary considerably with different physiologic forms.

Abbott 1938 (10) stated that in both standing cane and cuttings the red rot fungus developed more rapidly in P.O.J. 213 (a susceptible variety) than in Co. 281 (a resistant one). Within the invaded cells of P.O.J. 213 the hyphae developed rapidly, branched profusely, and frequently produced an abundance of conidia. In Co. 281, the progress of the fungus was much slower. Usually short lateral branches were produced in the invaded cells and conidial production was scanty. He concluded that his observations indicated that the resistance principle was contained within the protoplasm.

Abbott 1938 (10) stated that red rot resistant varieties showed a higher phenol content than the susceptible one. However, he pointed out that larger numbers of varieties in both red rot classes should be studied before making a generalization.

Classification of the cane varieties according to their resistance to red rot was attempted by Abbott 1935 (7) who suggested the following classes: resistant, moderately susceptible, susceptible, and very
susceptible. Later, Abbott 1938 (10) suggested the following classes:

Class 1. Resistant to nodal infection and to spread in the tissues.

Class 2. Moderately resistant to spread in the tissues; resistant to nodal infection. Class 3. Susceptible to spread in the tissues, but characterized by temporary check of the fungus at the nodes; resistant to nodal infection. Class 4. Very susceptible to spread in the tissues with little or no checking of the fungus at the nodes; resistant to nodal infection under most conditions. Class 5. Very susceptible to spread in the tissues and to nodal infection.

Abbott 1938 (10) concluded that any attempt to classify sugarcane varieties according to red rot resistance must be arbitrary to a certain extent. He further stated that because of the great importance of red rot as a seed-cane disease, this point was particularly stressed and the classes of resistance had been defined with special reference to this type of injury.

Abbott 1946 (11) stated that there was no such thing as immunity to red rot and few if any cane varieties were so resistant to this disease that their resistance could not be broken down by unfavorable soil and weather conditions. He further indicated that the variability of the fungus causing red rot was so great that it made it more difficult to develop cane varieties that were resistant to all forms of this fungus, and greatly complicated the problem of breeding for red rot resistance.

Kiryu 1940 (206) reported a method of varietal resistance trials of sugarcane to red rot. Fragments of four day old cultures were inserted into apertures 7 mm. in diameter punched in the center of the internode. Thirty to forty stalks of each variety were inoculated. Two to three months later, at harvest time, the inoculated stalks were split lengthwise and the
extent of the diseased tissue was measured. The resistance of a given
variety to the pathogen was compiled by the "theoretical figure" de-
A  given from the number of diseased internodes. When the area of the
affected portion of the inoculated stalks comprised more than 2/3 of
an internode, it was placed under the sign (+++), between 2/3 and 1/3
under (++), and below 1/3 under (+). The number of infected internodes
in the (+++) category was then multiplied by 3/3 and those in (++), and
(+) by 2/3 and 1/3 respectively. The degree of damage (b) was arrived
by dividing the totals of the three categories of each variety by the
number of stalks measured.

Environmental factors were reported to influence the develop-
ment of red rot in the stalks. Abbott 1938 (10) stated that after the seed
cuttings were planted, soil temperature had an important bearing on the
development of red rot within the stalk. Contrary to what might be ex-
pected from the fact that the optimum temperature for Physalospora
tucumanensis Speg. was relatively high (30° degrees C.), the disease
was more destructive to seed cane when soil temperatures following
planting were low rather than when they were high. During the late fall
and winter months, soil temperatures low enough to partly or completely
prevent the growth of sugarcane might not inhibit the red rot fungus.
Since the optimum for this fungus is about 5 degrees C. below that for
cane, it would be able to develop more rapidly than cane at temperatures
below the optimum, such as 15 to 20 degrees C., at which temperature the
cane remains inactive. Abbott concluded that field observations and lab-
oratory experiments have shown this to be true. He further stated that
a single freeze might destroy all the advance the sugarcane plant had been
able to make during the winter, whereas red rot was not so affected.
Further, cold weather might check the spread of red rot, but did not wipe out the progress it had already made.

Abbott 1936 (9) reported that C. P. 29-320 gave satisfactory stands on light, well drained soils. On heavier soils, germination was poor because of red rot. Abbott 1938 (10) stated that in Louisiana red rot was most severe on the heavy soils, which were usually poorly drained. Such conditions favored the disease through the retarding influence they had on the normal growth activities of the sugarcane plant, as well as by the direct favorable effect of high moisture on the fungus. There were also reports that high soil moisture content favored red rot as a cane disease.

Deep covering of seed pieces was reported to favor the development of red rot. Abbott 1936 (9) stated that deep covering not only prolongs the dormant period of the seed by keeping the temperature below the point of at which germination occurred, but by doing so it favored the spread of red rot, which developed more rapidly in dormant than in actively growing cane. Forbes and Gouaux 1943 (172) recommended that seed pieces of variety Co. 290 should not be covered with more than 3 inches of soil.

Abbott 1938 (10) stated that field injury from red rot was influenced by many factors, such as the vigor of the variety, susceptibility to borer attack, susceptibility to root rot, the type of soil in which the cane was planted, drainage, the preparation of the seedbed, as well as weather conditions. He further stated that these factors were not taken into account in determining inherent resistance or susceptibility to the spread of the disease in the tissues, but they must be considered in evaluating the commercial possibilities of a variety.
Various control methods have been suggested for controlling
the red rot disease of sugarcane. Planting red rot free seed and setting
aside certain fields of cane to be used for seed purposes as means of
controlling red rot were suggested by Barber 1901 (39), Edgerton 1911
(141), and Edgerton et al 1924 (156). Lewton-Brain suggested that con-
trol of borer would mean control of external attacks of red rot, just as
seed selection would control the internal attacks. Abbott 1935 (7) and
1936 (9) suggested that cultural practices that tended to stimulate
germination of seed pieces would help in reducing red rot injury to
seed cane. He suggested a thorough preparation of the seed bed well in
advance of planting time, and the avoidance of too deep covering of the
seed at planting time. Rands and Abbott 1937 (281) and Abbott 1938 (10)
suggested summer planting of cane, when excellent germination could be
obtained, as a means of escaping considerable injury from seed-rotting
diseases which affected canes planted in the fall. However, Arceneaux
1933 (27) pointed out that not all varieties responded well to summer
planting.

Destruction of cane trash and diseased material after harvest
has been recommended (39, 84, 141), as a means of reducing red rot in-
fection. Abbott 1938 (10) doubted that such a measure would have enough
effect under Louisiana conditions. He indicated that it was impossible to
get rid of the trash completely, and that the small quantity that would
always remain would probably contain enough of the red rot fungus.
Further, seed cuttings and stubble rhizomes that became exposed during
spring cultivation operations serve as a source of inoculum for leaf
infections.

Considering disinfection as a measure of controlling red rot, hot
water treatment of seed pieces at 52 degrees C. for 20 minutes and its
relation to the development and control of red rot was studied in Louisiana. Forbes 1942 (170), and Dunckelman et al 1946 (128) stated that the red rot spores present on the cane at the time of the hot water treatment were destroyed. Forbes 1942 (170) tested the effect of the hot water treatment on ten varieties of sugarcane representing all degrees of resistance and susceptibility to the red rot fungus and observed that in every variety tested the red rot damage was apparently more extensive in the cane that had been given the hot water treatment. The results of another series of experiments indicated that the red rot fungus in well established lesions was not entirely destroyed by the hot water treatment.

Fungicide applications to growing cane plants as a means of controlling red rot disease were studied in Louisiana. Wahid, Steib, and Chilton 1951 (366) stated that latent infection occurring in the bud and leaf-scar tissues of sugarcane led to attempts to reduce this infection by dusting growing cane with fungicides. A 10 per cent dust containing either Zerlate, Fermate, or Parzate was used. The dust was applied to growing canes of the variety Co. 290 each week. Tissues from the leaf scars and buds were plated on oatmeal agar. In the undusted canes, the fungus was obtained from 63 per cent of the leaf scars and 66 per cent of the buds. In the canes dusted with either Parzate, Fermate, or Zerlate the fungus was obtained from 25 per cent of the leaf scars and 29 per cent of the buds.

Varieties of sugarcane resistant to red rot have been used in many countries as a method of controlling this disease. Abbott 1938 (10) stated that unless a variety possessed a satisfactory degree of resistance to red rot, other control measures would be ineffective in many years.
Breeding varieties resistant to this disease was attempted many years ago. As this subject is more or less related to breeding varieties resistant to mosaic disease of sugarcane, a short resume of the literature on these two subjects will be given simultaneously.

Considering developing mosaic resistant varieties, the work of the Louisiana Experiment Station is briefly reviewed. Then a review is given of the report of the major experiment stations that developed varieties used in the present studies.

In Louisiana, research on sugarcane problems may be said to have started with the establishment of the Sugar Experiment Station in 1885 with Dr. W. C. Stubbs in charge. Taggart 1927 (352) summarized the early work of this station in relation to sugarcane varieties. The Louisiana Experiment Station entered this work by importing true seeds from tropical countries and germinating them at Audubon Park. In 1906, R. E. Blouin and his assistant Weller, for the first time outside the tropics, succeeded in germinating cane seed. This work was continued until by 1917, 1842 Louisiana seedlings were tested. Agee (25) presented the results obtained in this work.

The quarantine regulations required that all the imported cane should go first to the Federal Horticultural Board. In 1917, the Louisiana Agricultural Experiment Station found that the seeds received through Washington could not be germinated. The disinfection which these seeds had received before being released in Washington was fatal to the germinating power, and hence the work was abandoned (352). However, this work was considered so important and the Station urged its continuation.

In 1918, the Director of the Louisiana Experiment Station, W. R. Dodson, requested the United States Department of Agriculture to undertake
to germinate cane seeds, and to import cuttings of promising seedlings and grow them at Washington D.C., until they could meet the quarantine regulations, and then send them to Louisiana. United States Senator, Edward J. Gay of Louisiana took an active interest in this matter (156), and with the participation of Director Dodson, it was arranged that the United States Department of Agriculture (Foreign Seed and Plant Introduction) should secure cane seed and germinate them in Washington for the Louisiana Experiment Station. In 1920, this service was consolidated with the seedling work of the office of Sugar Plant Investigations. The Louisiana Experiment Station contributes a nominal sum to the work in Florida, and in return gets a portion of all seedlings obtained (352).

In 1922, the Louisiana Station received from Canal Point about 2500 seedlings. These were planted at Audubon Park, and the most promising of them were transferred to Baton Rouge in 1923, and continued under test. Edgerton, Taggart and Tims 1927 (156) summarized the agreement between the Bureau of Plant Industry of the U.S. Department of Agriculture, and the Louisiana Experiment Stations. Further, under an agreement between the American Sugar Cane League, the Office of Sugarcane Investigations, and the Louisiana State University Sugar Experiment Stations a number of field tests were established. Edgerton, 1950 (148), summarized the work done by the Department of Botany, Bacteriology and Plant Pathology of Louisiana State University and the Louisiana Experiment Station from 1908 to 1950.

In 1948, the Department of Plant Pathology expanded its breeding work, and with a joint agreement with the American Sugar Cane League and the U.S. Department of Agriculture, the seeds obtained from the crosses made at the Canal Point Station were divided equally between the
Louisiana Station and U. S. Department of Agriculture Station at Houma, La. The Plant Pathology Department started in 1949, a project to produce sugarcane seeds at Grand Isle, La. The seeds obtained were grown at the Louisiana Station in Baton Rouge. In 1951, seedlings were obtained from the crosses made at Grand Isle which marked the first seedlings to be produced in Louisiana under controlled conditions (94), (95), (129).

The United States Department of Agriculture program of breeding mosaic resistant cane varieties started in Florida in 1919, as the opinion prevailed that tropical forms of sugarcane did not bloom in Louisiana. An Island off the southeast coast of Florida was selected as a quarantine-receiving station for planting the first imported varieties specifically designated for mosaic resistance breeding. In 1920, additional plantings were made at a site selected on Lake Okeechobee, near Canal Point, Florida (347). According to an anonymous paper (338) the Canal Point Station was established in 1921 by the U. S. Department of Agriculture.

Brandes 1931 (60) pointed out that the activities of the Office of the Sugar Plant Investigations began in 1919 following an intensive study of cane diseases in the Gulf States. The study included a comprehensive survey of practically all cane areas. Contacts were formed with experiment station and commercial planters in several countries, and by correspondence or personal collections a large number of cane varieties were accumulated. These varieties and their progenies were tested in cooperation with the Louisiana State University and the Louisiana Experiment Station. The objectives of this work were discussed by Brandes and Klapaak 1925 (63), Brandes and Sartoris 1936 (65), Sartoris 1934 (299), 1941 (300), Summers et al 1948 (347) and Anonymous (292).
Summers et al 1948 (347) pointed out that the work involved
(1) undertaking many plant explorations over a large part of the immense
and largely primitive area of the world where sugarcane was indigenous,
(2) setting up temporary, intermediate receiving stations in Australia,
Tahiti, Caroline Islands, and other localities, for growing the collected
material under observation, (3) devising special plant quarantine prac-
tices and quarantine facilities (182), (4) continually improving techniques
for large-scale inoculations, (5) establishing a world reference collection
of sugarcane varieties, and other details. Grassel 1951 (182) discussed
the importance of importing sugarcane varieties, and the breeding possi-
bilities of some of them, especially the cases involving interspecific
hybridization.

Briefly, the breeding work at the Canal Point Station involved
selfing of different varieties as well as crossing them. The crossing
program involved interspecific and intraspecific hybridization.
Sartoris 1941 (300) pointed out that many of the newly released varieties
were trispecies hybrids, since each had noble blood (S. officinarum),
some Chuanee blood (S. barberi), and some wild blood (S. spontaneum).

The methods used in the selection work on the hybrids produced
by the Canal Point Station were modified through the past thirty years.
This was in response to changes in the objectives of the breeding program,
and, in response to the development of new techniques in phytopathological
tests. Among the important papers concerning the selection work on the
Canal Point seedlings are those by Arceneaux and Stevens 1929 (29),
Arceneaux and Gibbens 1931, (28), Arceneaux et al 1932 (30) and 1933
(31), Rands et al 1935 (283), Abbott et al 1936 (21) and 1937 (15), Rands et
al 1940 (285), Abbott and Summers 1942 (16), Summers and Abbott 1943
(344) 1945 (345) and 1949 (346), Abbott and Summers 1946 (17),
Abbott et al 1948 (22), Abbott and Summers 1950 (18), 1951 (19) and
1951a (20) and Abbott 1951 (13).

Considering breeding disease resistant varieties of sugarcane,
the first contribution to this subject can be traced back to the work done
in Java during the last century in an effort to control the sereh disease.
Jeswiet 1927 (198) pointed out that in combating the spread of sereh
disease in Java, many importations of sugarcane varieties were made
in the hope that immune canes would be found. The varieties Loethers,
Fidji, Batjan, etc., were imported, but none of them were immune to
sereh. In 1890, Kobus was sent to the British Indies, and he brought back
to Java some canes, among which was the Chunnee variety (Saccharum
barberi). This proved to be immune to sereh but was not of sufficient
weight. However, it was crossed with other varieties and from this the
P.O.J. varieties of Chunnee blood were obtained. After this the Java
sugar industry left the method of selecting from the original canes and
went to the second possibility, that of selecting from crosses between
the original varieties (198), (273), (67).

Chunnee, Saccharum barberi Jesw., was crossed with varieties
of S. officinarum by Kobus in an effort to obtain varieties that were re-
sistant to sereh as well as having desirable characters as a high sugar
content, etc. Some seedlings of these crosses, as P.O.J. 213, P.O.J. 234,
P.O.J. 36, were extensively cultivated in certain countries, and were used
to a considerable extent as parents in crosses conducted by other breeding
stations.

Saccharum spontaneum was crossed with S. officinarum in 1887,
by Soltwedel. He tried the crossings by castrating the flowers of both
species, but he did not succeed in his attempt. Later on Moquette and Wakker crossed the wild cane Kassoer with *S. officinarum*, but all the seedlings obtained were discarded. Kassoer was considered by Jeswiet in 1915 to be a natural hybrid of *S. officinarum*, most probably Black Cheribon, and *S. spontaneum* of Java. Bremer in Java, conducted in 1917, cytological studies which also indicated that Kassoer is a hybrid of these two species of the genus *Saccharum*.

Kassoer cane was used by Willbrink in 1910 in crosses with the noble varieties striped Preanger, Black Cheribon and P. C. J. 100. The progenies were used by Jeswiet in crosses with varieties of *S. officinarum*. This process has been called nobilization. By crossing P. C. J. 2364 by E. K. 28 = (P. C. J. 100 by Kassoer) × E. K. 28, Jeswiet obtained the canes P. O. J. 2725 and P. O. J. 2878 which were resistant to mosaic (198), (199). These varieties were grown extensively in several countries and were used in numerous crosses in the breeding program of several experiment stations.

In India, the Coimbatore Station was the first station to use varieties of *S. spontaneum* of India in crosses with *S. officinarum*. During the first few years of breeding at Coimbatore the Javanese variety P. O. J. 213 was largely used as a female parent. In addition, important varieties like Co. 281, and Co. 290 were obtained. These varieties were hybrids of three species of sugarcane, *S. officinarum*, *S. spontaneum*, and *S. barberti* (65).

Considering sugarcane breeding work in other stations, Morris 1906 (247) discussed some of the early work in different stations. McIntosh 1932 (252), 1942 (253), 1944 (254) and Stevenson (323), (325),
discussed the breeding work in Barbados. The breeding work there for many years was concentrated on crossing and selfing varieties of *S. officinarum*, as well as crossing some of the seedlings obtained. However, the breeders in Barbados realized that in order to get disease resistant varieties, they had to cross these varieties of *S. officinarum* with varieties of *S. spontaneum*. The breeding work there at the present time involves mainly intercrossing the Barbados and other noble varieties with other species of Saccharum, followed by backcrossing to noble varieties (nobilization). Agee (26), and Mangelsdorf 1946 (231) reviewed the early work on sugarcane breeding in Hawaii. Rosenfeld 1935 (296) discussed the symbols used in the nomenclature of sugarcane seedlings. The bibliography of his paper comprised references on the breeding work in several countries. Dillewijn 1946 (123) and 1946a (124) reviewed the work on sugarcane breeding in many countries.

Studies on the inheritance of resistance to mosaic disease of sugarcane have lagged behind studies on other phases of this disease. Apparently there were difficulties in conducting genetic studies on sugarcane. Gowgill 1919 (180) and Venkatraman 1927 (364) and Cheesman (92) pointed out in this connection that wide variations occurred among seedlings of the same parentage. Venkatraman stated that these variations were so wide, that in his opinion, they rendered unlikely any definite laws in the matter of inheritance in sugarcane. He further indicated that the study of inheritance in sugarcane presented certain special difficulties resulting from the peculiarities of the plant. The more important of these were outlined by him as follow:

First, special care is needed to grow canes from seed. Second, the occurrence of great variations among the seedlings of the same parent,
even when precautions are taken against foreign pollination. This throws doubt on the purity of the varieties employed. Third, the uncertainty of the matter of parentage of most batches of even artificially raised seedlings resulted from the inability to employ either emasculation or bagging in cane breeding operations. The minuteness and delicacy of the floral structures in the sugarcane renders emasculating both difficult and laborious. Fourth, a thorough knowledge of the mode of inheritance and segregation of characters requires a continuous study of more than one generation from seed; this is often impossible in sugarcane, either on account of the non-flowering of the hybrids (183) or the infertility of the flowers, when the hybrids do flower. Available information related to studies on the inheritance of resistance to mosaic disease of sugarcane is reviewed here.

All the varieties of *Saccharum officinarum* tested in several countries were found to be susceptible to sugarcane mosaic virus (62), (63), (65), (124), (198), (258), (253).

Considering crosses between varieties of *S. officinarum*, Brandes and Klapkaak 1925 (63) stated that all self-fertilized seedlings of varieties of *S. officinarum* were found to be susceptible to mosaic. Further, that more than one thousand seedlings of B-109 studied were found to be susceptible to mosaic. They also stated that all seedlings obtained from crosses between varieties of *S. officinarum* were susceptible to this disease. Jeswiet 1927 (198) discussed some of the crosses made in Java and Barbados between varieties belonging to *S. officinarum* and stated that all the seedlings tested were susceptible to mosaic. Brandes and Sartoris 1936 (65) discussed the varieties produced by the Barbados station and stated that they were all susceptible to mosaic. These
varieties resulted from selfing or crosses between varieties of *S. officinarum*.

McMartin 1949 (258) in Natal (South Africa) stated that all varieties of *S. officinarum*, whether naturally occurring types, or seedlings resulting from self-fertilization of a noble cane or of a cross between two of these types, were susceptible to mosaic disease.

Inmiss 1944 (196) reported on the reaction of 93 Barbados seedlings to mosaic in Jamaica. Out of 21 seedlings that resulted from crosses between noble varieties, there was only one seedling that was resistant to mosaic. The other 20 seedlings were either susceptible or very susceptible to mosaic. This resistant seedling (B. 3439) came from a cross between Ba. 11569 x Q. 813. However, this seedling is not immune to mosaic. He pointed out that a few stools did contract the disease in Jamaica, and showed severe symptoms of mosaic. Further, he classified the resistant varieties as resistant and very resistant to mosaic and variety B. 3439 was considered as resistant only. Apparently it is resistant commercially, and according to his statement, a few stools occasionally contracted the disease.

The varieties of *S. barberi* were reported by Brando and Sartoris 1936 (65) to be susceptible to mosaic. They further stated that these varieties were not severely injured by the disease but were tolerant to it.

Chumee, a variety of *S. barberi*, is one of the ancestors of many of the varieties used in the present studies. Brandes and Klapaak 1925 (63), in the U.S.A. stated that Chumee and all its seedlings were susceptible to mosaic. Further, all the seedlings obtained from crosses of Chumee and varieties of *S. officinarum* were susceptible to mosaic. Jeswiet 1927 (198) stated that Chumee was crossed with varieties of *S. officinarum*, by Kobus, and the seedlings obtained were susceptible to mosaic. The *F*₁ or first generation hybrids were crossed with varieties
of *S. officinarum* and the seedlings obtained were also susceptible to mosaic.

Varieties of *Saccharum robustum* Brandes and Jeswiet ex Grassel, tested in the U.S.A. were reported by Brandes and Sartorie 1936 (65) and Summers et al 1948 (347), to be susceptible to mosaic. Rands et al 1935 (283) reported that variety G. 107 (synthetic Kassoer) from Java was used in a cross with *S. robustum* (N.G. 251) from New Guinea. Out of 130 seedlings tested there was but one mosaic susceptible seedling.

Brandes et al 1939 (66) and Summers et al 1948 (347) pointed out that some varieties of *S. spontaneum* tested appeared to be immune to mosaic and, as a group, the injury of those found to be susceptible was so slight as to be practically negligible. Brandes 1931 (60) stated that the cross between mosaic-immune varieties of *S. spontaneum* from Java, and varieties of *S. officinarum* had always resulted in apparently immune seedlings in the F1 generation, and a great preponderance of apparently mosaic-immune seedlings in the F2 generation. In the F2 progeny exposed to natural infection at Audubon Park, 9 out of 650 seedlings showed mosaic symptoms. In July, 1922. Further, a selection of about 70 seedlings planted near Houma, were observed in July 1924 to be free from mosaic with the exception of two individuals.

Rands, Abbott and Summers 1935 (283), reported the results obtained in testing the progeny reaction to mosaic of crosses between varieties of *S. officinarum* and *S. spontaneum*. In four crosses, variety D. 95, was crossed with *S. spontaneum* (Tobango selection), *S. spontaneum* (Pasoeroean), *S. spontaneum* (Imp. 470), and *S. spontaneum* (Imp. 238). The number of seedlings tested of the progeny of these crosses were 147, 148, 149 and 148 respectively. The percentages of seedlings showing
mosaic symptoms in these progenies were 6.8, 9.5, 0.0 and 1.4 per cent.
In the cross between Otchite and *S. spontaneum* (Imp. 238), seventy-nine
seedlings were tested and 22.8 per cent showed mosaic symptoms.
Rands et al 1935 (283) pointed out that these results showed that in
contrast with experience in Java with Cheribon × Glagah crosses, which
are said to have given all mosaic immune offspring, in Glagah from
Pasoeroean used in their studies, as well as the Spontaneums from other
sources, (with the exception of Imp. 470), the crosses with other varieties
yielded some mosaic susceptible seedlings. They further stated that
chromosome studies were being made to determine definitely whether or
not these varieties of *S. spontaneum* were hybrids. They concluded that
incomplete dominance of resistance to mosaic was indicated by their re-
results, and that it might be explained by the slight heterozygosity of the
different spontaneums as indicated by known differences in their chromo-
some number.

Kassoer cane is one of the ancestors of many of the varieties
used in these studies. It has been mentioned earlier that taxonomic and
cytological studies lead to the belief that this variety was a natural hybrid
between a variety of *S. officinarum*, possibly Black Cheribon, and *S.
spontaneum* of Java. Kassoer was crossed with varieties of *S. officinarum*,
and one of the hybrids obtained (P.O.J. 2364) was crossed with a variety of
*S. officinarum* by Jeswiet (198). Both mosaic resistant (i.e. P.O.J. 2725)
and susceptible (i.e. P.O.J. 2714) varieties were obtained from that cross.
On the other hand by crossing Chunnee, *S. barberi*, with noble varieties
and back-crossing the hybrids twice to varieties of *S. officinarum*, Jeswiet
obtained varieties that were severely attacked by mosaic.

Brandes and Klaphaak 1925 (63) stated that all selfed seedlings of
Kassoer studied were resistant to mosaic. When Kassoer was crossed back on *S. officinarum*, seedlings were sometimes produced that were susceptible to mosaic. Further, some of the seedlings containing 1/3 of *S. spontaneum* were not immune to mosaic.

Davis 1929 (116) reported that P.O.J. 2725 x S.C. 12-4 (Noble) seedlings were interplanted with mosaic infected plants of P.O.J. 36. In addition 22 susceptible seedlings of B.H. 10-12 were planted. The mosaic spread rapidly among the latter seedlings, until at maturity 41 per cent of all the stalks (500) were infected. In contrast to this, about the same number of the hybrids between P.O.J. 2725 and S.C. 12-4 had an infection of only one per cent at maturity. Six seedlings of P.O.J. 2725 x B.H. 10-12 were tested at Mayaguez for mosaic resistance. Only one of these developed mosaic. He concluded that immunity to mosaic was transmitted from P.O.J. 2725, when this cane was crossed with S.C. 12-4 or B.H. 10-12. Further, that mosaic resistance amounting to immunity might be secured from selection among seedlings containing only one-eighth part "Kassoer blood".

Davis 1932 (117) stated that the inbred seedlings of Kassoer, U. S. 429, U. S. 541, U. S. 710, and U. S. 785 have proved almost or quite immune to mosaic in Puerto Rico. From the cross between P.O.J. 2725 and U. S. 785, 198 seedlings were obtained. Only two of the seedlings (one per cent) showed mosaic symptoms in the field. When P.O.J. 2725 was crossed with U. S. 531, 62 seedlings were obtained, and 5.5 per cent of these seedlings had mosaic. He expressed the belief that P.O.J. 2725 could not be the source of the high resistance to mosaic in both progenies, as among 321 seedlings of P.O.J. 2725 x S.C. 12-4 there were 117 or one-third, that proved to be susceptible. On the other hand self-pollinated
seedlings of U. S. 541 and the progeny of crosses between U. S. 541 and S. C. 12-4 were also highly resistant to mosaic. In his studies the sugarcane seedlings were planted in the field and interplanted with P.O.J. 36 and Honey Dip sorghum (117) (118).

In Louisiana, Rands, Abbott and Summers 1935 (283) reported on the results of certain crosses and the reaction of the hybrids to mosaic. The crosses between resistant × resistant varieties were: P.O.J. 2725 × C. P. 1165, P.O.J. 2725 × C. P. 1161, and P.O.J. 2725 × U. S. 1694. The numbers of seedlings tested in the progeny of each of these crosses were 60, 159, and 43 respectively. The percentages of mosaic diseased plants in these progenies were 3.3, 25.2, and 0.3 respectively. The crosses between susceptible × resistant varieties were: Co. 281 × U. S. 1694, D. 74 × U. S. 1694, P.O.J. 2753 × L 1081, P.O.J. 2775 × L 1081, Co. 281 × C. P. 28-44, and Co. 281 × C. P. 30-23. The numbers of seedlings studied in the progeny of each of these crosses were 453, 78, 127, 146, 74, and 140 respectively. The percentages of plants showing mosaic symptoms in the progeny of each of these crosses were 51.4, 50.0, 40.2, 24.7, 66.2 and 78.6. However, Abbott, Summers and Rands in 1936 (21) reported that the percentage of mosaic diseased plants in the progeny of the last cross was 91.4. In the cross between the two susceptible varieties Co. 281 × C. P. 28-36, there were 89 seedlings obtained and 64.0 per cent of them showed mosaic symptoms (283).

Abbott et al, 1936 (21), (15) gave the results obtained in the crosses between resistant × resistant varieties. In one cross P.O.J. 2725 was crossed with C. P. 1161, and in another cross C. P. 1165 was crossed with C. P. 27-108. The number of seedlings studied in each case was 156 and 159 respectively. The percentages of plants showing mosaic
symptoms were 40.4 and 19.5 respectively.

Considering the mode of inheritance of resistance to mosaic disease of sugarcane, Brandes 1931 (60) pointed out that the seedlings obtained from crosses between S. officinarum and S. spontaneum were immune to mosaic, as well as a very high percentage of the F2 generation. He further stated that it would seem reasonable to interpret this to mean that susceptibility to mosaic was a recessive character. However, he indicated that there were records of mosaic in variety Co. 205 at Pusa, India by McRea and at Canal Point by Bourne. This variety had been reported to be an F1 hybrid resulting from a cross of S. spontaneum (India) with S. officinarum. Brandes further stated that it was not known whether or not the variety of S. spontaneum used in that cross was immune to mosaic, and that unless the Spontaneum parent of Co. 205 was itself a hybrid it was unsafe to conclude that immunity to mosaic was a dominant character. Dutt et al 1936 (130) pointed out that Co. 205 showed mosaic symptoms at Coimbatore, but was free from mosaic in another part of India. Randis et al 1935 (283) and 1935a (284) expressed the belief that resistance to mosaic showed incomplete dominance.

Brandes 1931 (60) stated that resistance to mosaic in sugarcane or lack of it was not to be assigned to a single pair of allelomorphic factors. He noted that within the progenies of certain crosses there was a graduated range of response to mosaic, extending from extreme susceptibility to complete immunity, which he believed to be attributed to multiple factors.

Li, Cheng and Leung 1951 (221) expressed the belief that resistance to mosaic from Galagah was controlled by a set of genes scattered
on different chromosomes. Further, the degree of resistance, as expressed by the varieties derived from the classical cross between P.O.J. 2364 and E. K. 28, was both quantitative, i.e. the more Galagah chromosomes, the higher the resistance, and qualitative, i.e. having more resistant genes on certain chromosomes of Galagah. It has to be pointed out that Li et al did not report any work on mosaic of their own in their paper. This point will be covered in detail later in the discussion.

Chromosome behavior during meiosis is one of the items related to the interpretation of the data obtained in the present studies. No attempt is made here to give a complete review of the papers published on cytology of sugarcane; only the important papers related to the present studies are reviewed.

Saccharum officinarum is generally characterized by the diploid chromosome number 80 (67), (68), (70), (71), (72), (74). Many varieties which showed a great resemblance to S. officinarum, and usually are considered to belong to this species, have been shown by Jeswiet’s taxonomical studies to differ in definite points from noble canes. Bremer 1929 (70) and 1931 (72) showed that such varieties possessed deviating chromosome numbers. Among these varieties is Loethers.

Bremer 1924 (68) stated that the chromosome number of Loethers was higher by 19 than that of S. officinarum-forms. He was of the opinion that this variety was probably a hybrid between S. officinarum and an unknown cane with another chromosome number.

Variety P.O.J. 100 had been obtained by Wakker in 1873, from an inflorescence of Bandjarmassin hitam pollinated by the wind. Taxonomic studies by Jeswiet suggested that Loethers-cane was the male parent.
Cytological studies by Bremer 1924 (68) showed that P.O.J. 100 had 89 chromosomes. This number corresponded exactly with the sum of the haploid chromosome numbers of Bandjarmasin hitam (n = 40) and Loethers (n = 49). Variety P.O.J. 100 was the female parent of the varieties P.O.J. 2364 and E. K. 28. These varieties were used in the early breeding program in Java. A discussion of these varieties will be given later.

Bremer 1923 (67) and 1924 (68) described the reduction division in the pollen mother cells of some varieties of *S. officinarum*. In Black Cheribon it was shown that during metaphase there were both bivalent and univalent chromosomes. During anaphase the chromosomes derived from the "gemini" proceeded regularly toward the poles. The univalent chromosomes lagged behind and split lengthwise, and subsequently moved toward the poles. The result was that nuclei of the pollen grains probably obtained more than 40 chromosomes. This irregularity of chromosome behavior has been observed in other varieties of *S. officinarum* by Bremer.

*S. spontaneum* was grouped by several investigators into different forms. Bremer 1923 (67) observed 56 as the haploid number of chromosomes in Galagah Java. Bremer 1925 (69) observed that Galagah Tobango had 40 as the haploid number of chromosomes. This cane differed only to a slight extent in external characteristics from Galagah Java.

Dutt and Rao 1933 (131), observed that the Coimbatore form of *S. spontaneum* had 32 chromosomes as the haploid number. Singh 1934 (308) reported 32, 27 and 39 bivalent chromosomes in the Godavari, Dehra and Decca forms of *S. spontaneum* respectively. Janaki Ammal 1936 (197) studied different forms of *S. spontaneum* and reportedly had 48, 56, 64
and 80 as the diploid chromosome number. This suggested to her that the different types of *S. spontaneum* represent a polyploid series with a basic chromosome number of eight. She also observed that during meiosis the chromosomes were always associated as bivalent, except in Reilagaddi, in which a single tetravalent was observed.

Considering studies on interspecific hybrids within the genus *Saccharum*, Bremer in Java conducted an elaborate study on this subject. Bremer 1923 (67) in studying the pollen mother cells of Kassoer, observed that there were 61 bivalents, 12 univalents. He pointed out that Kassoer had 134 as the diploid chromosome number and approximately 67 as the haploid chromosome number. In G. 106 and G. 107, varieties that resulted from a cross between striped Preanger (a noble cane) and Galagah alastroeno (*S. spontaneum*), he pointed out that G. 106 had 135 as the diploid chromosome number. In G. 107 there were 64 paired chromosomes and 7 univalents. This pointed to a haploid chromosome number as about 68. These results showed that when *S. officinarum* (*n* = 40) was crossed with *S. spontaneum* Galagah (*n* = 56) the somatic chromosome number in the resulting F₁ progeny was 136 instead of the expected 96. Bremer was of the opinion that during fertilization the *S. officinarum* chromosomes had undergone a longitudinal split, thus doubling their number, while those of *S. spontaneum* remained unsplit. This resulted in the F₁ plants having 136 chromosomes, 80 of which came from the *S. officinarum* and 56 from *S. spontaneum*. He expressed the belief that the fertilizing sperm nucleus stimulated the female nuclear substance to undergo a longitudinal split of its chromosomes.

When the F₁ plants were selfed, the seedlings of the F₂ generation had 136 chromosomes or less in the diploid phase. However, when the F₁
plants were backcrossed to the *S. officinarum* the resulting hybrids had 148 as the diploid chromosome number instead of the 108 normally expected. This suggested again that the chromosome complement of the Officinarum parent was doubled. This backcrossing process is known as second nobilization. In the subsequent backcross the somatic number of chromosomes in the hybrids varied between 106 and 120. The average chromosome number in these plants was 114. This suggested that the chromosome number of the Officinarum parent did not double. In the case of continued nobilization this doubling did not occur any longer. However, when backcrossing the third nobilization plants (2n = 114) with *S. spontaneum*, chromosome doubling took place again, so that forms with 160 to 170 somatic chromosomes were obtained (70), (71).

Chromosome doubling occurred also when *S. officinarum* was crossed with *S. spontaneum* Coimbatore (230) (131). In the variety Co. 205, which resulted from a cross between Vellai (*S. officinarum*) and *S. spontaneum* Coimbatore (365), the somatic chromosome number was observed to be 112. This number equaled the sum of the haploid number of the spontaneum parent (n = 32) plus twice the haploid number of the officinarum parent (131).

It is noticed that the hybrids resulting from any certain cross, in the nobilization process, did not have the same chromosome number. For example, varieties P.O.J. 2725 and P.O.J. 2878 which resulted from one cross had the somatic chromosome numbers 106-107 and 119-120 respectively. (70), (221), (222). Loh and Tseng 1951 (226) in discussing sugarcane nobilization methods expressed the belief that the ability of a parent to transmit its visible characters to its offspring resulted from its capacity to maintain its chromosome balance. This means that such
a parent had the ability to make pairs of homologous chromosomes in its own way, irrespective of the other parent crossed with it.

Chunnee, a variety of *S. barberi*, was reported by Bremer 1923 (67) to have 90 or 91 as the diploid chromosome number. In metaphase of the meiotic division he observed the presence of univalent chromosomes in addition to the bivalent ones. Bremer 1931a (73) and 1932 (4) observed that the different varieties of *S. barberi* had from 82 to 123 as the diploid chromosome number. Varieties P.O.J. 118 and P.O.J. 213, that resulted from a cross between *S. officinarum* and Chunnee, were observed to have 62-64 as the haploid chromosome number and 124-128 as the diploid one.

When *Saccharum officinarum* (2n = 80) was crossed with *S. robustum* (2n = 84), the F₁ plants had a chromosome number exceeding the sum of the parental haploid chromosome number. Brandes and Sartoris 1936 (65) stated that the seedlings from the cross D-74 (2n = 80) × *S. robustum* (2n = 84) had 100 somatic chromosomes instead of the expected 82 chromosomes. It is noted that the F₁ plants had an increase of about 18 chromosomes. They expressed the belief that this could have resulted from doubling of some of the chromosomes of *S. officinarum*.

Many cases have been reported in the literature in which the same phenotypic character might be controlled by distinct gene pairs. Walker 1951 (368) stated that the best example of this phenomenon was illustrated by the work of Briggs and associates with powdery mildew of barley, (*Erysiphe graminis hordei* (DC) Marshall). The results obtained and conclusions reached by Briggs and his coworkers are related to the conclusion reached and discussion of the present studies on sugarcane. Therefore, a review of Briggs' studies on barley mildew is given here.
In 1933, extensive studies were started in California, on the inheritance of resistance in barley to powdery mildew caused by *Erysiphe graminis hordei*. The studies reviewed here are dealing with those made on one race of this fungus, namely race 3. Briggs and Barry 1937 (76) reported on the crosses between Hanna and Atlas, the former being a variety resistant to mildew and the latter a susceptible one. Studies on the $F_2$ and $F_3$ generations suggested that the resistance of Hanna in the cross Hanna X Atlas was incompletely dominant and conditioned by one pair of genes.

Goldfoil, a resistant variety of barley, was also resistant to race 3 of the mildew fungus. In the crosses between Goldfoil X Atlas, Briggs and Barry 1937 (76) obtained in $F_2$ a ratio of 3 resistant:01 susceptible. However, he considered the plants giving types 1 and 2 of infection as heterozygous and the data suggested a 1:2:1 ratio. This suggested that resistance in Goldfoil was conditioned mainly by one pair of genes. Crosses were made between the two resistant varieties Goldfoil and Hanna, and plants of the $F_2$ generation showed a ratio of 15 (resistant):1 susceptible. This suggested that the factor for resistance of Goldfoil was different from the one of Hanna and he designated the factor "g" for that in Goldfoil and "H" for that in Hanna.

Briggs 1938 (75) reported on studies with the three mildew-resistant varieties, - Arlington Awnless (*Hordeum intermedium haxtoni tosum*), Chinerme and Nigrate (*H.i. martoni ingritonsum*). These varieties were crossed with the mildew-susceptible Atlas. The $F_2$ generation showed a segregation of 15:1 ratio and suggested that each of the three resistant varieties differed from Atlas in two independent major factors and that susceptibility was a recessive character. By crossing
these three resistant varieties with each other, no susceptible plants
were obtained in the $F_2$ generations, indicating that these three varieties
had at least one pair of factors for resistance in common.

By crossing each of the same three resistant varieties, Arlington Awnless, Chinerme and Migrate with Hanna and Goldfoil
(both were also resistant to mildew), some susceptible plants were
obtained in the $F_2$ generation with a ratio of 63 (resistant):1 susceptible.
This indicated that in each cross, one parent contributed with two factors
and the other with one factor for resistance. From this data, Briggs
1938 (75) stated that the factor for resistance of Hanna and that of Goldfoil
were not carried by the former three varieties.

Briggs and Stanford 1938 (77) discussed the inheritance of re-
stance to race 3 of the mildew fungus in three additional resistant
varieties: Algerian (C.L 1179), an unnamed variety S.P.L 4592, and
Kwan (C.L 1016). Algerian was completely resistant to race 3 (Type O
of infection). On the other hand, S.P.L 4592 showed type 1 of infection
in the greenhouse, while Kwan gave type 2 of infection in the greenhouse.
Types 1 and 2 of infection were closer to type "O" than to type "4",
and these varieties were considered resistant. Crosses between Atlas X=
Algerian, S. P. L 4592 X Atlas, and S. P. L 4592 X Algerian showed that
both of the last two varieties differed from Atlas in one major factor for
resistance to mildew, that the factor of Algerian was the same one in
S. P. L 4592, and that resistance was completely dominant. Results
obtained from other crosses showed that this factor in Algerian differed
from that of Hanna, of Goldfoil and of the two factors of Arlington Awnless
and Chinerme. Briggs and Stanford (77) designated the factor for resis-
tance of Algerian of S.P.L 4592 as the Algerian factor ($ML_aML_a$).
Croses of Kwan x Atlas showed that Kwan differed from Atlas in one factor for resistance. Further studies indicated that the factor for resistance to mildew of the Kwan variety was different from those in the other seven resistant varieties mentioned before. Further, the factor for resistance of Kwan and that of Algerian were linked with the crossover value of 9.81 (77).

Another factor for resistance to mildew was identified by Stanford and Briggs 1940 (315) in the Psakon variety. Results obtained from crosses between Atlas and Psakon suggested that Psakon had one dominant factor for resistance. The results from crosses between Psakon and the other resistant varieties mentioned before which each had a single factor for resistance, indicated that the factor in Psakon differed from them. In crosses between Arlington x Psakon, and Nigrate x Psakon, no susceptible plants were obtained in the F2 generation. This suggested that the factor of Psakon was identical to one of the two found in Arlington and Nigrate, and it was called MLp (315).

The Duplex variety was shown to have three independent factor pairs for resistance to mildew, and one of them was recessive. Results obtained from several crosses showed that Duplex had the factors present in Goldfoil, Kwan and Algerian. Thus, Duplex factorial composition was designated as follows: MLbMLb, MLpMLp, MLdMLd.

Stanford and Briggs 1940 (77) concluded that studies on varieties of barley resistant to Erysiphe graminis hordei physiologic race 3, showed that there were seven different genetic factors for mildew resistance - six dominant and one recessive. The number of factors in a single variety varied from one to three factor pairs. Of the seven factors identified, two were definitely linked. The other five
factors appeared to be independent, although the Duplex factor might possibly be linked with either the Hanna or the Psakon factors.

Favret 1949 (65) reported on further studies on the inheritance of resistance to physiologic race 3 of *Erysiphe graminis hordae*. The results obtained showed that both the Monte Criste and Engeldow India varieties carried a dominant gene for immunity, which has been called MLm and was linked with one of the Nigrate factors determined by Briggs and Stanford (77). The resistance of West China barley was governed by a single recessive factor, which has been called MLw. Favret further stated that it was probable that the resistance of Gopal barley was governed by two major genes, linked with a crossover value of about 15-20 per cent. He concluded that with the two factors above mentioned, nine genes were already known to be involved in the resistance to mildew race 3 in barley, the largest number of loci related to resistance to a plant disease.
MATERIALS AND METHODS

The studies presented here were made on sugarcane seedlings grown from true seeds which came from crosses between different varieties of sugarcane. These crosses were made at the Experiment Station of the United States Department of Agriculture at Canal Point, Florida, in the period between the months of November and January, for three successive years. Those made in the period between November 1948 and January 1949 were designated in these studies as the "1948 Crosses". Those made during the similar period of the following years were called the 1949 and 1950 crosses consecutively.

The seeds were sent to Baton Rouge and were planted as soon as they were received, in flats containing a soil mixture consisting of 75 per cent organic soil, 20 per cent loam (bottom soil), and 5 per cent sand. This mixture previously had been subjected to steam for 2 hours under 18 pounds pressure. The flats were placed on benches in the greenhouse. When the seedlings were about 4 inches tall they were transferred to clay pots 2-1/2 to 3 inches in diameter. These pots contained a mixture of 75 per cent bottom soil, 20 per cent organic matter, 5 per cent sand, and the mixture had been steamed as above. One seedling was planted in each pot.

During a period of 7 to 17 days after potting the seedlings were inoculated with sugarcane mosaic virus. The plants were watered with a solution of an ammonium sulphate fertilizer three days before inoculation and at about weekly intervals thereafter.

The inoculum used was obtained from plants (1-2 feet high) of variety Co. 290 showing mosaic symptoms. Portions of the youngest
parts of the stem, ranging in size between 6 to 12 inches, as well as the top young leaves were cut and ground in a meat chopper. The juices obtained were strained through a double layer of cheese cloth. The juice was prepared about every hour. However, sometimes it was noticed that the juice would change in color and produce an unusual odor. In such cases the juice was discarded at once and fresh juice was prepared. In this regard, we quote Dr. E. M. Summer's statement, during the discussion that followed the presentation of a paper by Stevenson (322) at a meeting of the sixth Congress of the International Society of Sugarcane Technologists: "some batches of juice give almost no infection, indicating that something occasionally happens to inactivate the juice".

The abrasive added to the juice was 220-mesh carborandum. However, in inoculating seedlings of the 1948 crosses, occasionally 30-mesh sand was used. No effort was made to proportion the amounts of inoculum and abrasives. The inoculum mixed with the carborandum was picked between the thumb and index finger and rubbed on the spindle.

The plants were kept about four weeks in the greenhouse. Then they were moved outside the greenhouse to allow space for more seedlings. For example, in the spring of 1950, approximately 55,893 seedlings were inoculated. The plants were examined frequently and those showing mosaic symptoms were discarded, and those that did not show mosaic symptoms were planted in the field during the months of April and May. The plants were later examined in the field for mosaic symptoms. It was found that some of these seedlings that showed no mosaic symptoms in the greenhouse, began to show the symptoms of the disease in the field. Such plants were counted, and the number of diseased plants in the greenhouse and the number of plants showing the mosaic symptoms in the field,
for each cross was recorded. The seedlings of the 1948 crosses were examined in the field during the last two weeks of September and the first week of October 1949. The seedlings of the 1949 and 1950 crosses were examined during July 1950 and 1951 respectively.

Another phase of these studies involved the comparison of two inoculation techniques. Progenies from each of 18 crosses were separated into two groups. The pot inoculation technique described previously was used in one group, while in the other group, another technique called here "the flat inoculation technique" was used as follows.

The true seeds were planted as described previously for the pot inoculation technique. When the seedlings were about 4 to 5 inches tall, they were sprayed with juice obtained from mosaic-diseased plants of variety Co. 290, using a household sprayer. The juice was prepared in the same manner as previously described for use in the pot inoculation technique. After the seedlings were sprayed, parts of the top leaves, and the top of parts of the unrolled leaves were clipped immediately, using a pair of shears. The inoculum that remained in the gun after the first spray was then used for spraying the seedlings again after the clipping. About two weeks after being inoculated with the sugarcane mosaic virus the seedlings were transplanted to 3-inch pots, one seedling per pot. Thereafter, they received the same treatments as those described for the seedlings inoculated with the "pot inoculation technique". The seedlings showing mosaic symptoms in the greenhouse were discarded and their number recorded. Those that did not show mosaic symptoms were transplanted to the field in April 1950, and were examined for mosaic symptoms in July of the same year.
Studies on the recovery from mosaic symptoms were made on the progeny of the 1950 crosses. On April 14, 1951, the seedlings that showed mosaic symptoms in the greenhouse were set in the field two feet apart in rows. These rows were spaced at 6 foot intervals. During the period between July 4 and July 14, 1951, the seedlings were examined for recovery from mosaic and the number of recovered seedlings in the progeny of each cross were recorded. Drought occurred during the summer, causing the plants to be relatively small, thereby making it easy to examine them for mosaic recovery. During the period between August 26 and August 29, 1951, the plants were reexamined for recovery, and the numbers of recovered seedlings were again recorded.

A stool was considered to have recovered when every leaf in each stalk was free from mosaic symptoms. The appearance of mosaic symptoms on a single leaf was sufficient to classify the plant as having mosaic. It was also observed that in some cases some stalks of a single stool showed no mosaic symptoms, while other stalks of the same stool showed the disease symptoms. Such stools were called "partially recovered", and their number was recorded during the August examination.

Studies on the inheritance of resistance to the red rot disease of sugarcane, caused by Physalospora tucumanensis Speg, were conducted on the progeny of certain crosses. The plants used in these studies were the first stubble of seedlings of the 1943 and 1949 crosses, which had been previously inoculated with sugarcane mosaic virus in the greenhouse and did not show mosaic symptoms at the time of their transplanting to the field. Rand, Abbott, and Summers 1935 (283), conducted studies on 705 seedlings and they concluded that resistance or susceptibility to either mosaic or red rot was independent of the reaction of the same seedlings
to the other disease.

The seedlings tested for red rot resistance were the progenies of 14 crosses involving resistant X resistant parents, resistant X susceptible parents and susceptible X susceptible parents. The method of inoculation as well as the source of the fungus used are described in the following paragraphs.

The red rot fungus was obtained from diseased stalks of cane variety Co. 290, grown in the field at the Louisiana Agricultural Experiment Station, Baton Rouge, La. Both white and dark cultures of the fungus were isolated and five of them were tested for their pathogenicity. In each case, a spore suspension of the culture tested was used to inoculate four stalks of variety Co. 290. The stalks were wrapped in a moist cloth, then kept in the laboratory at a temperature of 70\(^\circ\) degrees F., then split and examined. After examining the inoculated stalks, the most pathogenic culture, which was of the light culture type, was selected and used as a source of inoculum in the tests conducted on the progeny of the 1948 and 1949 crosses.

Oatmeal agar was used as the culture medium for *Physalospora tucumanensis* Speg. It was made by using 65 grams of oatmeal and 17 grams of bacto-agar per liter of water. The oatmeal was placed in a filter flask containing 500 cc. of water, and the flask was placed on a water bath for 4 to 7 minutes. The material was then strained through a cheese cloth. The agar was mixed in 500 cc. of water and heated on a low gas flame until the agar was dissolved. The oatmeal extract and water agar were thoroughly mixed. The medium was sterilized by autoclaving for 20 minutes at 17 pounds pressure. Then it was poured in petri dishes (8.9 cm. in diameter). One drop of 50 per cent lactic acid was added to
each plate to acidify the agar and reduce the bacterial contamination. Small pieces of the mycelium of the red rot fungus were then transferred to the medium.

The fungus cultures were kept at a temperature between 27 and 30 degrees Centigrades for 3 or 4 days, then were put under blue light for 2 or 3 days at a temperature of 710 degrees F. It was previously observed that there was a rapid production of conidia when the fungus was grown under blue light. The source of light used was daylight fluorescent tubes covered with blue cellophane paper.

Considering the technique of inoculation, spore suspensions from the light culture of the fungus were prepared. The sugarcane stalks were prepared for the inoculation by removing the sheaths and tops and then cutting into 4 to 5 foot lengths. Three stalks from each stock were cut, tied together with a hay baling wire and tagged. Then a hole was punched in the middle of each stalk. A special punch similar to a cork borer with an opening 2.5 mm. was used. Using an eye dropper, a few drops of the spore suspension were introduced into these holes. The inoculated stalks were then buried in the field and covered with 5-7 inches of soil. Later, the stalks were dug up, washed with water, split with a cane knife and examined. Checks were made by inoculating 40 stalks of variety Co. 290, and 20 stalks each of C. P. 34-120, C. P. 36-13 and C. P. 36-105. Five stalks of Co. 290 that were free from borer infection were not inoculated and used as another check. Later, the stalks were dug up, washed with water, split lengthwise and examined. The reactions of the seedlings to red rot were placed in four classes: 1) resistant, 2) moderately resistant, 3) moderately susceptible and 4) susceptible.
RESULTS

The sugarcane seedlings were inoculated with sugarcane mosaic virus, and any mosaic symptoms appearing on them were observed and recorded. The period of incubation varied from four days and in a few instances to a period of seven weeks, and perhaps longer. Some of the seedlings were apparently healthy when they were transplanted to the field, but later they showed mosaic symptoms. In such cases, the mosaic disease in the field could have occurred by natural infection or as a result of the inoculation done in the greenhouse; assuming that the latter case occurred in some instances, the incubation period lasted probably more than seven weeks. The incubation period differed among the plants within the same cross. In addition, the progeny of some crosses, especially some of those that had variety F. 36-819 as a female parent, had a very short period of incubation, and these progenies showed somewhat more severe symptoms of the disease.

Different patterns of mosaic symptoms were observed on the seedlings in the greenhouse as well as in the field, although there was only one strain of the sugarcane mosaic virus used in inoculating the seedlings. The diversity of the genetic constitution of the seedlings appeared to be responsible for the occurrence of these different mosaic patterns.

The results of the studies of inheritance of resistance in sugarcane to sugarcane mosaic virus are recorded in tables 1 to 33. The number of plants showing mosaic symptoms in the greenhouse was recorded and such plants were then discarded. The percentage of these plants per cross was computed and appears in the tables under "percentage of mosaic discards". The plants that were apparently healthy in the
greenhouse were transplanted to the field. Later, mosaic symptoms were observed on some of these plants, and the number of plants showing the disease symptom per cross was recorded. The total number of mosaic-diseased plants in both the greenhouse and the field per cross was obtained and the percentage per cross was computed and appears in the following tables under "total percentage of mosaic".

Each of the tables 1 to 14 shows the reaction to sugarcane mosaic virus of the progeny of one female when used in crosses with several males.
Table 1. Mosaic resistance data: Analysis relative to female parent C. P. 27-139.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-114</td>
<td>C.P. 36-105</td>
<td>low %</td>
<td>197</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>48-115</td>
<td>C.P. 44-156</td>
<td>none</td>
<td>169</td>
<td>0</td>
<td>1.78</td>
</tr>
<tr>
<td>48-152</td>
<td>C.P. 43-74</td>
<td>80%</td>
<td>847</td>
<td>5.9</td>
<td>8.38</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: none
Table 2. Mosaic resistance data: Analysis relative to female parent C. P. 29-103.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-36</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>506</td>
<td>41.89</td>
<td>43.68</td>
</tr>
<tr>
<td>48-46</td>
<td>C.P. 38-41</td>
<td>none</td>
<td>352</td>
<td>66.29</td>
<td>67.61</td>
</tr>
<tr>
<td>48-48</td>
<td>C.P. 36-105</td>
<td>low %</td>
<td>306</td>
<td>58.17</td>
<td>60.13</td>
</tr>
<tr>
<td>48-49</td>
<td>C.P. 33-372</td>
<td>none</td>
<td>514</td>
<td>63.22</td>
<td>65.95</td>
</tr>
<tr>
<td>48-61</td>
<td>C.P. 33-224</td>
<td>none</td>
<td>402</td>
<td>22.89</td>
<td>25.12</td>
</tr>
<tr>
<td>48-70</td>
<td>C.P. 27-108</td>
<td>none</td>
<td>714</td>
<td>24.64</td>
<td>26.33</td>
</tr>
<tr>
<td>48-72</td>
<td>F. 36-273</td>
<td>none</td>
<td>654</td>
<td>16.74</td>
<td>18.13</td>
</tr>
<tr>
<td>48-91</td>
<td>C.P. 44-156</td>
<td>none</td>
<td>282</td>
<td>1.77</td>
<td>7.8</td>
</tr>
<tr>
<td>48-98</td>
<td>C.P. 43-74</td>
<td>50%</td>
<td>417</td>
<td>8.15</td>
<td>22.06</td>
</tr>
<tr>
<td>48-104</td>
<td>C.P. 42-10</td>
<td>trace</td>
<td>575</td>
<td>2.26</td>
<td>9.04</td>
</tr>
<tr>
<td>48-112</td>
<td>U.S. 1694</td>
<td>none</td>
<td>105</td>
<td>0</td>
<td>1.9</td>
</tr>
<tr>
<td>48-145</td>
<td>C.P. 27-48</td>
<td>none</td>
<td>120</td>
<td>40.83</td>
<td>41.67</td>
</tr>
<tr>
<td>48-146</td>
<td>C.P. 33-229</td>
<td>none</td>
<td>119</td>
<td>20.17</td>
<td>21.01</td>
</tr>
<tr>
<td>49-252</td>
<td>C.P. 48-126</td>
<td>none</td>
<td>601</td>
<td>83.03</td>
<td>83.86</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: trace
Table 3. Mosaic resistance data: Analysis relative to female parent C. P. 29-116.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-5</td>
<td>C.P. 30-24</td>
<td>none</td>
<td>402</td>
<td>2.74</td>
<td>2.74</td>
</tr>
<tr>
<td>48-9</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>362</td>
<td>13.11</td>
<td>16.94</td>
</tr>
<tr>
<td>48-25</td>
<td>C.P. 36-105</td>
<td>low %</td>
<td>236</td>
<td>28.39</td>
<td>30.01</td>
</tr>
<tr>
<td>58-63</td>
<td>C.P. 36-138</td>
<td>80%</td>
<td>192</td>
<td>20.31</td>
<td>23.96</td>
</tr>
<tr>
<td>48-68</td>
<td>C.P. 36-156</td>
<td>?</td>
<td>327</td>
<td>33.33</td>
<td>36.39</td>
</tr>
<tr>
<td>48-100</td>
<td>C.P. 48-106</td>
<td>20%</td>
<td>301</td>
<td>1.99</td>
<td>4.98</td>
</tr>
<tr>
<td>48-101</td>
<td>C.P. 33-224</td>
<td>none</td>
<td>383</td>
<td>0.0</td>
<td>0.78</td>
</tr>
<tr>
<td>48-84</td>
<td>C.P. 44-156</td>
<td>none</td>
<td>236</td>
<td>0.0</td>
<td>1.27</td>
</tr>
<tr>
<td>49-61</td>
<td>Co. 356</td>
<td>none</td>
<td>569</td>
<td>2.99</td>
<td>5.80</td>
</tr>
<tr>
<td>49-62</td>
<td>C.P. 43-74</td>
<td>80%</td>
<td>197</td>
<td>3.55</td>
<td>6.09</td>
</tr>
<tr>
<td>49-19</td>
<td>C.P. 46-199</td>
<td>none</td>
<td>519</td>
<td>0.8</td>
<td>1.9</td>
</tr>
<tr>
<td>49-35</td>
<td>C.P. 46-73</td>
<td>none</td>
<td>396</td>
<td>0.0</td>
<td>4.3</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: trace
### Table 4. Mosaic resistance data: Analysis relative to female parent C. P. 33-229.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-167</td>
<td>C.P. 33-224</td>
<td>none</td>
<td>282</td>
<td>0</td>
<td>1.77</td>
</tr>
<tr>
<td>48-193</td>
<td>Open Poll.</td>
<td>?</td>
<td>501</td>
<td>0.98</td>
<td>2.35</td>
</tr>
<tr>
<td>49-55</td>
<td>C.P. 43-74</td>
<td>80%</td>
<td>193</td>
<td>16.58</td>
<td>17.10</td>
</tr>
<tr>
<td>49-85</td>
<td>C.P. 36-211</td>
<td>none</td>
<td>1024</td>
<td>4.69</td>
<td>5.08</td>
</tr>
<tr>
<td>49-103</td>
<td>C.P. 33-372</td>
<td>free</td>
<td>446</td>
<td>13.45</td>
<td>14.57</td>
</tr>
<tr>
<td>49-60</td>
<td>Co. 356</td>
<td>none</td>
<td>733</td>
<td>0.54</td>
<td>0.54</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: none
<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-3</td>
<td>C.P. 30-24</td>
<td>none</td>
<td>1456</td>
<td>1.92</td>
<td>2.68</td>
</tr>
<tr>
<td>48-23</td>
<td>C.P. 36-105</td>
<td>low %</td>
<td>163</td>
<td>16.56</td>
<td>23.31</td>
</tr>
<tr>
<td>48-39</td>
<td>C.P. 38-12</td>
<td>none</td>
<td>323</td>
<td>8.05</td>
<td>8.36</td>
</tr>
<tr>
<td>48-38</td>
<td>C.P. 36-156</td>
<td>?</td>
<td>566</td>
<td>13.6</td>
<td>15.55</td>
</tr>
<tr>
<td>48-40</td>
<td>C.P. 36-138</td>
<td>90%</td>
<td>715</td>
<td>19.44</td>
<td>23.5</td>
</tr>
<tr>
<td>48-41</td>
<td>C.P. 33-372</td>
<td>none</td>
<td>269</td>
<td>0.37</td>
<td>1.49</td>
</tr>
<tr>
<td>48-42</td>
<td>C.P. 33-224</td>
<td>none</td>
<td>488</td>
<td>15.16</td>
<td>16.80</td>
</tr>
<tr>
<td>48-43</td>
<td>C.P. 38-41</td>
<td>none</td>
<td>546</td>
<td>3.30</td>
<td>5.31</td>
</tr>
<tr>
<td>48-59</td>
<td>F. 36-273</td>
<td>none</td>
<td>170</td>
<td>1.18</td>
<td>2.35</td>
</tr>
<tr>
<td>48-89</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>1188</td>
<td>0.51</td>
<td>0.89</td>
</tr>
<tr>
<td>48-90</td>
<td>(P.C.J. 2725 X C.P. 36-105)</td>
<td>?</td>
<td>204</td>
<td>0.0</td>
<td>1.96</td>
</tr>
<tr>
<td>48-105</td>
<td>C.P. 42-10</td>
<td>trace</td>
<td>157</td>
<td>0.0</td>
<td>1.27</td>
</tr>
<tr>
<td>48-106</td>
<td>C.P. 43-74</td>
<td>80%</td>
<td>392</td>
<td>0.0</td>
<td>5.1</td>
</tr>
<tr>
<td>48-108</td>
<td>C.P. 33-229</td>
<td>none</td>
<td>125</td>
<td>0.0</td>
<td>0.8</td>
</tr>
<tr>
<td>48-118</td>
<td>C.P. 27-48</td>
<td>none</td>
<td>151</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>48-124</td>
<td>C.P. 43-64</td>
<td>50%</td>
<td>134</td>
<td>0.0</td>
<td>0.75</td>
</tr>
<tr>
<td>49-36</td>
<td>C.P. 46-201</td>
<td>none</td>
<td>158</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>49-145</td>
<td>C.P. 27-108</td>
<td>none</td>
<td>917</td>
<td>19.3</td>
<td>22.25</td>
</tr>
<tr>
<td>49-3</td>
<td>C.P. 30-24</td>
<td>none</td>
<td>244</td>
<td>0.4</td>
<td>1.6</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: res.
Table 6. Mosaic resistance data: Analysis relative to female parent C. P. 36-13.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-4</td>
<td>C.P. 30-24</td>
<td>none</td>
<td>1075</td>
<td>8.56</td>
<td>10.1</td>
</tr>
<tr>
<td>48-7</td>
<td>C.P. 36-138</td>
<td>90%</td>
<td>512</td>
<td>15.62</td>
<td>21.09</td>
</tr>
<tr>
<td>48-8</td>
<td>C.P. 36-105</td>
<td>low %</td>
<td>1893</td>
<td>2.54</td>
<td>3.59</td>
</tr>
<tr>
<td>48-17</td>
<td>C.P. 1161</td>
<td>none</td>
<td>792</td>
<td>36.11</td>
<td>36.86</td>
</tr>
<tr>
<td>48-18</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>525</td>
<td>3.62</td>
<td>5.71</td>
</tr>
<tr>
<td>48-19</td>
<td>C.P. 33-372</td>
<td>none</td>
<td>482</td>
<td>8.3</td>
<td>14.11</td>
</tr>
<tr>
<td>48-20</td>
<td>F. 36-372</td>
<td>none</td>
<td>497</td>
<td>35.81</td>
<td>38.83</td>
</tr>
<tr>
<td>48-21</td>
<td>Co. 285</td>
<td>none</td>
<td>416</td>
<td>20.43</td>
<td>35.57</td>
</tr>
<tr>
<td>48-22</td>
<td>C.P. 1165</td>
<td>none</td>
<td>712</td>
<td>10.25</td>
<td>11.10</td>
</tr>
<tr>
<td>48-67</td>
<td>C.P. 36-156</td>
<td>?</td>
<td>461</td>
<td>23.64</td>
<td>26.03</td>
</tr>
<tr>
<td>48-78</td>
<td>C.P. 27-108</td>
<td>none</td>
<td>100</td>
<td>4.00</td>
<td>11.0</td>
</tr>
<tr>
<td>48-80</td>
<td>C.P. 36-211</td>
<td>none</td>
<td>134</td>
<td>3.33</td>
<td>3.33</td>
</tr>
<tr>
<td>48-85</td>
<td>C.P. 44-156</td>
<td>none</td>
<td>212</td>
<td>0.0</td>
<td>0.47</td>
</tr>
<tr>
<td>48-107</td>
<td>C.P. 43-74</td>
<td>80%</td>
<td>714</td>
<td>7.42</td>
<td>21.29</td>
</tr>
<tr>
<td>48-128</td>
<td>C.P. 33-224</td>
<td>none</td>
<td>291</td>
<td>0.0</td>
<td>1.72</td>
</tr>
<tr>
<td>48-141</td>
<td>C.P. 42-10</td>
<td>trace</td>
<td>154</td>
<td>0.0</td>
<td>5.84</td>
</tr>
<tr>
<td>48-192</td>
<td>C.P. 33-150</td>
<td>20%</td>
<td>291</td>
<td>0.68</td>
<td>1.72</td>
</tr>
<tr>
<td>48-198</td>
<td>P. 33-37</td>
<td>trace</td>
<td>102</td>
<td>0.0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: Res.
Table 7. Mosaic resistance data; Analysis relative to female parent C. P. 36-105.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-44</td>
<td>C.P. 38-41</td>
<td>none</td>
<td>552</td>
<td>25.72</td>
<td>27.17</td>
</tr>
<tr>
<td>48-86</td>
<td>C. P. 44-156</td>
<td>none</td>
<td>158</td>
<td>0.0</td>
<td>6.06</td>
</tr>
<tr>
<td>48-87</td>
<td>C.P. 33-224</td>
<td>none</td>
<td>174</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>48-195</td>
<td>Open Pollen</td>
<td>?</td>
<td>594</td>
<td>0.0</td>
<td>4.04</td>
</tr>
<tr>
<td>49-94</td>
<td>C.F. 47-120</td>
<td>none</td>
<td>199</td>
<td>13.5</td>
<td>17.5</td>
</tr>
<tr>
<td>49-95</td>
<td>C.F. 36-211</td>
<td>none</td>
<td>493</td>
<td>4.66</td>
<td>8.11</td>
</tr>
<tr>
<td>49-105</td>
<td>C.P. 27-108</td>
<td>none</td>
<td>123</td>
<td>26.83</td>
<td>27.64</td>
</tr>
<tr>
<td>49-140</td>
<td>P. 33-37</td>
<td>trace</td>
<td>673</td>
<td>67.76</td>
<td>69.24</td>
</tr>
<tr>
<td>49-172</td>
<td>73-45</td>
<td>none</td>
<td>307</td>
<td>36.48</td>
<td>36.80</td>
</tr>
<tr>
<td>49-281</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>688</td>
<td>54.8</td>
<td>60.17</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: low %
Table 8. Mosaic resistance data: Analysis relative to female parent C. P. 38-34.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>49-70</td>
<td>C. P. 43-74</td>
<td>80%</td>
<td>96</td>
<td>17.7</td>
<td>18.75</td>
</tr>
<tr>
<td>49-75</td>
<td>C. P. 27-108</td>
<td>none</td>
<td>344</td>
<td>6.7</td>
<td>12.5</td>
</tr>
<tr>
<td>49-77</td>
<td>C. P. 30-24</td>
<td>none</td>
<td>264</td>
<td>15.53</td>
<td>16.2</td>
</tr>
<tr>
<td>49-78</td>
<td>28NG39-491</td>
<td>?</td>
<td>158</td>
<td>79.74</td>
<td>80.3</td>
</tr>
<tr>
<td>49-98</td>
<td>C. P. 1165</td>
<td>none</td>
<td>133</td>
<td>42.1</td>
<td>42.1</td>
</tr>
<tr>
<td>49-99</td>
<td>C. P. 36-211</td>
<td>none</td>
<td>687</td>
<td>47.6</td>
<td>50.51</td>
</tr>
<tr>
<td>49-177</td>
<td>F. 33-37</td>
<td>none</td>
<td>177</td>
<td>69.49</td>
<td>70.62</td>
</tr>
<tr>
<td>49-30</td>
<td>Co. 356</td>
<td>none</td>
<td>690</td>
<td>9.1</td>
<td>14.6</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: 20%
Table 9. Mosaic resistance data: Analysis relative to female parent C. P. 43-64.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-54</td>
<td>C. P. 36-105</td>
<td>low %</td>
<td>708</td>
<td>52.82</td>
<td>62.30</td>
</tr>
<tr>
<td>48-55</td>
<td>C. P. 30-24</td>
<td>none</td>
<td>265</td>
<td>37.74</td>
<td>43.01</td>
</tr>
<tr>
<td>48-56</td>
<td>C. P. 36-156</td>
<td>?</td>
<td>225</td>
<td>8.89</td>
<td>20.44</td>
</tr>
<tr>
<td>48-58</td>
<td>C. P. 36-211</td>
<td>none</td>
<td>484</td>
<td>63.64</td>
<td>65.91</td>
</tr>
<tr>
<td>48-64</td>
<td>C. P. 36-138</td>
<td>90%</td>
<td>175</td>
<td>5.71</td>
<td>13.22</td>
</tr>
<tr>
<td>48-65</td>
<td>C. P. 27-108</td>
<td>none</td>
<td>625</td>
<td>4.16</td>
<td>6.56</td>
</tr>
<tr>
<td>48-66</td>
<td>C. P. 1165</td>
<td>none</td>
<td>224</td>
<td>0.0</td>
<td>4.51</td>
</tr>
<tr>
<td>48-69</td>
<td>C. P. 38-34</td>
<td>20%</td>
<td>380</td>
<td>0.26</td>
<td>8.68</td>
</tr>
<tr>
<td>48-71</td>
<td>C. P. 1161</td>
<td>none</td>
<td>208</td>
<td>40.87</td>
<td>42.79</td>
</tr>
<tr>
<td>48-81</td>
<td>C. P. 44-156</td>
<td>none</td>
<td>174</td>
<td>0.0</td>
<td>5.75</td>
</tr>
<tr>
<td>48-82</td>
<td>C. P. 33-224</td>
<td>none</td>
<td>196</td>
<td>0.4</td>
<td>10.20</td>
</tr>
<tr>
<td>48-99</td>
<td>C. P. 48-106</td>
<td>20%</td>
<td>125</td>
<td>2.4</td>
<td>7.20</td>
</tr>
<tr>
<td>48-110</td>
<td>C. P. 43-74</td>
<td>80%</td>
<td>194</td>
<td>0.0</td>
<td>2.58</td>
</tr>
<tr>
<td>48-125</td>
<td>P.O.J. 2575 × C. P. 36-105</td>
<td>?</td>
<td>267</td>
<td>1.5</td>
<td>9.66</td>
</tr>
<tr>
<td>49-31</td>
<td>C. P. 33-372</td>
<td>none</td>
<td>482</td>
<td>37.14</td>
<td>44.81</td>
</tr>
<tr>
<td>49-59</td>
<td>Co. 356</td>
<td>none</td>
<td>467</td>
<td>9.2</td>
<td>13.0</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: 50%
Table 10. Mosaic resistance data: Analysis relative to female parent Co. 281.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-135</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>128</td>
<td>13.28</td>
<td>18.75</td>
</tr>
<tr>
<td>48-136</td>
<td>C.P. 33-224</td>
<td>none</td>
<td>115</td>
<td>0.0</td>
<td>24.35</td>
</tr>
<tr>
<td>48-164</td>
<td>C.P. 43-74</td>
<td>80%</td>
<td>147</td>
<td>10.38</td>
<td>26.53</td>
</tr>
<tr>
<td>48-165</td>
<td>C.P. 36-211</td>
<td>none</td>
<td>223</td>
<td>0.0</td>
<td>8.52</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: 100%
Table 11. Mosaic resistance data: Analysis relative to female parent F. 31-962.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-130</td>
<td>C.P. 36-105</td>
<td>low %</td>
<td>203</td>
<td>0</td>
<td>9.36</td>
</tr>
<tr>
<td>48-154</td>
<td>C.P. 27-38</td>
<td>none</td>
<td>175</td>
<td>0</td>
<td>0.57</td>
</tr>
<tr>
<td>48-156</td>
<td>C.P. 27-48</td>
<td>none</td>
<td>166</td>
<td>9.04</td>
<td>13.25</td>
</tr>
<tr>
<td>48-157</td>
<td>C.P. 44-156</td>
<td>none</td>
<td>146</td>
<td>0.68</td>
<td>10.27</td>
</tr>
<tr>
<td>48-166</td>
<td>C.P. 43-74</td>
<td>80%</td>
<td>278</td>
<td>3.24</td>
<td>8.99</td>
</tr>
<tr>
<td>48-179</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>112</td>
<td>1.79</td>
<td>4.46</td>
</tr>
<tr>
<td>48-188</td>
<td>C.P. 27-108</td>
<td>none</td>
<td>157</td>
<td>0</td>
<td>5.73</td>
</tr>
<tr>
<td>48-201</td>
<td>C.P. 36-138</td>
<td>90%</td>
<td>200</td>
<td>15.5</td>
<td>24.00</td>
</tr>
<tr>
<td>48-202</td>
<td>U.S. 1694</td>
<td>none</td>
<td>149</td>
<td>0</td>
<td>14.76</td>
</tr>
<tr>
<td>49-236</td>
<td>Naz. 716</td>
<td>?</td>
<td>1105</td>
<td>58.46</td>
<td>59.64</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: Trace
Table 12. Mosaic resistance data: Analysis relative to female parent F. 36-819. *

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-76</td>
<td>C.P. 27-108</td>
<td>none</td>
<td>324</td>
<td>0</td>
<td>2.78</td>
</tr>
<tr>
<td>49-184</td>
<td>C.P. 49-49</td>
<td>none</td>
<td>762</td>
<td>85.17</td>
<td>87.93</td>
</tr>
<tr>
<td>49-201</td>
<td>C.P. 48-116</td>
<td>none</td>
<td>221</td>
<td>59.7</td>
<td>59.7</td>
</tr>
<tr>
<td>49-225</td>
<td>71-46</td>
<td>none</td>
<td>212</td>
<td>70.6</td>
<td>70.6</td>
</tr>
<tr>
<td>49-226</td>
<td>88-45</td>
<td>20%</td>
<td>1010</td>
<td>94.26</td>
<td>94.46</td>
</tr>
<tr>
<td>49-243</td>
<td>512-43</td>
<td>none</td>
<td>1644</td>
<td>57.96</td>
<td>60.1</td>
</tr>
<tr>
<td>49-257</td>
<td>36-46</td>
<td>none</td>
<td>695</td>
<td>75.1</td>
<td>75.97</td>
</tr>
<tr>
<td>49-265</td>
<td>610-45</td>
<td>20%</td>
<td>224</td>
<td>57.14</td>
<td>57.14</td>
</tr>
<tr>
<td>49-266</td>
<td>49-9</td>
<td>10%</td>
<td>897</td>
<td>20.28</td>
<td>20.84</td>
</tr>
<tr>
<td>49-271</td>
<td>28NG72-514</td>
<td>?</td>
<td>323</td>
<td>43.0</td>
<td>44.0</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: none
Table 13. Mosaic resistance data: Analysis relative to female parent Imp. 1183.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-162</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>129</td>
<td>1.55</td>
<td>5.42</td>
</tr>
<tr>
<td>48-180</td>
<td>C.P. 43-74</td>
<td>80%</td>
<td>149</td>
<td>2.01</td>
<td>35.57</td>
</tr>
<tr>
<td>48-181</td>
<td>C.P. 42-10</td>
<td>trace</td>
<td>154</td>
<td>0</td>
<td>3.90</td>
</tr>
<tr>
<td>48-189</td>
<td>(X17-6-46)</td>
<td>?</td>
<td>106</td>
<td>0</td>
<td>0.00</td>
</tr>
<tr>
<td>48-209</td>
<td>C.P. 36-211</td>
<td>none</td>
<td>120</td>
<td>0</td>
<td>5.00</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: 40%
Table 14. Mosaic resistance data: Analysis relative to female parent P.O.J. 2725.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Male Parent</th>
<th>Reaction of male parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-13</td>
<td>C.P. 30-24</td>
<td>none</td>
<td>269</td>
<td>39.94</td>
<td>37.17</td>
</tr>
<tr>
<td>48-14</td>
<td>C.P. 1165</td>
<td>none</td>
<td>259</td>
<td>20.46</td>
<td>22.01</td>
</tr>
<tr>
<td>48-34</td>
<td>(P.O.J. 2725 X C.P. 30-138)</td>
<td>?</td>
<td>106</td>
<td>29.24</td>
<td>35.85</td>
</tr>
<tr>
<td>48-35</td>
<td>C.P. 27-108</td>
<td>none</td>
<td>218</td>
<td>22.47</td>
<td>23.85</td>
</tr>
<tr>
<td>48-37</td>
<td>C.P. 33-229</td>
<td>none</td>
<td>280</td>
<td>26.43</td>
<td>26.43</td>
</tr>
<tr>
<td>48-47</td>
<td>C.P. 38-41</td>
<td>none</td>
<td>426</td>
<td>54.93</td>
<td>54.93</td>
</tr>
<tr>
<td>48-53</td>
<td>C.P. 36-105</td>
<td>low %</td>
<td>169</td>
<td>14.2</td>
<td>20.71</td>
</tr>
<tr>
<td>48-57</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>371</td>
<td>76.54</td>
<td>77.36</td>
</tr>
<tr>
<td>48-102</td>
<td>C.P. 48-106</td>
<td>20%</td>
<td>358</td>
<td>0.11</td>
<td>13.13</td>
</tr>
<tr>
<td>49-66</td>
<td>C.P. 43-74</td>
<td>80%</td>
<td>370</td>
<td>1.35</td>
<td>2.43</td>
</tr>
<tr>
<td>49-67</td>
<td>Co. 356</td>
<td>none</td>
<td>1688</td>
<td>3.2</td>
<td>5.27</td>
</tr>
<tr>
<td>50-65</td>
<td>C.P. 42-13</td>
<td>none</td>
<td>212</td>
<td>34.9</td>
<td>36.3</td>
</tr>
<tr>
<td>50-66</td>
<td>C.P. 48-53</td>
<td>none</td>
<td>108</td>
<td>25.9</td>
<td>25.9</td>
</tr>
</tbody>
</table>

* Reaction of female parent to mosaic: none
Table 1 shows the results obtained when C. P. 27-139, a variety resistant to mosaic, was used as a female parent. When crossed with C. P. 44-156, a variety resistant to mosaic, only 1.78 per cent of the progeny showed mosaic symptoms. When C. P. 36-105, a variety that shows usually a low percentage of mosaic, was used as a male parent, the progeny showed no mosaic symptoms. When C. P. 43-74, a variety very susceptible to mosaic, was used as a male parent, 8.4 per cent of the progeny showed symptoms of this disease.

In Table 2, are given the results obtained when C. P. 29-103, a variety that shows a trace of mosaic, was used as a female parent. When it was crossed with resistant varieties such as C. P. 38-41, C. P. 33-872, C. P. 27-48, and C. P. 48-126, the percentages of mosaic symptoms observed in the progeny were, 67.6, 65.9, 41.6 and 83.6 per cent respectively. These are very high percentages of mosaic. When the same female parent was crossed with C. P. 43-74, a very susceptible variety, only 22.1 per cent of the progeny showed the disease symptoms. A more or less similar ratio of mosaic diseased plants was obtained when the same female parent was crossed with other resistant varieties C. P. 33-224, C. P. 27-108, F. 36-273 and C. P. 33-229. The percentages of diseased plants in the progeny of these crosses were 25.1, 26.3, 18.1, and 21.0 per cent. These results show that moderate to high percentages of mosaic-diseased plants were obtained when C. P. 29-103 was used as a female parent. However, only in 3 out of 14 crosses lower percentages of diseased plants were obtained in the progeny. When C. P. 44-156, C. P. 42-10, and U. S. 1694 were used as males, percentages of diseased plants in the progeny were 7.8, 9.0 and 1.9 per cent.
The results obtained when C. P. 29-116, a variety that shows traces of mosaic, was used as a female parent are given in Table 3. When it was crossed with the susceptible varieties, C. P. 48-106, and C. P. 43-74, percentages of 4.9 and 6.1 of diseased plants were obtained in the progeny. Similar low percentages of mosaic were obtained in the progeny of crosses with the resistant varieties, C. P. 30-24, C. P. 33-224, C. P. 44-156, C. P. 46-199, C. P. 46-73 and Co. 356. The percentages of mosaic infected plants in the progeny of these crosses were 2.7, 0.8, 1.3, 1.9, 4.3 and 5.8 respectively. Although low percentages of mosaic-diseased plants were obtained in the progeny of 8 crosses, a moderately high percentage of diseased plants was obtained when the same female parent, C. P. 29-116, was crossed with four other male varieties C. P. 38-34, C. P. 36-138, C. P. 36-105 and C. P. 36-156. The first two male varieties are susceptible to mosaic, the third one shows a low percentage of mosaic, and the reaction of the fourth variety to mosaic is unknown. The percentages of mosaic-infected plants in the progeny of these crosses were 16.9, 23.9, 30.0 and 36.4 respectively.

Table 4 shows the results obtained when C. P. 33-229, a mosaic-resistant variety was used as a female in 6 crosses. Low percentages of mosaic were obtained in the progeny of the crosses with the resistant varieties C. P. 33-224, C. P. 36-211 and Co. 356. The percentages of diseased plants in the progeny of these crosses were 1.8, 5.0 and 0.5 respectively. In addition when C. P. 33-229 was used as a female in an open-pollinated cross, 2.4 per cent of the progeny showed mosaic symptoms. When it was crossed with the susceptible variety C. P. 43-74, 17.1 per cent of the progeny showed the disease symptoms. Another moderate percentage of mosaic (14.6) was obtained in the cross with the
male variety C. P. 33-372.

Variety C. P. 34-120 was used as a female parent in 19 crosses.

The results given in table 5 show that a very low percentage of mosaic-diseased progeny was obtained when it was crossed with the resistant parents C. P. 33-372, C. P. 38-41, F. 36-273, C. P. 33-229, C. P. 27-48 and C. P. 46-201. The percentages of mosaic obtained were 1.5, 5.3, 2.4, 0.8, 10.01 and 0.0. Variety C. P. 30-24 was crossed with C. P. 34-120 in 1948 as well as in 1949. The percentages of mosaic-diseased progeny were 2.7 and 1.6 respectively. Low percentages of diseased progeny were also obtained when C. P. 34-120 was crossed with varieties C. P. 38-12, C. P. 42-10 and (P.O.J. 2725 x C. P. 36 x 105). The first variety was resistant to mosaic, the second one showed a trace of mosaic, while the third variety's reaction to mosaic was unknown. The susceptible varieties C. P. 36-138, C. P. 38-34 and C. P. 43-64 were crossed with C. P. 34-120. The percentages of diseased plants among the progeny were 23.5, 0.89 and 0.75. It is observed that very low percentages of diseased plants were obtained in the crosses with two susceptible male parents, while in another cross with a susceptible male parent a moderate percentage of diseased plants was obtained in the progeny. However, other moderate percentages of mosaic were obtained in the progeny of crosses which involved the resistant male parents C. P. 33-224, and C. P. 27-108, as well as in the progeny of the crosses which involved the male parent C. P. 36-105, a variety whose reaction to mosaic was reported as having low percentages of mosaic. It is also observed in table 5, that when C. P. 34-120 was used as a female parent in 19 crosses, there were very low percentages of diseased plants in the progeny of 14 crosses, and moderate percentages of mosaic-diseased
plants in the progeny of the other 5 crosses.

Table 6 shows the results obtained when C. P. 36-13, a mosaic-resistant variety, was used as a female parent in crosses with 18 difference varieties. Low percentages of mosaic-diseased plants were obtained in the progeny of the crosses with the mosaic resistant varieties C. P. 36-211, C. P. 44-156, C. P. 33-224, P. 33-37. The percentages of mosaic obtained were 3.3, 0.5, 1.7 and 0.0. In addition, low percentages of mosaic (5.7 and 1.72) were obtained in the crosses with the mosaic-susceptible varieties C. P. 38-34 and C. P. 44-150, as well as with crosses of varieties showing a trace of mosaic, as C. P. 42-10 and C. P. 36-105. Moderate percentages of mosaic were obtained when variety C. P. 36-13 was crossed with the resistant varieties C. P. 30-24, C. P. 1165 and C. P. 27-108, and C. P. 33-372 as well as with the susceptible varieties C. P. 36-138 and C. P. 43-74. The percentages of mosaic-infected plants were 10.1, 11.1, 11.0, 14.11, 21.1 and 21.3. Higher percentages of mosaic were recorded when resistant varieties C. P. 1161, F. 36-273 and Co. 285 were used as male parents. It is observed from table 6 that when C. P. 36-13 was used as a female parent in different crosses, in 8 crosses out of 18 there were very low percentages of mosaic-diseased plants in the progeny and that it occurred whether a mosaic-resistant or a mosaic-susceptible variety was used as a male parent. Moderate percentages of mosaic were obtained in the progeny of 6 crosses that involved a resistant or a susceptible variety as male parents. In 4 crosses, higher percentages of mosaic-diseased plants were obtained when three different resistant parents were used as male parents while the reaction to mosaic of the fourth male parent was unknown.
When using, as a female parent, C. P. 36-105, a variety that shows a low percentage of mosaic diseased plants in the field, results were obtained as shown in table 7. It was crossed with 7 resistant varieties, and the percentages of mosaic-diseased plants in the progeny of these crosses varied from 36.8 to no diseased plants at all. When it was crossed with the resistant varieties C. P. 48-41, C. P. 44-156, C. P. 33-224, C. P. 47-120, C. P. 36-211, C. P. 27-108 and 73-45, the percentages of mosaic-diseased plants in the progeny were 27.2, 6.1, 0.0, 17.5, 8.1, 27.6, 36.8 respectively. Higher percentages of mosaic-diseased plants were obtained when the same female parent, C. P. 36-105, was crossed with the susceptible varieties P. 33-37 and C. P. 38-34. The percentages of diseased plants in the progeny of these crosses were 69.2 and 60.1 respectively.

Table 8 shows the results obtained when C. P. 38-34, a mosaic susceptible variety was used as a female parent in 8 crosses. When it was crossed with the mosaic susceptible variety C. P. 43-74, in the progeny the percentage of plants showing mosaic symptoms was less than when it was crossed with the resistant varieties C. P. 1165, C. P. 36-211, P. 33-37 and Co. 356. On the other hand when the same female parent was crossed with the resistant varieties C. P. 27-108, C. P. 30-24, and Co. 356 in the progeny the percentage of diseased plants was slightly lower than when the mosaic susceptible variety C. P. 43-74 was used as the male parent.

In table 9 are given the results obtained when the mosaic susceptible variety C. P. 43-64 was used as a female parent in crosses with 17 different varieties. The least percentage of mosaic (2.6) was obtained when the susceptible variety C. P. 43-74 was used as a male
parent. Considerably high percentages of mosaic diseased plants were obtained in the progeny of the crosses with the resistant varieties C. P. 30-24, C. P. 36-211, C. P. 1161 and C. P. 33-372. Low percentages of mosaic diseased plants were obtained in the progeny of the crosses with the resistant varieties C. P. 27-108, C. P. 1165, C. P. 44-156 and C. P. 33-224, as well as in the progeny of the crosses with the susceptible varieties C. P. 38-34 and C. P. 43-74. A similar percentage of mosaic diseased plants were obtained when the same female parent C. P. 43-64 was crossed with the resistant parent Co. 356 and when it was crossed with the susceptible parent C. P. 36-138.

The mosaic susceptible variety Co. 281 was used as a female parent with 4 different varieties. The results given in table 10 show that when the susceptible varieties C. P. 38-34 and C. P. 43-74 were used as male parents, the percentages of mosaic diseased progeny of these crosses were 18.8 and 26.5, respectively, while in the crosses with the resistant varieties C. P. 33-224 and C. P. 36-211 the percentages of diseased plants in the progeny were 24.4 and 8.5, respectively.

Table 11 shows the results obtained when F. 31-962, a variety that shows a trace of mosaic, was used as a female in crosses with 10 different varieties. When it was crossed with the resistant varieties C. P. 27-38 and C. P. 27-108, and with the susceptible variety C. P. 38-34, percentages of 0.6, 5.7 and 4.5 of diseased plants were obtained in the progeny. Moderate percentages of mosaic diseased plants were obtained when the same female parent was crossed with the resistant varieties C. P. 27-48, C. P. 44-156, U. S. 1694, as well as with the susceptible varieties, C. P. 43-74 and C. P. 36-138. The percentages of diseased plants in these crosses were 13.22, 10.3, 14.8, 8.9 and 24.0 respectively.
It is noticed that low to moderate percentages of diseased plants were obtained in the progeny of nine crosses when variety F. 31-962 was used as a female parent, only in the cross with variety Nas. 716 was a high percentage of diseased plants observed in the progeny.

Table 12 shows the results obtained when F. 36-819, a mosaic resistant variety was used as a female parent in crosses with 10 different varieties. Only one cross was made in 1948 and the percentage of mosaic diseased plants in the progeny was very low (2.78). The progeny of the 1949 crosses showed high percentages of diseased plants. The percentages of diseased plants recorded in the progeny with the resistant varieties, C. P. 49-49, C. P. 48-116, 71-64, 512-43 and 36-46 were 87.9, 59.7, 70.6, 60.1 and 75.97. In the crosses with the susceptible varieties 88-45, 610-45 and 49-9 the percentages of diseased plants in the progeny were 70.6, 57.1 and 20.8.

Table 13 shows that when Imp. 1183, a mosaic susceptible variety was crossed with each of the resistant variety C. P. 36-211, the susceptible variety C. P. 38-34, and with the C. P. 42-10 a variety that shows a trace of mosaic, low percentages of mosaic diseased plants were obtained in the progeny of these crosses. The percentages of diseased plants obtained were 5.0, 5.4 and 3.9. No diseased plants were obtained when the same female parent, Imp. 1183, was crossed with variety (X17-6-46). The reaction of the latter variety to mosaic is unknown. Only in one out of the five crosses when Imp. 1183 was used as a female parent high percentage of diseased plants was observed in the progeny. This was observed in the cross with the susceptible variety C. P. 43-74.

Variety P.O.J. 2725, a mosaic resistant variety, was crossed with
13 other varieties. Table 14 shows that low percentages of mosaic diseased plants were observed only in the progeny of two crosses; namely with the susceptible variety C. P. 43-74 and with the resistant variety Co. 356. A moderate percentage (13.1) of diseased plants was observed when the same female parent was crossed with the susceptible variety C. P. 48-106. Higher percentages of diseased plants were obtained in the progeny of the crosses involving P.O.J. 2725 with each of the following mosaic resistant varieties: C.P. 30-24, C. P. 1165, C. P. 27-108, C. P. 33-229, C. P. 38-41, C. P. 42-13 and C. P. 48-53. The percentages of diseased plants were 37.2, 22.0, 23.8, 26.4, 54.9, and 25.9. In the cross with susceptible variety C. P. 38-34, 77.4 per cent of the progeny showed mosaic symptoms.

Each of the tables 15 to 32 shows the reaction to sugarcane mosaic virus of the progeny of one male when used in crosses with several females.
Table 15. Mosaic resistant data: Analysis relative to male parent C. P. 27-108.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-194</td>
<td>Open pollen</td>
<td>?</td>
<td>235</td>
<td>0.0</td>
<td>2.13</td>
</tr>
<tr>
<td>48-70</td>
<td>C.P. 29-103</td>
<td>trace</td>
<td>714</td>
<td>24.64</td>
<td>26.33</td>
</tr>
<tr>
<td>48-78</td>
<td>C.P. 36-13</td>
<td>res.</td>
<td>100</td>
<td>4.0</td>
<td>11.0</td>
</tr>
<tr>
<td>48-77</td>
<td>C.P. 38-27</td>
<td>none</td>
<td>286</td>
<td>0.0</td>
<td>5.24</td>
</tr>
<tr>
<td>48-65</td>
<td>C.P. 43-64</td>
<td>50%</td>
<td>625</td>
<td>4.16</td>
<td>6.56</td>
</tr>
<tr>
<td>48-188</td>
<td>F. 31-962</td>
<td>trace</td>
<td>157</td>
<td>0.0</td>
<td>5.73</td>
</tr>
<tr>
<td>48-76</td>
<td>F. 36-819</td>
<td>none</td>
<td>324</td>
<td>0.0</td>
<td>2.78</td>
</tr>
<tr>
<td>48-35</td>
<td>P.O.J. 2725</td>
<td>none</td>
<td>218</td>
<td>22.47</td>
<td>23.85</td>
</tr>
<tr>
<td>49-74</td>
<td>C.P. 33-224</td>
<td>none</td>
<td>464</td>
<td>3.9</td>
<td>5.2</td>
</tr>
<tr>
<td>49-75</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>344</td>
<td>6.7</td>
<td>12.5</td>
</tr>
<tr>
<td>49-105</td>
<td>C.P. 36-105</td>
<td>low %</td>
<td>123</td>
<td>26.8</td>
<td>27.64</td>
</tr>
<tr>
<td>49-145</td>
<td>C.P. 34-120</td>
<td>res.</td>
<td>917</td>
<td>19.3</td>
<td>22.25</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: none
Table 16. Mosaic resistance data: Analysis relative to male parent C. P. 30-24.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-5</td>
<td>C.P. 29-116</td>
<td>trace</td>
<td>402</td>
<td>2.74</td>
<td>2.74</td>
</tr>
<tr>
<td>48-12</td>
<td>C.P. 29-120</td>
<td>trace</td>
<td>121</td>
<td>7.48</td>
<td>9.92</td>
</tr>
<tr>
<td>48-3</td>
<td>C.P. 34-120</td>
<td>res.</td>
<td>1456</td>
<td>1.92</td>
<td>2.68</td>
</tr>
<tr>
<td>48-4</td>
<td>C.P. 36-13</td>
<td>res.</td>
<td>1075</td>
<td>8.56</td>
<td>10.14</td>
</tr>
<tr>
<td>48-55</td>
<td>C.P. 43-64</td>
<td>50%</td>
<td>265</td>
<td>37.74</td>
<td>43.01</td>
</tr>
<tr>
<td>48-74</td>
<td>H. 32-8560</td>
<td>trace</td>
<td>96</td>
<td>0.0</td>
<td>1.04</td>
</tr>
<tr>
<td>48-13</td>
<td>P.O. 2725</td>
<td>none</td>
<td>269</td>
<td>34.94</td>
<td>37.17</td>
</tr>
<tr>
<td>49-3</td>
<td>C.P. 34-120</td>
<td>res.</td>
<td>244</td>
<td>0.4</td>
<td>1.64</td>
</tr>
<tr>
<td>49-4</td>
<td>C.P. 42-13</td>
<td>none</td>
<td>947</td>
<td>4.2</td>
<td>6.4</td>
</tr>
<tr>
<td>49-8</td>
<td>C.P. 44-126</td>
<td>none</td>
<td>309</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>49-27</td>
<td>C. 42-70</td>
<td>none</td>
<td>162</td>
<td>16.1</td>
<td>16.66</td>
</tr>
<tr>
<td>49-77</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>264</td>
<td>15.5</td>
<td>16.2</td>
</tr>
<tr>
<td>50-30</td>
<td>C.P. 44-126</td>
<td>none</td>
<td>178</td>
<td>5.3</td>
<td>5.3</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: none
Table 17. Mosaic resistance data: Analysis relative to male parent C. P. 33-224. *

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-61</td>
<td>C.P. 29-103</td>
<td>trace</td>
<td>402</td>
<td>22.89</td>
<td>25.12</td>
</tr>
<tr>
<td>48-101</td>
<td>C.P. 29-116</td>
<td>trace</td>
<td>383</td>
<td>0.0</td>
<td>0.78</td>
</tr>
<tr>
<td>48-167</td>
<td>C.P. 33-229</td>
<td>none</td>
<td>282</td>
<td>0.0</td>
<td>1.77</td>
</tr>
<tr>
<td>48-42</td>
<td>C.P. 34-120</td>
<td>res.</td>
<td>488</td>
<td>15.16</td>
<td>16.8</td>
</tr>
<tr>
<td>48-128</td>
<td>C.P. 36-13</td>
<td>res.</td>
<td>291</td>
<td>0.0</td>
<td>1.72</td>
</tr>
<tr>
<td>48-87</td>
<td>C.P. 36-105</td>
<td>low %</td>
<td>174</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>48-82</td>
<td>C.P. 43-64</td>
<td>50 %</td>
<td>196</td>
<td>0.0</td>
<td>10.20</td>
</tr>
<tr>
<td>48-136</td>
<td>Co. 281</td>
<td>100 %</td>
<td>115</td>
<td>0.0</td>
<td>24.35</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic; none
Table 18. Mosaic resistance data: Analysis relative to male parent C. P. 33-372.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-49</td>
<td>C.P. 29-103</td>
<td>trace</td>
<td>514</td>
<td>63.22</td>
<td>65.95</td>
</tr>
<tr>
<td>48-19</td>
<td>C.P. 36-13</td>
<td>res.</td>
<td>482</td>
<td>8.3</td>
<td>14.11</td>
</tr>
<tr>
<td>48-41</td>
<td>C.P. 34-120</td>
<td>res.</td>
<td>269</td>
<td>0.3</td>
<td>1.49</td>
</tr>
<tr>
<td>49-31</td>
<td>C.P. 43-64</td>
<td>50%</td>
<td>482</td>
<td>37.1</td>
<td>44.81</td>
</tr>
<tr>
<td>49-103</td>
<td>C.P. 33-229</td>
<td>none</td>
<td>446</td>
<td>13.5</td>
<td>14.57</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: none
**Table 19. Mosaic resistance data: Analysis relative to male parent C. P. 36-105, *\(^\dagger\)**

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discard</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-114</td>
<td>C.P. 27-139</td>
<td>none</td>
<td>197</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>48-48</td>
<td>C.P. 29-103</td>
<td>trace</td>
<td>306</td>
<td>58.17</td>
<td>60.13</td>
</tr>
<tr>
<td>48-25</td>
<td>C.P. 29-116</td>
<td>trace</td>
<td>236</td>
<td>28.39</td>
<td>30.01</td>
</tr>
<tr>
<td>48-23</td>
<td>C.P. 34-120</td>
<td>res.</td>
<td>163</td>
<td>16.56</td>
<td>23.31</td>
</tr>
<tr>
<td>48-8</td>
<td>C.P. 36-13</td>
<td>res.</td>
<td>1893</td>
<td>2.54</td>
<td>3.59</td>
</tr>
<tr>
<td>48-95</td>
<td>F. 40-96</td>
<td>none</td>
<td>164</td>
<td>0.0</td>
<td>0.61</td>
</tr>
<tr>
<td>48-54</td>
<td>C.P. 43-64</td>
<td>50%</td>
<td>708</td>
<td>52.82</td>
<td>62.30</td>
</tr>
<tr>
<td>48-130</td>
<td>F. 31-962</td>
<td>trace</td>
<td>203</td>
<td>0.0</td>
<td>9.36</td>
</tr>
<tr>
<td>48-53</td>
<td>P.O.J. 2725</td>
<td>none</td>
<td>169</td>
<td>14.2</td>
<td>20.71</td>
</tr>
<tr>
<td>49-1</td>
<td>C.P. 42-13</td>
<td>none</td>
<td>246</td>
<td>6.5</td>
<td>6.5</td>
</tr>
<tr>
<td>49-24</td>
<td>Cl. 41-142</td>
<td>none</td>
<td>225</td>
<td>28.8</td>
<td>30.66</td>
</tr>
<tr>
<td>49-45</td>
<td>C.P. 28-19</td>
<td>trace</td>
<td>413</td>
<td>20.3</td>
<td>26.8</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: low %
Table 20. Mosaic resistance data: Analysis relative to male parent C. P. 36-138.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-63</td>
<td>C.P. 29-116</td>
<td>trace</td>
<td>192</td>
<td>20.31</td>
<td>23.96</td>
</tr>
<tr>
<td>48-40</td>
<td>C.P. 34-120</td>
<td>res.</td>
<td>715</td>
<td>19.44</td>
<td>23.5</td>
</tr>
<tr>
<td>48-41</td>
<td>C.P. 33-372</td>
<td>none</td>
<td>269</td>
<td>0.37</td>
<td>1.49</td>
</tr>
<tr>
<td>48-7</td>
<td>C.P. 36-13</td>
<td>res.</td>
<td>512</td>
<td>15.62</td>
<td>21.09</td>
</tr>
<tr>
<td>48-64</td>
<td>C.P. 43-64</td>
<td>50%</td>
<td>175</td>
<td>3.71</td>
<td>13.22</td>
</tr>
<tr>
<td>48-201</td>
<td>F; 31-962</td>
<td>trace</td>
<td>200</td>
<td>15.5</td>
<td>24.00</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: 90%
Table 21. Mosaic resistance data: Analysis relative to male parent C. P. 36-211. *

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-80</td>
<td>C. P. 36-13</td>
<td>res.</td>
<td>134</td>
<td>3.33</td>
<td>3.33</td>
</tr>
<tr>
<td>48-58</td>
<td>C. P. 43-64</td>
<td>50%</td>
<td>484</td>
<td>63.64</td>
<td>65.91</td>
</tr>
<tr>
<td>48-165</td>
<td>Co. 281</td>
<td>100%</td>
<td>223</td>
<td>0.0</td>
<td>8.52</td>
</tr>
<tr>
<td>48-209</td>
<td>Imp. 1183</td>
<td>40%</td>
<td>120</td>
<td>0.0</td>
<td>5.0</td>
</tr>
<tr>
<td>49-85</td>
<td>C. P. 33-229</td>
<td>none</td>
<td>1024</td>
<td>4.7</td>
<td>5.08</td>
</tr>
<tr>
<td>49-95</td>
<td>C. P. 36-105</td>
<td>low %</td>
<td>493</td>
<td>4.6</td>
<td>9.11</td>
</tr>
<tr>
<td>49-99</td>
<td>C. P. 38-34</td>
<td>20%</td>
<td>687</td>
<td>47.6</td>
<td>50.51</td>
</tr>
<tr>
<td>49-101</td>
<td>C. P. 43-74</td>
<td>80%</td>
<td>521</td>
<td>65.1</td>
<td>65.1</td>
</tr>
<tr>
<td>49-115</td>
<td>F. 36-273</td>
<td>none</td>
<td>130</td>
<td>3.07</td>
<td>3.07</td>
</tr>
<tr>
<td>49-116</td>
<td>C. P. 46-101</td>
<td>none</td>
<td>132</td>
<td>2.27</td>
<td>2.27</td>
</tr>
<tr>
<td>49-117</td>
<td>C. P. 44-156</td>
<td>none</td>
<td>978</td>
<td>27.0</td>
<td>27.7</td>
</tr>
<tr>
<td>49-120</td>
<td>F. 31-762</td>
<td>res.</td>
<td>357</td>
<td>36.69</td>
<td>36.97</td>
</tr>
<tr>
<td>50-80</td>
<td>Cl. 41-142</td>
<td>none</td>
<td>210</td>
<td>40.5</td>
<td>40.9</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: none
Table 22. Mosaic resistance data: Analysis relative to male parent C. P. 38-34.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discs</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-36</td>
<td>C.P. 29-103</td>
<td>trace</td>
<td>506</td>
<td>41.89</td>
<td>43.68</td>
</tr>
<tr>
<td>48-9</td>
<td>C.P. 29-116</td>
<td>trace</td>
<td>362</td>
<td>13.11</td>
<td>16.94</td>
</tr>
<tr>
<td>48-185</td>
<td>C.P. 29-320</td>
<td>5%</td>
<td>174</td>
<td>0.0</td>
<td>3.45</td>
</tr>
<tr>
<td>48-89</td>
<td>C.P. 34-120</td>
<td>res.</td>
<td>1188</td>
<td>0.51</td>
<td>0.89</td>
</tr>
<tr>
<td>48-18</td>
<td>C.P. 36-13</td>
<td>res.</td>
<td>525</td>
<td>3.62</td>
<td>5.71</td>
</tr>
<tr>
<td>48-69</td>
<td>C.P. 43-64</td>
<td>50%</td>
<td>380</td>
<td>0.26</td>
<td>8.68</td>
</tr>
<tr>
<td>48-135</td>
<td>Co. 281</td>
<td>100%</td>
<td>128</td>
<td>13.28</td>
<td>18.75</td>
</tr>
<tr>
<td>48-179</td>
<td>F. 31-962</td>
<td>trace</td>
<td>112</td>
<td>1.79</td>
<td>4.46</td>
</tr>
<tr>
<td>48-111</td>
<td>H. 32-8560</td>
<td>trace</td>
<td>94</td>
<td>3.19</td>
<td>9.57</td>
</tr>
<tr>
<td>48-162</td>
<td>Imp. 1183</td>
<td>40%</td>
<td>129</td>
<td>1.55</td>
<td>5.42</td>
</tr>
<tr>
<td>48-57</td>
<td>P.O.J. 2725</td>
<td>none</td>
<td>371</td>
<td>76.54</td>
<td>77.36</td>
</tr>
<tr>
<td>49-281</td>
<td>C.P. 36-105</td>
<td>low %</td>
<td>688</td>
<td>54.8</td>
<td>60.17</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: 20%
Table 23. Mosaic resistance data: Analysis relative to male parent C. P. 38-41.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-46 C.P. 29-103</td>
<td>trace</td>
<td>352</td>
<td>66.29</td>
<td>67.61</td>
<td></td>
</tr>
<tr>
<td>48-43 C.P. 34-120</td>
<td>res.</td>
<td>546</td>
<td>3.30</td>
<td>5.31</td>
<td></td>
</tr>
<tr>
<td>48-44 C.P. 36-105</td>
<td>low %</td>
<td>552</td>
<td>25.72</td>
<td>27.17</td>
<td></td>
</tr>
<tr>
<td>48-47 P.O.J. 2725</td>
<td>none</td>
<td>426</td>
<td>54.93</td>
<td>54.93</td>
<td></td>
</tr>
<tr>
<td>48-279 C. 28</td>
<td>40%</td>
<td>313</td>
<td>26.5</td>
<td>31.3</td>
<td></td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: none
<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-104</td>
<td>C.P. 29-103</td>
<td>trace</td>
<td>575</td>
<td>2.26</td>
<td>9.04</td>
</tr>
<tr>
<td>48-105</td>
<td>C.P. 34-120</td>
<td>res.</td>
<td>157</td>
<td>0.0</td>
<td>1.27</td>
</tr>
<tr>
<td>48-141</td>
<td>C.P. 36-13</td>
<td>res.</td>
<td>154</td>
<td>0.0</td>
<td>5.84</td>
</tr>
<tr>
<td>48-181</td>
<td>Imp. 1183</td>
<td>40%</td>
<td>154</td>
<td>0.0</td>
<td>3.9</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: trace
Table 25. Mosaic resistance data; Analysis relative to male parent C. P. 42-13.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>50-15</td>
<td>P.O.J. 2775</td>
<td>4%</td>
<td>209</td>
<td>77.5</td>
<td>88.6</td>
</tr>
<tr>
<td>50-4</td>
<td>457-48</td>
<td>none</td>
<td>720</td>
<td>55.2</td>
<td>56.7</td>
</tr>
<tr>
<td>50-17</td>
<td>448-48</td>
<td>none</td>
<td>382</td>
<td>68.1</td>
<td>72.7</td>
</tr>
<tr>
<td>50-65</td>
<td>P.O.J. 2725</td>
<td>none</td>
<td>212</td>
<td>34.9</td>
<td>36.3</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: None
Table 26. Mosaic resistance data: Analysis relative to male parent C. P. 43-74.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-152</td>
<td>C.P. 27-139</td>
<td>none</td>
<td>847</td>
<td>5.9</td>
<td>8.38</td>
</tr>
<tr>
<td>48-98</td>
<td>C.P. 29-103</td>
<td>trace</td>
<td>417</td>
<td>8.15</td>
<td>22.06</td>
</tr>
<tr>
<td>48-172</td>
<td>C.P. 31-511</td>
<td>res.</td>
<td>170</td>
<td>0.0</td>
<td>5.1</td>
</tr>
<tr>
<td>48-106</td>
<td>C.P. 34-120</td>
<td>res.</td>
<td>392</td>
<td>0.0</td>
<td>5.1</td>
</tr>
<tr>
<td>48-107</td>
<td>C.P. 36-13</td>
<td>res.</td>
<td>714</td>
<td>7.42</td>
<td>21.29</td>
</tr>
<tr>
<td>48-110</td>
<td>C.P. 43-64</td>
<td>50%</td>
<td>194</td>
<td>0.0</td>
<td>2.58</td>
</tr>
<tr>
<td>48-164</td>
<td>Co. 281</td>
<td>100%</td>
<td>147</td>
<td>10.88</td>
<td>26.53</td>
</tr>
<tr>
<td>48-166</td>
<td>F. 31-962</td>
<td>trace</td>
<td>278</td>
<td>3.24</td>
<td>8.99</td>
</tr>
<tr>
<td>48-180</td>
<td>Imp. 1183</td>
<td>40%</td>
<td>149</td>
<td>2.0</td>
<td>35.57</td>
</tr>
<tr>
<td>49-28</td>
<td>Cl. 41-142</td>
<td>none</td>
<td>525</td>
<td>4.0</td>
<td>15.4</td>
</tr>
<tr>
<td>49-33</td>
<td>C.P. 36-187</td>
<td>none</td>
<td>434</td>
<td>21.9</td>
<td>29.1</td>
</tr>
<tr>
<td>49-53</td>
<td>C.P. 36-211</td>
<td>none</td>
<td>2134</td>
<td>9.9</td>
<td>11.3</td>
</tr>
<tr>
<td>49-55</td>
<td>C.P. 33-229</td>
<td>none</td>
<td>193</td>
<td>16.6</td>
<td>17.10</td>
</tr>
<tr>
<td>49-62</td>
<td>C.P. 29-116</td>
<td>trace</td>
<td>197</td>
<td>3.6</td>
<td>6.09</td>
</tr>
<tr>
<td>49-66</td>
<td>P.O.J. 2725</td>
<td>none</td>
<td>370</td>
<td>1.3</td>
<td>2.43</td>
</tr>
<tr>
<td>49-70</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>96</td>
<td>7.7</td>
<td>18.75</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: 80%
Table 27. Mosaic resistance data: Analysis relative to male parent C. P. 44-156.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-115</td>
<td>C.P. 27-139</td>
<td>none</td>
<td>169</td>
<td>0.0</td>
<td>1.78</td>
</tr>
<tr>
<td>48-91</td>
<td>C.P. 29-103</td>
<td>trace</td>
<td>282</td>
<td>1.77</td>
<td>7.8</td>
</tr>
<tr>
<td>48-84</td>
<td>C.P. 29-116</td>
<td>trace</td>
<td>236</td>
<td>0.0</td>
<td>1.27</td>
</tr>
<tr>
<td>48-85</td>
<td>C.P. 36-13</td>
<td>res.</td>
<td>212</td>
<td>0.0</td>
<td>0.47</td>
</tr>
<tr>
<td>48-86</td>
<td>C.P. 36-105</td>
<td>low %</td>
<td>158</td>
<td>0.0</td>
<td>6.06</td>
</tr>
<tr>
<td>48-81</td>
<td>C.P. 43-64</td>
<td>50%</td>
<td>174</td>
<td>0.0</td>
<td>5.75</td>
</tr>
<tr>
<td>48-157</td>
<td>F. 31-962</td>
<td>trace</td>
<td>146</td>
<td>0.68</td>
<td>10.27</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: none
Table 28. Mosaic resistance data: Analysis relative to male parent C. P. 48-106.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-100</td>
<td>C.P. 29-116</td>
<td>trace</td>
<td>301</td>
<td>1.99</td>
<td>4.98</td>
</tr>
<tr>
<td>48-99</td>
<td>C.P. 43-64</td>
<td>125</td>
<td>2.4</td>
<td>7.20</td>
<td></td>
</tr>
<tr>
<td>48-102</td>
<td>P.O.J. 2725</td>
<td>none</td>
<td>358</td>
<td>0.11</td>
<td>13.13</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: 20%
Table 29. Mosaic resistance data: Analysis relative to male parent C. P. 1165.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-22</td>
<td>C.P. 36-13</td>
<td>trace</td>
<td>712</td>
<td>10.25</td>
<td>11.10</td>
</tr>
<tr>
<td>48-66</td>
<td>C.P. 43-64</td>
<td>50%</td>
<td>224</td>
<td>0.0</td>
<td>4.51</td>
</tr>
<tr>
<td>48-196</td>
<td>H. 32-8560</td>
<td>trace</td>
<td>96</td>
<td>1.05</td>
<td>4.17</td>
</tr>
<tr>
<td>48-14</td>
<td>P.O.J. 2725</td>
<td>res.</td>
<td>259</td>
<td>20.46</td>
<td>22.01</td>
</tr>
<tr>
<td>49-98</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>133</td>
<td>42.1</td>
<td>42.1</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: apparently immune
Table 30. Mosaic resistance data: Analysis relative to male parent Co. 356.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>React. of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>49-58</td>
<td>F. 36-273</td>
<td>none</td>
<td>182</td>
<td>3.8</td>
<td>3.85</td>
</tr>
<tr>
<td>49-59</td>
<td>C.P. 43-64</td>
<td>50%</td>
<td>467</td>
<td>9.2</td>
<td>13.1</td>
</tr>
<tr>
<td>49-60</td>
<td>C.P. 33-229</td>
<td>none</td>
<td>733</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>49-61</td>
<td>C.P. 29-116</td>
<td>trace</td>
<td>569</td>
<td>5.8</td>
<td>5.80</td>
</tr>
<tr>
<td>49-67</td>
<td>P.O.J.2725</td>
<td>none</td>
<td>1688</td>
<td>3.2</td>
<td>5.2</td>
</tr>
<tr>
<td>49-68</td>
<td>C.P. 44-101</td>
<td>trace</td>
<td>689</td>
<td>17.3</td>
<td>18.1</td>
</tr>
<tr>
<td>49-80</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>690</td>
<td>9.1</td>
<td>14.6</td>
</tr>
<tr>
<td>50-59</td>
<td>C.P. 45-184</td>
<td>none</td>
<td>692</td>
<td>18.5</td>
<td>18.5</td>
</tr>
<tr>
<td>50-77</td>
<td>C.P. 44-155</td>
<td>none</td>
<td>434</td>
<td>22.8</td>
<td>22.8</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: none
Table 31. Mosaic resistance data: Analysis relative to male parent P. 33-37. *

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-198</td>
<td>C.P. 36-13</td>
<td>res.</td>
<td>102</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>49-177</td>
<td>C.P. 38-34</td>
<td>20%</td>
<td>177</td>
<td>69.5</td>
<td>70.62</td>
</tr>
<tr>
<td>49-233</td>
<td>C.P. 47-43</td>
<td>?</td>
<td>243</td>
<td>0.82</td>
<td>0.82</td>
</tr>
<tr>
<td>49-140</td>
<td>C.P. 36-105</td>
<td>low %</td>
<td>673</td>
<td>67.7</td>
<td>69.24</td>
</tr>
<tr>
<td>49-273</td>
<td>B. 35-9</td>
<td>none</td>
<td>151</td>
<td>19.9</td>
<td>20.5</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: none
Table 32. Mosaic resistance data: Analysis relative to male parent U. S. 1694.*

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Female Parent</th>
<th>Reaction of female parent to mosaic</th>
<th>No. plants inoculated</th>
<th>% Mosaic discards</th>
<th>Total % of mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>48-112</td>
<td>C.P. 29-103</td>
<td>trace</td>
<td>105</td>
<td>0.0</td>
<td>1.9</td>
</tr>
<tr>
<td>48-73</td>
<td>C.P. 43-64</td>
<td>50%</td>
<td>183</td>
<td>19.12</td>
<td>19.12</td>
</tr>
<tr>
<td>48-202</td>
<td>F. 31-962</td>
<td>trace</td>
<td>149</td>
<td>0.0</td>
<td>14.76</td>
</tr>
</tbody>
</table>

* Reaction of male parent to mosaic: none
Table 15 shows the results obtained when C. P. 27-108, a mosaic-resistant variety, was used as a male parent in crosses with 11 other varieties. Low percentages of diseased plants were obtained in the progeny of the crosses with the resistant varieties C. P. 38-27, F. 36-819, C. P. 33-224 and F. 31-962. In crosses with other resistant varieties, namely, C. P. 29-103, C. P. 36-13, P.O.J. 2725, C. P. 38-34, and C. P. 34-120, high percentages of diseased plants were obtained. The susceptible varieties C. P. 43-64 and C. P. 38-34 were also crossed with C. P. 27-108. In the progeny, the percentages of diseased plants were 6.6 and 12.5 respectively.

When using, as a male parent, variety C. P. 30-24, a mosaic-resistant variety, results were obtained as shown in table 16. In the crosses with six resistant varieties and with three varieties that show trace of mosaic in the field, the percentages of diseased plants were very low in the progeny of six crosses and moderate in the progeny of the other three crosses. High percentages of diseased plants were obtained in the progeny of the crosses with the resistant variety P.O.J. 2725 and with the susceptible variety C. P. 43-64. In the cross with the other susceptible variety C. P. 38-34, the percentage of diseased plants in the progeny was moderate.

In table 17 are given the results obtained when C. P. 33-224, a mosaic-resistant variety, was crossed with eight different varieties. In the progeny of the crosses with the susceptible varieties C. P. 43-64 and Co. 281 the percentages of diseased plants were 10.2 and 24.4, respectively. More or less similar percentages were obtained when the male parent was crossed with C. P. 34-120 and C. P. 29-103. The first variety is considered a resistant variety and the latter one shows
a trace of mosaic in the field. Considerably lower percentages of
diseased plants were obtained in the crosses with the varieties
C. P. 33-229, C. P. 29-116, and C. P. 36-105, the first variety being
resistant to mosaic, while the second and third varieties show a trace
and a low percentage of mosaic in the field.

Table 18 shows the results obtained when C. P. 33-372, a
mosaic-resistant variety, was used as a male parent in crosses with
five different varieties. Only in one cross was the percentage of
diseased plants in the progeny low. This was in the cross with C. P.
34-120, a resistant variety. Moderate percentages of diseased plants
were obtained in the progeny of the crosses with the resistant varieties
C. P. 36-13 and C. P. 33-229. High percentages of diseased plants
were obtained in the progeny of the crosses with varieties C. P. 29-103
and C. P. 43-64. The first variety shows a trace of mosaic in the field,
while the latter one is susceptible to this disease.

Table 19 shows the results obtained when C. P. 36-105, a
variety that shows a low percentage of mosaic, was crossed with 12
different varieties. The percentages of diseased plants obtained in the
progeny of the crosses with the resistant varieties C. P. 27-139, C. P.
36-13, F. 40-96 and C. P. 42-13, were 0.0, 3.6, 0.6 and 6.5 respectively.
Moderate percentages of diseased plants were obtained in the progeny of
the crosses with the resistant varieties C. P. 34-120 and P.O.J. 2725,
as well as in the progeny of the cross with F. 31-962, a variety that
shows a trace of mosaic in the field. High percentages of diseased
plants were obtained in the progeny of the crosses with varieties C. P.
29-103, C. P. 29-116, and C. P. 28-19. These three varieties show
traces of mosaic in the field. In addition, high percentages of diseased
plants were obtained in the progeny of the crosses with varieties C. P. 43-64 and Cl. 41-142. The first variety is susceptible to mosaic, and the latter is resistant.

The mosaic-susceptible variety C. P. 36-138 was used as a male parent in crosses with six different varieties. The results appear in table 20. When it was crossed with another susceptible variety, C. P. 43-64, the percentage of diseased plants in the progeny was 13.2. Higher percentages of diseased plants were obtained when the same male was crossed with the resistant varieties C. P. 34-120, C. P. 36-13. The percentages of diseased plants in the progeny of these crosses were 23.5 and 21.1. Similar percentages of mosaic were also obtained in the progeny of the crosses with varieties C. P. 29-116 and F. 31-962. These two varieties show a trace of mosaic in the field. The percentages of diseased plants in the progeny of these crosses were 23.96 and 24.0.

Table 21 shows the results obtained when C. P. 36-211, mosaic-resistant, was used in crosses with 13 different varieties. Low percentages of diseased plants were obtained in the progeny of five crosses. The female parent in four of these crosses was resistant to mosaic, and the fifth female parent was susceptible to this disease. Moderate percentages of diseased plants were obtained in the progeny of two crosses only. The female parents in these crosses were Co. 281 and C. P. 36-105. The first variety is susceptible to mosaic, while the latter one shows a low percentage of mosaic in the field. High percentages of diseased plants were obtained in the progeny of the crosses with the resistant varieties C. P. 44-156, F. 31-762 and Cl. 41-142, as well as in the progeny with the susceptible varieties C. P. 43-64, C. P. 38-34 and C. P. 43-74.
The mosaic-susceptible variety C. P. 38-34 was used in crosses with 12 different varieties. The results are given in table 22. It was crossed with the susceptible varieties C. P. 43-64, Co. 281 and Imp. 1183. In the progeny the percentages of diseased plants were 8.7, 18.8 and 5.4 respectively. On the other hand, when it was crossed with the resistant variety P.O.J. 2725, the percentage of plants showing mosaic symptoms in the progeny was 77.4. High percentages of diseased plants were also obtained when the same male C. P. 38-34, was crossed with C. P. 29-103 and C. P. 36-105. Low percentages of diseased plants were obtained in the progeny of the crosses with the resistant varieties C. P. 34-120, C. P. 36-13 and variety F. 31-962. The last variety shows a trace of mosaic in the field.

Table 23 shows the results obtained when C. P. 38-41, a mosaic-resistant variety, was crossed with five different varieties. It is observed that the percentages of diseased plants were high in the progeny of four crosses. Only in the cross with variety C. P. 34-120 was there a low percentage of diseased plants in the progeny.

In table 24 is given the results obtained when C. P. 42-10, a variety that shows a trace of mosaic in the field, was crossed with four varieties that differ in their degree of resistance to mosaic. In the progeny of three of these crosses the percentages of diseased plants were low. A moderate percentage (9.0) of diseased plants was obtained in the progeny with variety C. P. 29-103.

The mosaic-resistant variety C. P. 42-13 was used as a male parent in crosses with three other resistant varieties and with P.O.J. 2775. The latter variety shows four per cent of mosaic in the field. The results given in the table show that in the progeny of these four
crosses, the percentages of plants showing mosaic symptoms were high.

The mosaic-susceptible variety C. P. 43-74 was used as a male parent in 16 crosses. Table 26 shows that low percentages of diseased plants were obtained in the progeny of three crosses only. The female parents in these crosses were the resistant varieties C. P. 34-120 and P.O.J. 2725, and the susceptible variety C. P. 43-64. Moderate percentages of diseased plants were obtained in the progeny of ten crosses. Only one female parent in these crosses was a susceptible variety (C. P. 38-34), while six of the other female parents were resistant and three of them were varieties that showed a trace of mosaic in the field. High percentages of diseased plants were obtained in the progeny of these three crosses. The female parents in these crosses were the susceptible varieties C. P. 281 and Imp. 1183, as well as the resistant variety C. P. 36-187. It is observed also in table 26, that C. P. 43-74 was crossed with four mosaic-susceptible varieties. In the progeny of these crosses the percentages of diseased plants were 2.6, 18.8, 26.5 and 35.6.

Table 27 shows the results obtained when C. P. 44-156, a mosaic-resistant variety, was used as a male parent in seven crosses. In the progeny of four crosses the percentages of diseased plants were low, while in the progeny of the three other crosses moderate percentages of diseased plants were obtained. However, the percentage of diseased plants in any cross did not exceed 10 per cent.

The mosaic-susceptible variety C. P. 48-106 was used as a male parent in three crosses. The results given in table 28 show that when it was crossed with another susceptible variety (C. P. 43-64) the percentage of diseased plants in the progeny was 7.2, while a slightly
higher percentage of diseased plants (13.3) was obtained in the progeny of the cross with the resistant variety P.O.J. 2725. A low percentage of diseased plants was obtained in the progeny of the cross with C. P. 48-106, a variety that shows a trace of mosaic in the field.

Table 29 shows the results obtained when C. P. 1165, a mosaic-resistant variety, was used as a male parent in five crosses. When it was crossed with the susceptible varieties C. P. 43-64 and C. P. 38-34, a low percentage (4.5) of diseased plants was obtained in the progeny of the cross with the first variety, while a high percentage (4.2) of diseased plants was obtained in the progeny of the cross with the second variety. When it was crossed also with C. P. 36-13 and H. 32-8560, both being varieties that show traces of mosaic in the field, a moderate percentage (11.1) of diseased plants was obtained in the progeny of the cross with the former variety, while a low percentage (4.2) was obtained in the progeny of the cross with the latter variety. When the same male, C. P. 1165, was crossed with the resistant variety P.O.J. 2725, the percentage of plants showing mosaic symptoms in the progeny was 22.0.

In Table 30, is given the results obtained when Co. 356, a mosaic-resistant variety was used as a male parent in nine crosses. Low percentages of diseased plants were obtained in the progeny of four crosses. Moderate percentages of diseased plants were obtained in the progeny of the crosses with the susceptible varieties C. P. 43-64 and C. P. 38-34, as well as in the progeny of the crosses with the resistant varieties C. P. 45-184, C. P. 44-155 and C. P. 44-101.

The mosaic-resistant variety P. 33-37 was used as a male parent in five crosses. The results appearing in table 31 indicate that
no plants showed mosaic symptoms in the progeny of the cross with
the resistant variety C. P. 36-13. A very low percentage of diseased
plants was also obtained in the progeny of the cross with variety C. P.
47-43. In the progeny of the cross with the resistant variety B. 35-9,
20.5 per cent of the plants showed mosaic symptoms. High percent-
ages of mosaic were obtained in the progeny of the crosses with the
susceptible variety C. P. 38-34 and in the progeny of the crosses with
variety C. P. 36-105. The latter variety shows a low percentage of
mosaic in the field.

Table 32 shows the results obtained when U. S. 1694, a mosaic-
resistant variety was used as a male parent in three crosses. A low
percentage (1.9) of diseased plants was obtained in the progeny of the
crosses with the susceptible variety C. P. 43-64 and in the progeny of
the crosses with F. 31-962, a variety that shows a trace of mosaic in
the field.

The following table shows the results obtained in certain
crosses not listed in the preceding tables. The varieties involved
were used once or twice only in the crosses and so they were grouped
together in the following table which is self-explanatory.
### Table 33. Mosaic resistance data. Analysis relative to crosses with parents as indicated.

<table>
<thead>
<tr>
<th>Cross No.</th>
<th>Parents</th>
<th>No. plants inoculated</th>
<th>% mosaic discards</th>
<th>Total % mosaic</th>
</tr>
</thead>
<tbody>
<tr>
<td>49-246</td>
<td>C. 28² X P. 33-32³</td>
<td>839</td>
<td>66.86</td>
<td>71.27</td>
</tr>
<tr>
<td>49-250</td>
<td>Cl. 41-223⁴ X P. 33-32³</td>
<td>1975</td>
<td>49.67</td>
<td>52.96</td>
</tr>
<tr>
<td>50-19</td>
<td>C.P. 44-55³ X C.P. 47-68⁴</td>
<td>156</td>
<td>21.79</td>
<td>24.35</td>
</tr>
<tr>
<td>50-84</td>
<td>C.P. 44-155⁴ X C.P. 48-156⁴</td>
<td>146</td>
<td>24.6</td>
<td>27.3</td>
</tr>
</tbody>
</table>

1 Shows trace of mosaic
2 Shows 40% mosaic
3 Reaction to mosaic unknown
4 Immune to mosaic
The results obtained in comparing two inoculation techniques:
(1) pot inoculation and (2) flat inoculation, are given in tables 34 through 37.
Table 34. Comparison of two inoculation techniques of mosaic in sugarcane seedlings.

<table>
<thead>
<tr>
<th>Cross</th>
<th>Flat Inoculation</th>
<th>Pot Inoculation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. plants</td>
<td>% Mos.</td>
</tr>
<tr>
<td>Cl. 41-142 X C.P. 43-74</td>
<td>528</td>
<td>76.7</td>
</tr>
<tr>
<td>C.P. 42-13 X C.P. 30-24</td>
<td>527</td>
<td>66.8</td>
</tr>
<tr>
<td>C.P. 29-116 X C.P. 46-73</td>
<td>313</td>
<td>54.9</td>
</tr>
<tr>
<td>C.P. 36-211 X C.P. 43-74</td>
<td>837</td>
<td>64.3</td>
</tr>
<tr>
<td>C.P. 42-13 X F. 36-273</td>
<td>341</td>
<td>51.3</td>
</tr>
<tr>
<td>C.P. 42-13 X C.P. 36-105</td>
<td>187</td>
<td>43.3</td>
</tr>
<tr>
<td>C.P. 43-64 X Co. 356</td>
<td>437</td>
<td>50.3</td>
</tr>
</tbody>
</table>

1 Percentage of diseased plants discarded in the greenhouse.
2 Total percentage of plants showing mosaic symptoms (field and greenhouse).
Table 35. Comparison of two inoculation techniques of mosaic in sugarcane seedlings.

<table>
<thead>
<tr>
<th>Cross</th>
<th>Flat inoculation</th>
<th></th>
<th>Pot inoculation</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. plants</td>
<td>% Mos. 1</td>
<td>Total % mosaic</td>
<td>No. plants</td>
</tr>
<tr>
<td>C.P. 29-116 X C.P. 46-199</td>
<td>345</td>
<td>19.7</td>
<td>20.0</td>
<td>519</td>
</tr>
<tr>
<td>C.P. 33-229 X Co. 356</td>
<td>948</td>
<td>19.2</td>
<td>19.2</td>
<td>733</td>
</tr>
<tr>
<td>C.P. 33-224 X C.P. 27-108</td>
<td>380</td>
<td>23.2</td>
<td>23.4</td>
<td>464</td>
</tr>
<tr>
<td>C.P. 36-187 X C.P. 43-74</td>
<td>479</td>
<td>37.4</td>
<td>40.1</td>
<td>434</td>
</tr>
<tr>
<td>C.P. 42-13 X C.P. 44-101</td>
<td>216</td>
<td>50.0</td>
<td>---</td>
<td>189</td>
</tr>
</tbody>
</table>

1 Percentage of diseased plants discarded in the greenhouse.

2 Total percentage of plants showing mosaic symptoms (Field and greenhouse).
Table 36. Comparison of two inoculation techniques on occurrence of mosaic in sugarcane seedlings.

<table>
<thead>
<tr>
<th>Cross</th>
<th>Flat Inoculation</th>
<th>Pot Inoculation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. plants % Mos. discards Total % mosaic</td>
<td>No. plants % Mos. discards Total % mosaic</td>
</tr>
<tr>
<td>C.P. 38-34 X Co. 356</td>
<td>533 18.9 21.9</td>
<td>690 9.1 14.6</td>
</tr>
<tr>
<td>C.P. 34-120 X C.P. 30-24</td>
<td>195 7.7 7.7</td>
<td>244 0.4 1.6</td>
</tr>
<tr>
<td>P.C.T. 2725 X Co. 356</td>
<td>1716 6.5 7.3</td>
<td>1688 3.2 5.3</td>
</tr>
<tr>
<td>C.P. 44-126 X C.P. 30-24</td>
<td>146 2.7 2.7</td>
<td>309 0.0 0.0</td>
</tr>
<tr>
<td>C.P. 44-101 X Co. 356</td>
<td>314 18.8 18.8</td>
<td>689 17.2 18.2</td>
</tr>
<tr>
<td>C.P. 46-193 X C.P. 44-154</td>
<td>599 13.4 18.2</td>
<td>908 21.1 25.7</td>
</tr>
</tbody>
</table>

1 Percentage of diseased plants discarded in the greenhouse.

2 Total percentage of plants showing mosaic symptoms (Field and greenhouse).
Table 34 shows the results obtained from the studies made on the progeny of seven crosses. In all cases, when the flat inoculation technique was used a much higher percentage of plants showing mosaic symptoms was found than in those in which the pot inoculation technique was used. For example, in the progeny of the cross Cl. 41-142 x C. P. 43-4, the percentage of plants showing mosaic symptoms, when inoculated in the flat, was 77.7, while the percentage of diseased plants, when inoculated in the pots, was 15.4, a difference of 62.3 per cent. In the progeny of the cross C. P. 42-13 x C. P. 30-24, 66.8 per cent of the plants examined showed mosaic symptoms when the flat technique was used. When the pot inoculation technique was used, only 6.4 per cent of the plants inoculated showed symptoms of the disease, a difference of 60.4 per cent. In all the differences between the percentage of plants showing mosaic symptoms by using these two inoculation techniques ranged from 62.3 per cent in one cross to 37.2 per cent in another cross.

Table 35 shows that when the flat inoculation technique was used there was always a higher percentage of plants showing mosaic symptom than when the pot inoculation technique was used. In the progeny of the cross C. P. 29-116 x C. P. 46-199, the percentage of plants showing mosaic symptoms when inoculated in the flats, was 20.0 while the percentage of diseased plants, when inoculated in the pots was 1.9, a difference of 18.1 per cent. In the progeny of the five crosses studied, the differences between the percentages of plants showing mosaic symptoms by using these two inoculation techniques ranged from 18.1 per cent in one cross to 10.9 per cent in another cross.

Table 36 shows the results obtained from the studies made on the progeny of six crosses. In four crosses, a higher percentage of
plants showing mosaic symptoms was found than in those when the pot inoculation technique was used. The differences ranged from 7.3 per cent in one cross to 2.7 in another.

In the progeny of the cross C.P. 44-101 x Co. 356, 18.8 per cent of the plants showed mosaic symptoms when the flat inoculation technique was used. When the pot inoculation technique was used, 18.2 per cent of the plants inoculated showed mosaic symptoms, a difference of only 0.6 per cent.

In one case only, a higher percentage of plants showing mosaic symptoms was obtained when the pot inoculation technique was used, than when the flat inoculation technique was used. This was in the progeny of the cross C. P. 46-193 x C. P. 44-154. The percentage of diseased plants was 18.8 when the seedlings were inoculated in the flats. On the other hand, when the pot inoculation technique was used, 25.7 per cent of the plants showed mosaic symptoms, a difference of 6.9.

Throughout the experiments reported on the preceding pages, whenever there was a large number of true sugarcane seeds obtained from a certain cross, the seeds were planted in more than one flat. In practically every instance the results obtained in duplicate flats were similar. In a single instance, for unknown reasons, the results differed. These results are given in a separate table (Table 37).

Table 37 shows that when the progeny of the cross C.P. 36-105 x 28NG39-491, were inoculated by using the flat inoculation technique, 42.1 per cent of the seedlings showed mosaic symptoms. When the pot inoculation technique was used the seeds were planted in two flats, and later transplanted to pots where they were inoculated. The seedlings that were obtained from one flat showed 18.9 per cent mosaic symptoms, while
86.6 per cent of the seedlings obtained from the other flat showed symptoms of this disease.
### Table 37. Comparison of two inoculation techniques of mosaic virus in the progeny of the cross C. P. 36-105 X 28NG39-491.

<table>
<thead>
<tr>
<th>Flat No.</th>
<th>Flat Inoculation</th>
<th>Pot Inoculation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. seedlings inoculated</td>
<td>% Mosaic discards</td>
</tr>
<tr>
<td>1</td>
<td>1620</td>
<td>41.2</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
The results of the studies on the recovery from sugarcane mosaic symptoms in the sugarcane seedlings are given in tables 38 through 41.
Table 38. Recovery from mosaic symptoms in sugarcane seedlings grown in the field.

<table>
<thead>
<tr>
<th>Cross</th>
<th>No. seedlings examined</th>
<th>Examined between July 4-14 % seedlings recovered</th>
<th>Examined between August 24-28 % seedlings recovered</th>
</tr>
</thead>
<tbody>
<tr>
<td>457-48 X C.P. 42-13</td>
<td>398</td>
<td>4.3</td>
<td>23.6</td>
</tr>
<tr>
<td>P.O.J. 2775 X C.P. 42-13</td>
<td>162</td>
<td>0.0</td>
<td>1.2</td>
</tr>
<tr>
<td>448-48 X C.P. 42-13</td>
<td>260</td>
<td>0.4</td>
<td>18.8</td>
</tr>
<tr>
<td>P.O.J. 2725 X C.P. 42-13</td>
<td>74</td>
<td>1.4</td>
<td>12.2</td>
</tr>
<tr>
<td>C.P. 44-155 X C. 356</td>
<td>99</td>
<td>8.1</td>
<td>8.1</td>
</tr>
<tr>
<td>Cl. 41-142 X C.P. 36-211</td>
<td>85</td>
<td>0.0</td>
<td>6.0</td>
</tr>
</tbody>
</table>

1 Seedlings that have both recovered and diseased stalks

2 Seedlings that did not show any mosaic symptoms
Table 39. Recovery from mosaic symptoms in sugarcane seedlings grown in the field.

<table>
<thead>
<tr>
<th>Cross</th>
<th>No. seedlings examined</th>
<th>Examine between July 4 - 14</th>
<th>% seedlings recovered</th>
<th>Examine between August 24 - 28</th>
<th>% seedlings recovered</th>
<th>% seedlings recovered</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.P. 44-55 x C.P. 47-68</td>
<td>34</td>
<td>0.0</td>
<td>11.8</td>
<td>8.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 34-120 x C.P. 30-24</td>
<td>46</td>
<td>8.7</td>
<td>17.4</td>
<td>28.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P.O. 2725 x C.P. 48-53</td>
<td>27</td>
<td>25.9</td>
<td>18.5</td>
<td>51.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 44-155 x C.P. 48-156</td>
<td>36</td>
<td>0.0</td>
<td>2.7</td>
<td>0.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 38-34 x C.P. 36-211</td>
<td>33</td>
<td>12.1</td>
<td>18.2</td>
<td>24.2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 40. Recovery from mosaic symptoms in sugarcane seedlings grown in the field.

<table>
<thead>
<tr>
<th>Cross</th>
<th>No. seedlings examined</th>
<th>Examined between July 4 - 14</th>
<th>No. seedlings recovered</th>
<th>Examined between August 24 - 28</th>
<th>No. seedlings partially recovered</th>
<th>No. seedlings recovered</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.P. 44-126 x C.P. 30-24</td>
<td>9</td>
<td>3</td>
<td>0</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 48-153 x do</td>
<td>13</td>
<td>5</td>
<td>2</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 48-184 x Co. 356</td>
<td>17</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. 48-120 x C.P. 46-115</td>
<td>6</td>
<td>2</td>
<td>0</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 48-153 x C.P. 47-146</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 44-55 x Cl. 46-51</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 46-160 x C.P. 47-6</td>
<td>16</td>
<td>0</td>
<td>0</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>do x C.P. 44-75</td>
<td>15</td>
<td>0</td>
<td>2</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 49-21 x C.P. 30-24</td>
<td>12</td>
<td>0</td>
<td>1</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 48-120 x do</td>
<td>10</td>
<td>2</td>
<td>0</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 46-50 x C.P. 42-13</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 44-120 x C.P. 46-115</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 48-10 x C.P. 42-13</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 44-155 x C.P. 48-53</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>do x C.P. 30-24</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 47-161 x do</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 44-155 x C.P. 36-211</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 48-45 x C.P. 30-24</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 36-105 x do</td>
<td>7</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P.C.J. 2725 x do</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 29-103 x C.P. 48-53</td>
<td>7</td>
<td>1</td>
<td>0</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Red Prenger x C. do</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 48-86 x C.P. 30-24</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 40. Cont’d.

<table>
<thead>
<tr>
<th>Cross</th>
<th>No. seedlings examined</th>
<th>Examined between July 4 - 14</th>
<th>No. seedlings recovered</th>
<th>Examined between August 24 - 28</th>
<th>No. seedlings partially recovered</th>
<th>No. seedlings recovered</th>
</tr>
</thead>
<tbody>
<tr>
<td>CL 41-142 x C.P. 47-156</td>
<td>5</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 45-197 x Co. 356</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>449-48 x C.P. 36-211</td>
<td>8</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>449-48 x C.P. 42-13</td>
<td>8</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 48-36 x C.P. 36-211</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 44-101 x C.P. 48-156</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 49-47 x C.P. 27-108</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CL 41-14 x C.P. 48-156</td>
<td>18</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>481-48 x C.P. 36-211</td>
<td>9</td>
<td>3</td>
<td>0</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>285-48 x do</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CL 29-115 x Co. 356</td>
<td>4</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 29-103 x do</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.P. 38-34 x C.P. 47-127</td>
<td>4</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>132-48 x C.P. 36-211</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>256-48 x do</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CL 38-34 x C.P. 42-13</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CL 41-142 x C.P. 33-372</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>216</strong></td>
<td><strong>23</strong></td>
<td><strong>15</strong></td>
<td><strong>85</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Percentage</strong></td>
<td><strong>10.6</strong></td>
<td><strong>6.9</strong></td>
<td><strong>39.4</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Table 41. Summation of the results of studies on recovery from mosaic symptoms in sugarcane seedlings grown in the field.

<table>
<thead>
<tr>
<th>Date of examination</th>
<th>No. of seedlings examined</th>
<th>% of partially recovered seedlings</th>
<th>% of recovered seedlings</th>
</tr>
</thead>
<tbody>
<tr>
<td>July 4 - 14</td>
<td>1470</td>
<td>not recorded</td>
<td>4.4</td>
</tr>
<tr>
<td>August 26 - 29</td>
<td>1470</td>
<td>14.0</td>
<td>19.0</td>
</tr>
</tbody>
</table>

1. Seedlings that have both recovered and diseased stalks.

2. Seedlings that did not show any mosaic symptoms.
Table 38 shows that at the time of the first examination, made between July 4 to 14, 1951, many seedlings had recovered from mosaic symptoms. In the progeny of three of the crosses examined, the percentages of recovered seedlings were 0.4, 4.3, and 3.1. On the other hand, the seedlings of the other three crosses did not show any recovery.

A seedling was considered as recovered in these studies when all the leaves of all the stalks did not show any mosaic symptoms. It was also noticed that in other seedlings, some of the stalks within a single seedling showed mosaic symptoms and the others did not. Such seedlings were called "partially recovered". In the second examination, made between August 24 to August 28, 1951, the number of partially recovered seedlings were recorded. It is noticed that in all the crosses recorded in table 38 there were seedlings that showed this phenomenon.

In table 38, it may be noted that the number of recovered seedlings increased between July 4th and August 24th. This increase was observed in the progeny of two of the three crosses that showed recovery during July. In addition, many of the seedlings of two of the three crosses that did not show recovery during July, showed recovery from mosaic symptoms during August. The percentage of recovered seedlings per cross varied from as high as 22.4 to as low as 3.1.

In table 39, the results are given on recovery from mosaic symptoms in the progeny of five crosses. It is to be noted that the number of seedlings studied per cross was comparatively low. In the 1950 breeding season, the weather conditions were not favorable, and the number of seeds obtained per cross was comparatively low. The percentage of seed germination was low also.

It is observed in table 39, that the seedlings of two out of the five
crosses studied did not show any recovery during July. The percentage of these seedlings varied from 8.8 to 51.6 per cross. In addition, in all the crosses there were seedlings that showed partial recovery.

There were many crosses in which the number of seedlings studied was less than 20 per cross. These are listed in table 40, and the results of the recovery studies are given there.

The summation of the results on the apparent recovery from mosaic symptoms are given in table 41. There were 1470 seedlings showing mosaic symptoms planted in the field in April 1951. In the period between July 4 and July 14, 1951, 4.4 per cent of these seedlings showed apparent recovery from the mosaic symptoms. In the period between August 24 and August 28, 1951, 19.0 per cent of the seedlings showed recovery from the symptoms while 14.0 per cent of the seedlings showed partial recovery.
The results obtained in the studies of inheritance of resistance to red rot disease of sugarcane are given in tables 42 through 52.

**Table 42. Results obtained in studies of the reaction of progeny to red rot.**

**Cross 48-25**  
Parents C.P. 29*116 x C.P. 36-105

<table>
<thead>
<tr>
<th>Inoculation date</th>
<th>Examination date</th>
<th>Res.</th>
<th>Mod. Res.</th>
<th>Mod. Susc.</th>
<th>Susc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>11-9-50</td>
<td>2-15-51</td>
<td>6</td>
<td>7</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>11-10-50</td>
<td>2-22-51</td>
<td>13</td>
<td>12</td>
<td>18</td>
<td>16</td>
</tr>
<tr>
<td>11-11-50</td>
<td>2-22-51</td>
<td>4</td>
<td>8</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>11-14-50</td>
<td>2-26-51</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td><strong>Total No. of plants</strong></td>
<td></td>
<td>23</td>
<td>27</td>
<td>28</td>
<td>32</td>
</tr>
<tr>
<td><strong>Percentage</strong></td>
<td></td>
<td>20.9</td>
<td>24.5</td>
<td>25.5</td>
<td>29.1</td>
</tr>
</tbody>
</table>

**Table 43. Results obtained in studies on the reaction of progeny to red rot.**

**Cross 48-8**  
Parents C.P. 36-13 x C.P. 36-105

<table>
<thead>
<tr>
<th>Inoculation date</th>
<th>Examination date</th>
<th>Res.</th>
<th>Mod. Res.</th>
<th>Mod. Susc.</th>
<th>Susc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>11-14-50</td>
<td>2-23-51</td>
<td>1</td>
<td>3</td>
<td>4</td>
<td>14</td>
</tr>
<tr>
<td>11-14-50</td>
<td>2-26-51</td>
<td>1</td>
<td>2</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>11-17-50</td>
<td>3-2-51</td>
<td>9</td>
<td>11</td>
<td>12</td>
<td>28</td>
</tr>
<tr>
<td><strong>Total No. of plants</strong></td>
<td></td>
<td>11</td>
<td>16</td>
<td>25</td>
<td>51 103</td>
</tr>
<tr>
<td><strong>Percentage</strong></td>
<td></td>
<td>10.7</td>
<td>15.5</td>
<td>24.3</td>
<td>49.5</td>
</tr>
</tbody>
</table>
Table 44. Results obtained in studies of the reaction of progeny to red rot.

**Cross 48-48**
**Parents C.P. 29-103 x C.P. 36-105**

<table>
<thead>
<tr>
<th>Inoculation date</th>
<th>Examination date</th>
<th>Res.</th>
<th>Mod. Res.</th>
<th>Mod. Susc.</th>
<th>Susc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>11-9-50</td>
<td>2-15-51</td>
<td>10</td>
<td>14</td>
<td>15</td>
<td>16</td>
</tr>
<tr>
<td>11-10-50</td>
<td>2-22-51</td>
<td>4</td>
<td>1</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total No. of plants</strong></td>
<td></td>
<td>14</td>
<td>15</td>
<td>18</td>
<td>16 63</td>
</tr>
<tr>
<td><strong>Percentage</strong></td>
<td></td>
<td>22.2</td>
<td>23.8</td>
<td>28.6</td>
<td>25.4</td>
</tr>
</tbody>
</table>

Table 45. Results obtained in studies of the reaction of progeny to red rot.

**Cross 48-130**
**Parents F. 31-962 x C.P. 36-105**

<table>
<thead>
<tr>
<th>Inoculation date</th>
<th>Examination date</th>
<th>Res.</th>
<th>Mod. Res.</th>
<th>Mod. Susc.</th>
<th>Susc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>11-14-50</td>
<td>3-2-51</td>
<td>20</td>
<td>9</td>
<td>17</td>
<td>13</td>
</tr>
<tr>
<td>11-16-50</td>
<td>3-2-51</td>
<td>27</td>
<td>16</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td><strong>Total No. of plants</strong></td>
<td></td>
<td>47</td>
<td>25</td>
<td>22</td>
<td>23</td>
</tr>
<tr>
<td><strong>Percentage</strong></td>
<td></td>
<td>40.2</td>
<td>21.4</td>
<td>18.8</td>
<td>19.6</td>
</tr>
</tbody>
</table>

Table 46. Results obtained in studies of the reaction of progeny to red rot.

**Cross 48-10**
**Parents C.P. 34-120 x C.P. 27-108**

<table>
<thead>
<tr>
<th>Inoculation date</th>
<th>Examination date</th>
<th>Res.</th>
<th>Mod. Res.</th>
<th>Mod. Susc.</th>
<th>Susc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>11-8-50</td>
<td>2-27-51</td>
<td>13</td>
<td>14</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>11-8-50</td>
<td>3-5-51</td>
<td>10</td>
<td>13</td>
<td>10</td>
<td>13</td>
</tr>
<tr>
<td>11-9-50</td>
<td>2-15-51</td>
<td>3</td>
<td>4</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td><strong>Total No. of plants</strong></td>
<td></td>
<td>26</td>
<td>31</td>
<td>26</td>
<td>27</td>
</tr>
<tr>
<td><strong>Percentage</strong></td>
<td></td>
<td>23.6</td>
<td>28.2</td>
<td>23.6</td>
<td>24.6</td>
</tr>
</tbody>
</table>
Table 47. Results obtained in studies of the reaction of progeny to red rot.

**Cross 48-23**
Parents C.P. 34-120 x C.P. 36-105

<table>
<thead>
<tr>
<th>Inoculation date</th>
<th>Examination date</th>
<th>Res.</th>
<th>Mod. Res.</th>
<th>Mod. Susc.</th>
<th>Susc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>11-9-50</td>
<td>2-16-51</td>
<td>9</td>
<td>13</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>11-10-50</td>
<td>2-23-51</td>
<td>18</td>
<td>7</td>
<td>14</td>
<td>9</td>
</tr>
<tr>
<td>11-13-50</td>
<td>2-27-51</td>
<td>6</td>
<td>8</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td><strong>Total No. of plants</strong></td>
<td></td>
<td>33</td>
<td>28</td>
<td>22</td>
<td>16</td>
</tr>
<tr>
<td><strong>Percentage</strong></td>
<td></td>
<td>33.3</td>
<td>28.3</td>
<td>22.2</td>
<td>16.2</td>
</tr>
</tbody>
</table>

Table 48. Results obtained in studies of the reaction of progeny to red rot.

**Cross 48-104**
Parents C.P. 29-103 x C.P. 42-10

<table>
<thead>
<tr>
<th>Inoculation date</th>
<th>Examination date</th>
<th>Res.</th>
<th>Mod. Res.</th>
<th>Mod. Susc.</th>
<th>Susc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>11-6-51</td>
<td>3-5-51</td>
<td>4</td>
<td>7</td>
<td>12</td>
<td>74</td>
</tr>
<tr>
<td><strong>Percentage</strong></td>
<td></td>
<td>4.1</td>
<td>7.2</td>
<td>12.4</td>
<td>76.3</td>
</tr>
</tbody>
</table>

Table 49. Results obtained in studies of the reaction of progeny to red rot.

**Cross 48-70**
Parents C.P. 29-103 x C.P. 27-108

<table>
<thead>
<tr>
<th>Inoculation date</th>
<th>Examination date</th>
<th>Res.</th>
<th>Mod. Res.</th>
<th>Mod. Susc.</th>
<th>Susc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>11-14-50</td>
<td>2-26-51</td>
<td>18</td>
<td>8</td>
<td>9</td>
<td>2</td>
</tr>
<tr>
<td>11-14-50</td>
<td>3-11-51</td>
<td>32</td>
<td>17</td>
<td>16</td>
<td>14</td>
</tr>
<tr>
<td><strong>Total number of plants</strong></td>
<td></td>
<td>50</td>
<td>25</td>
<td>25</td>
<td>16</td>
</tr>
<tr>
<td><strong>Percentage</strong></td>
<td></td>
<td>43.1</td>
<td>21.6</td>
<td>21.6</td>
<td>13.7</td>
</tr>
</tbody>
</table>
Table 50. Results obtained in studies of the reaction of progeny to red rot.

**Cross 48-188**

**Parents F. 31-962 x C.P. 27-108**

<table>
<thead>
<tr>
<th>Inoculation Date</th>
<th>Examination Date</th>
<th>Res.</th>
<th>Mod. Res.</th>
<th>Mod. Susc.</th>
<th>Susc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>11-14-50</td>
<td>2-26-51</td>
<td>7</td>
<td>11</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>11-14-50</td>
<td>3-1-51</td>
<td>6</td>
<td>6</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td><strong>Total No: of plants</strong></td>
<td></td>
<td>13</td>
<td>17</td>
<td>14</td>
<td>20 64</td>
</tr>
<tr>
<td><strong>Percentage</strong></td>
<td></td>
<td>20.3</td>
<td>26.6</td>
<td>21.9</td>
<td>31.2</td>
</tr>
</tbody>
</table>
Table 51. Summation of the results of red rot studies on the progeny of the 1948 crosses.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>C.P. 29-116(R.) x C.P. 36-105 (R.)</td>
<td>23</td>
<td>20.9</td>
<td>27</td>
<td>24.5</td>
</tr>
<tr>
<td>C.P. 36-13(R.) x C.P. 36-105(R.)</td>
<td>11</td>
<td>10.7</td>
<td>16</td>
<td>15.5</td>
</tr>
<tr>
<td>F. 31-962(R.) x C.P. 36-105(R.)</td>
<td>47</td>
<td>40.2</td>
<td>25</td>
<td>21.4</td>
</tr>
<tr>
<td>C.P. 29-103(M.R.) x C.P. 36-105(R.)</td>
<td>14</td>
<td>22.2</td>
<td>15</td>
<td>23.8</td>
</tr>
<tr>
<td>C.P. 34-120(S.) x C.P. 36-105(R.)</td>
<td>33</td>
<td>33.3</td>
<td>28</td>
<td>28.3</td>
</tr>
<tr>
<td>C.P. 29-103(M.R.) x C.P. 42-10(S.)</td>
<td>4</td>
<td>4.1</td>
<td>7</td>
<td>7.2</td>
</tr>
<tr>
<td>C.P. 29-103(M.R.) x C.P. 27-108(M.S.)</td>
<td>50</td>
<td>43.1</td>
<td>25</td>
<td>21.6</td>
</tr>
<tr>
<td>F. 31-962(R.) x C.P. 27-108(M.S.)</td>
<td>13</td>
<td>20.3</td>
<td>17</td>
<td>26.6</td>
</tr>
<tr>
<td>C. 34-102(S.) x C.P. 27-108(M.S.)</td>
<td>26</td>
<td>23.6</td>
<td>31</td>
<td>28.2</td>
</tr>
</tbody>
</table>
Table 52. Results obtained in studies of the reaction of progeny of the 1949 crosses to red rot.

<table>
<thead>
<tr>
<th>Cross</th>
<th>R. No. plants</th>
<th>R. %</th>
<th>Mod. Res. No. plants</th>
<th>Mod. Res. %</th>
<th>Mod. Susc. No. plants</th>
<th>Mod. Susc. %</th>
<th>Susc. No. plants</th>
<th>Susc. %</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.P. 46-193(R.)² x C.P. 44-154(M.R.)</td>
<td>32</td>
<td>31.4</td>
<td>33</td>
<td>21.6</td>
<td>15</td>
<td>14.7</td>
<td>33</td>
<td>32.4</td>
</tr>
<tr>
<td>C.P. 38-34(S.) x Co. 356(S.)</td>
<td>29</td>
<td>22.8</td>
<td>17</td>
<td>13.4</td>
<td>21</td>
<td>16.5</td>
<td>60</td>
<td>47.2</td>
</tr>
<tr>
<td>C.P. 38-34(S.) x C.P. 36-138(S.)</td>
<td>28</td>
<td>26.4</td>
<td>17</td>
<td>16.0</td>
<td>20</td>
<td>18.9</td>
<td>41</td>
<td>38.7</td>
</tr>
<tr>
<td>C.P. 43-64(R.) x Co. 356(S.)</td>
<td>28</td>
<td>23.9</td>
<td>21</td>
<td>17.9</td>
<td>21</td>
<td>17.9</td>
<td>47</td>
<td>40.2</td>
</tr>
<tr>
<td>C.P. 43-64(R.) x C.P. 33-372(S.)</td>
<td>38</td>
<td>33.6</td>
<td>18</td>
<td>15.9</td>
<td>21</td>
<td>18.6</td>
<td>36</td>
<td>31.9</td>
</tr>
</tbody>
</table>
Table 42 shows the red rot reaction of the progeny of the cross between C. P. 29-116, a variety resistant to red rot, and C. P. 36-105, also a variety resistant to red rot. Only 20.9 per cent of the seedlings studied were resistant. In addition 56.6 per cent of the seedlings were commercially susceptible. This includes the seedlings classified in the table under "moderately susceptible" and "susceptible".

In the cross between the two red rot resistant varieties C.P. 36-13 x C.P. 36-105, only 10.7 per cent of the progeny were resistant to red rot. Furthermore, 73.8 per cent of the seedlings were commercially susceptible to this disease. The results are given in detail in table 44.

When C. P. 29-103, a red rot resistant variety was crossed with C.P. 36-105, a variety resistant to this disease, 22.2 per cent of the progeny were resistant to red rot. As shown in table 44, the percentage of plants was about similar in each of the four classes: resistant, moderately resistant, moderately susceptible and susceptible.

Among the progeny of the cross between the two red rot resistant varieties F. 31-962 x C.P. 36-105, there were 40.2 per cent of the seedlings resistant to red rot, while 38.4 per cent of the seedlings were commercially susceptible. It was observed that the internode tissues in many of the seedlings studied were severely infected by red rot. On the other hand, the nodes of the plants, classified here as resistant and moderately resistant were very resistant to this disease. The results are given in detail in table 45.

When the red rot susceptible variety C. P. 34-120 was crossed with the moderately susceptible variety C. P. 27-108, red rot resistant seedlings were obtained in the progeny. Table 47 shows that 23.6 per cent
and 28.2 per cent of the seedlings studied were resistant and moderately resistant respectively. In addition, the percentage of plants in each of the four classes was almost similar.

In the cross between the red rot susceptible variety C. P. 34-120 and the resistant variety C. P. 36-105, 33.8 per cent of the progeny were resistant to red rot. Table 47 shows that the percentage of the commercially susceptible plants (Mod. S. and S.) was 38.4.

When C. P. 29-103, a variety moderately resistant to red rot, was crossed with the susceptible variety C. P. 42-10, only 4.1 per cent of the progeny were resistant to red rot. On the other hand a very high percentage of the progeny were susceptible to this disease. Table 48 shows that 88.7 per cent of the progeny were commercially susceptible to this disease.

Table 49 shows that among the progeny of the cross between the moderately resistant variety C. P. 29-103 and C. P. 27-108 (susceptible to red rot), 43.1 per cent of the seedlings were resistant to red rot. There was also 13.7 per cent of the seedlings susceptible to this disease. Only 43.2 per cent of the progeny showed reaction similar to that of either of the parents, that is, they were either moderately resistant or moderately susceptible.

In the cross between the red rot resistant variety F. 31-962 and C. P. 27-108, a moderately susceptible variety, 20.3 per cent of the progeny were resistant to this disease. Table 50 shows that 21.9 per cent of the progeny were moderately susceptible, while 31.2 of the seedlings were susceptible to this disease.

Table 51 gives the summation of the results of red rot studies made on the progeny of the 1948 crosses. It was observed that in the
crosses between resistant x resistant parents there were always susceptible plants among the progeny. In the cross between susceptible x susceptible parents, about one-fourth of the progeny were resistant to red rot. In the cross between resistant x susceptible parents, the resistant plants among the progeny were equal in number and sometimes higher than those in the progeny of the crosses between resistant x resistant parents.

Table 52 shows the results obtained in the studies made on testing the red rot reaction of the progenies of the 1949 crosses. In the cross between the two resistant varieties C. P. 46-193 x C. P. 44-154, 14.7 per cent and 32.4 per cent of the progenies were moderately susceptible and susceptible to red rot, respectively. In the two crosses involving susceptible x susceptible parents, resistant seedlings always were obtained among the progeny. In crosses between the resistant x susceptible parents, namely C. P. 436-4 x Co. 356, and in the cross between the two susceptible parents C. P. 38-34 x C. P. 36-138, the percentages of resistant plants among the progeny was similar.
DISCUSSION

I. Inheritance of Resistance to Mosaic

The parentage of most of the varieties involved in the crosses used in these studies is given in appendix II. It is given in such a way that the parentage of each variety can be traced back as many generations as possible. It is observed that the great majority of the varieties used resulted from crosses between different species of the genus *Saccharum*, and of further intercrossing of the hybrids obtained, or from crossing the hybrids to other varieties of *S. officinarum*. This subject has been presented in more detail in the review of the literature, in which it was pointed out that the original crosses made in Java and India were conducted with a major objective of incorporating disease resistance into the noble cane varieties, *Saccharum officinarum*.

All the varieties of *S. officinarum* tested in several countries were found to be susceptible to sugarcane mosaic virus (62), (63), (65), (123), (124), (198), (253), (258). All self-fertilized seedlings of varieties of *S. officinarum*, and seedlings resulting from crosses between varieties of this same species were found by several investigators and in different countries to be susceptible to mosaic (63), (65), (196), (198), (258).

The varieties of *S. barberi* were reported by Brandes and Sartoris (65) to be susceptible to mosaic. However, they were not severely injured by the disease but were tolerant to it. Chunnee, a variety of *S. barberi*, and one of the ancestors of many of the varieties used in these studies, was observed to be susceptible to mosaic in the U.S.A. (63). All the seedlings of this variety were also susceptible to this disease (63). Jeswiet, 1927 (198) in Java stated that all the seedlings obtained from crosses between Chunnee and varieties of *S. officinarum* were susceptible.
to mosaic. When the first generation hybrids were backcrossed to varieties of S. officinarum, all the seedlings obtained were susceptible to mosaic. Apparently the mosaic-resistant varieties used in these studies which resulted from tri-species hybrids, namely S. officinarum, Chunnee variety of S. barberi, and S. spontaneum, did not obtain their resistance to mosaic from either S. officinarum or S. barberi. S. spontaneum stands as the only species that has contributed resistance to mosaic in the varieties studied.

S. spontaneum is composed of numerous forms that vary in their chromosome number, and constitute a series extending from latitude 40° N. to the equator. The groups concerned here are those that enter into the parentage of the varieties used in these studies. These are the Glagah group of Java, and the group from India used by the Coimbatore station in breeding work in the early days and which was the parent of varieties Co. 205 and Co. 206. This latter variety was a parent of variety Co. 281. This group of S. spontaneum canes is called here S. spontaneum of India.

Brandes et al 1939 (6) and Summers et al 1948 (347) observed that varieties of S. spontaneum collected from different countries when tested appeared to be immune to mosaic, and, as a group, the injury of those found to be susceptible was so slight to be practically negligible.

The first generation hybrids (F1) of crosses between mosaic-immune varieties of S. spontaneum from Java (Glagah) and varieties of S. officinarum were reported by Brandes 1931 (60) to be apparently immune to mosaic. In the F2 progeny exposed to natural infection at Audubon Park (Louisiana), 9 out of 650 seedlings showed mosaic symptoms (60). On the other hand Rands et al 1935 (283) stated that Glagah from Pasoeroean used in their studies as well as the S. spontaneum from other sources, with the exception of Imp. 470 variety, when crossed with other varieties of
S. officinarum, gave some mosaic susceptible seedlings in the F₁ generation hybrids. They concluded that incomplete dominance of resistance to mosaic was indicated by their results, and that it might be explained by the slight heterozygosity of the different S. spontaneum as indicated by known differences in their chromosome number.

The conclusion reached by Rands et al (283), that resistance to mosaic was inherited as an incomplete dominant character, is questioned, since the occurrence in the F₁ generation of a low percentage of susceptible plants is not an indication of incomplete dominance. The occurrence of these few susceptible plants can be attributed to pollen contamination, to chromosomal irregularity during meiosis in these highly polyploid canes, to the fact that the varieties were not homozygous for resistance, or to the possibility that the S. spontaneum varieties used in their studies were natural hybrids. This last possibility had been indicated by them as they stated that chromosome counts were being made to determine definitely whether or not the varieties used were hybrids.

Kassoer cane is one of the ancestors of many of the varieties used in these studies. Taxonomical studies by Jeswiet (198) and cytological studies by Bremer (67) indicated that this variety was a natural hybrid between S. officinarum and S. spontaneum of Java (Glagah). It is resistant to mosaic and its selfed seedlings were reported by Brandes and Klaphaak (63) as resistant to mosaic. Davis (117), (118) in Puerto Rico observed that self-pollinated seedlings of variety U. S. 541 and the progeny of crosses between U. S. 541 and S. C. 12-4 were also highly resistant to mosaic. Variety U. S. 541 is a self-pollinated seedling of Kassoer. In earlier studies Davis (116) concluded that mosaic resistance amounting to immunity might be secured by selection among seedlings containing only
one-eighth Kassoer germ plasm.

Apparently Kassoer obtained its resistance to mosaic from *S. spontaneum* of Java (Glagah) which is known to be resistant to this disease. As all the varieties of *S. officinarum* are susceptible to mosaic, Kassoer was used as a parent in crosses with other varieties in Java and the U.S.A. This leads to the conclusion that the seedlings that show resistance to mosaic and that were derived by crossing noble varieties with Kassoer and backcrossing to other noble varieties, had derived their resistance to mosaic from Kassoer. The latter in turn had obtained its resistance to mosaic from *S. spontaneum* of Java (Glagah).

A variety of *S. spontaneum* of India was used as a parent in different crosses at the Coimbatore Station in India. The variety Co. 205, which resulted from a cross between *S. officinarum* and *S. spontaneum* of India, was reported as susceptible to mosaic at Coimbatore, but was free from mosaic in other parts of India (130). Brandes and Sartoris 1936 (65) stated that the *F*₁ generation hybrids of crosses between *S. spontaneum* of India and *S. officinarum* were susceptible to mosaic. However, it is not clear that their statement was based on actual experimental data, but may have been based on a single observation that Co. 205 was susceptible to mosaic. One of the possibilities that arise out of their statement is that resistance to mosaic is inherited in a different manner in *S. spontaneum* of India and in *S. spontaneum* of Java (Glagah). The *F*₁ generation hybrids of crosses between *S. officinarum* and *S. spontaneum* of Java are resistant to mosaic, while the *F*₁ generation plants of crosses between *S. officinarum* and *S. spontaneum* of India are susceptible. Another possibility is that the variety of *S. spontaneum* India that was used in these crosses was not homozygous for the genes conditioning resistance to mosaic. A further possibility was
that Co. 205 did not result from such a cross but came from pollen contamination.

The variety Co. 214 resulted from backcrossing an F₁ generation hybrid between S. spontaneum of India and S. officinarum to another variety of the latter species (65). Summers et al 1948 (347) reported that this variety was immune to mosaic in an eight year period of observation. As the varieties of S. officinarum, and the seedlings resulting from intercrossing varieties of this species are susceptible to mosaic, the factors conditioning resistance to this disease in variety Co. 214, should have been obtained from S. spontaneum of India. Furthermore, the variety Co. 214 resulted from backcrossing an F₁ hybrid to S. officinarum. Since Co. 214 was immune to mosaic, this indicates that resistance to this disease in crosses between S. officinarum and S. spontaneum of India was inherited as a dominant character.

The conclusion reached after reviewing the literature is that all varieties of S. officinarum are susceptible to mosaic disease of sugarcane and that they do not carry genes conditioning resistance to this disease. In crosses between S. officinarum and S. spontaneum of Java (Glagah), the resistance to mosaic behaves as a dominant character. There are no definite data to show the mosaic reaction of the F₁ generation hybrids between S. officinarum and S. spontaneum of India. In crosses between some varieties of S. spontaneum collected from another locality (283) the resistance to mosaic disease appears to be inherited as a dominant character.

The results obtained in the present studies on inheritance of resistance to mosaic disease of sugarcane are summarized in appendix L. It is shown that mosaic-susceptible plants were obtained in the progeny of crosses between resistant x resistant varieties. Over 50 per cent of the
seedlings studied in nine crosses were susceptible to mosaic. Furthermore, in 26 crosses the percentage of mosaic-susceptible plants ranged between zero and 5 per cent. On the other hand, mosaic-resistant plants were obtained in the progeny of all the crosses between susceptible x susceptible varieties. In these crosses, the percentage of mosaic-susceptible plants did not exceed 50 per cent of the progeny. In addition the percentage of mosaic-susceptible plants was less than 15 per cent in the progeny of five crosses involving susceptible x susceptible parents.

The presence of large numbers of mosaic resistant plants in the progeny of crosses between susceptible x susceptible varieties suggests that resistance to mosaic is due to complementary factors. This indicates that in a cross between two susceptible varieties, there is one complementary factor or one set of complementary factors present in one parent which differs from the other factor or set of factors in the other parent. The progeny that get both complementary factors or sets of factors from the two parents would be resistant to mosaic.

Assuming that resistance to mosaic is conditioned by two dominant complementary genes, the symbols A and B are designated for each of these factors. A mosaic-resistant variety must have both of these genes. Its genetic constitution will be A-B. The symbol "-" indicates the possibility that the dominant allele A, or the recessive allele "a" may be present or absent. Any variety that does not have both of these genes will be susceptible to mosaic. A susceptible variety may have only one of these two genes, namely A or B. Such a variety may be of the constitution A- or B-. The results obtained in these studies on the crosses between susceptible x susceptible varieties will be discussed in the following paragraphs.
The mosaic-susceptible variety C. P. 38-34 apparently has chromosomes from *S. spontaneum* of Java (Glagah) and probably chromosomes of *S. spontaneum* of India, which were apparently obtained through its female parent Co. 421. This last variety was obtained from a cross between P.O.J. 2878 x Co. 285. As shown in appendix II, Dutt and Rao were of the opinion that *S. spontaneum* was the male parent of Co. 285. The mosaic-susceptible variety C. P. 43-74 has chromosomes from *S. spontaneum* of Java and probably also chromosomes from *S. spontaneum* of India. It came from the cross Co. 281 x C. P. 1165. Abbott 1938 (10) was of the opinion that C. P. 1165 has *S. spontaneum* in its ancestry. In the cross C. P. 38-34 x C. P. 43-74, 81 per cent of the seedlings obtained were resistant to mosaic (table 8). Since the varieties C. P. 38-34 and C. P. 43-74 are susceptible to mosaic, neither of them has both of the two complementary genes conditioning resistance to mosaic. It follows that each variety has only one or none of these two genes. If variety C. P. 38-34 has one gene only, namely the A gene, and variety C. P. 43-74 has the same gene A, all the progeny from a cross between these two varieties would be susceptible to mosaic. To obtain resistant plants, one of the parents, for example C. P. 38-34, should have one of the two genes, i.e., A; and the other parent, C. P. 43-74, should have the other gene, namely B. Variety C. P. 38-34 would produce the gametes A, AA, and gametes that do not have the gene A. The production of gametes having the genes AA, would be due to the irregularity of chromosome behavior, which results in the addition of a chromosome carrying the gene A. Variety C. P. 43-74 would produce the following gametes: B, BB, and gametes that would not have the gene B. The irregularity of chromosome behavior may result in the addition of chromosome carrying the gene B. The mosaic resistant
plants obtained in the progeny of the cross between these two varieties resulted from the fertilization of a female gamete of the variety C. P. 38-34 having the gene A by a gamete (male) of variety C. P. 43-74 which has the gene B.

The mosaic susceptible variety C. P. 43-44 was used as a female parent in a cross with mosaic-susceptible C. P. 38-34 and a high percentage of the progeny was resistant to mosaic. Variety C. P. 43-64, being susceptible to mosaic, may possess one of the two dominant complementary genes or it may not possess any of them. As indicated before, variety C. P. 38-34 carries the gene A, and as mosaic resistant varieties were obtained from the cross, C. P. 43-64 x C. P. 38-34, it follows that variety C. P. 43-64 probably possesses the other complementary gene, namely B. It was also indicated before that variety C. P. 43-74 possesses the gene B. Since both of the two susceptible varieties C. P. 43-64 and C. P. 43-74 are assumed to possess the gene "B" and lack A, it is expected that no resistant plants would be obtained from a cross between these two varieties. However, the results obtained (table 9) show that in the cross between them, a high percentage of the progeny was resistant to mosaic. This indicates that they possess different complementary genes, since each of these varieties does not possess the gene A. It follows that variety C. P. 43-64 possess a third gene, which is designated by the symbol C. As the results indicate the assumption that resistance to mosaic is conditioned by only two complementary genes does not hold, and the above results indicate the presence of at least three complementary factors. The following discussion is on that basis.

Assuming that variety C. P. 38-34 possesses the gene A, it follows that the susceptible variety C. P. 43-74 should possess the
genes B-C-. The resistant plants obtained from a cross between these two varieties would possess the genes A-B-C-

In the crosses of susceptible x susceptible varieties, a very high percentage of mosaic resistant plants was obtained among the progeny. This suggests the possibility in favor of selection toward gametes that carry the chromosome containing the genes for resistance. There is also another possibility that there are minor genes involved in addition to the complementary genes. Furthermore, it is likely that a susceptible variety possesses more than one of the set of the complementary genes. For example, if variety C. P. 38-34 possesses only the gene A-, it will produce gametes that carry the gene A- and gametes that lack it. However, if this variety possesses the genes A and B, the gametes produced would be: A-, B-, A-B- and possibly gametes that lack both of these genes. It was stated before, that variety C. P. 43-74 possesses the genes B-C-, the gametes that it produces would be: B-, C-, B-C- and possibly gametes that lack these genes. In a cross between these two varieties, the gametes having the gene C- produced by variety C. P. 43-74 when they fertilize eggs of variety C. P. 38-34 carrying the genes A-B-, will produce resistant plants. If the latter variety carries the gene A- only, when its gametes fertilize eggs of the former variety that possess the gene C-, the plants obtained would be susceptible to mosaic. Thus, a higher percentage of mosaic resistant plants is likely to be obtained in a cross between these two varieties if variety C. P. 38-34 carries the genes A-B- than if it carries the gene A only. The results obtained favor such an assumption. For convenience, the variety C. P. 38-34 is given the constitution A-B-. It follows that variety C. P. 43-74 will carry either the factors A-C- or B-C-. Applying the same assumption to the
other crosses involving susceptible varieties, it is likely that variety C. P. 43-64 carries another gene in addition to the gene C-assigned to it earlier. It follows that the constitution of variety C. P. 43-64 is A-C- or B-C-

It was indicated before that each of the varieties C. P. 43-64 and C. P. 43-74 carried either the genes AC or BC, since resistant plants were obtained from the cross between these two susceptible varieties. It follows that each of the varieties has one of the complementary genes which is not present in the other variety. So, if variety C. P. 43-64 is of the constitution A-C-, and variety C. P. 43-74 is either of the constitution A-C- or B-C-, it follows that the last variety should be of the constitution BC. Also, if variety C. P. 43-64 is of the constitution B-C- variety C. P. 43-74 will have the genes A-C-

When the mosaic-susceptible variety C. P. 43-64 was crossed with the susceptible variety C. P. 36-138, 87 per cent of the progeny was resistant to this disease. It was indicated before that variety C. P. 43-64 carried the genes A-C- or B-C-. Variety C. P. 36-138 should then carry the complementary genes which variety C. P. 43-64 does not have. If variety C. P. 43-64 of the constitution A-C-, a resistant plant would result from the union of a gamete carrying the genes AC, produced by this variety, with a gamete that has the gene B produced by variety C. P. 36-138. A high percentage of resistant plants was obtained from the cross between these two varieties, so it is more likely that variety C. P. 36-138 carried one of the complementary genes present in the variety C. P. 43-64, plus the other gene not present in said variety. Thus, if variety C. P. 43-64 is of the constitution A-C-, variety C. P. 36-138 will be of the constitution B-C-. 
Variety C. P. 36-138 was obtained from the cross Crystalina x 28 NG2S1. (Imp. 496). The female parent is a noble variety while the male parent was supposed to be a variety of S. robustum. As was indicated in the preceding paragraph, variety C. P. 36-138 carries at least one of the complementary genes conditioning resistance to mosaic, and it is more likely to carry two of these genes. It is also known that the noble varieties are susceptible to mosaic, and crosses between such varieties always yielded susceptible plants. Therefore, it is more likely that one factor of the two genes present in C. P. 36-138 came from the male parent S. robustum. There is another possibility that C. P. 36-138 resulted from the fertilization of the egg of the Crystalina parent by contaminating pollen that carried one or two complementary genes. Variety C. P. 43-64 came from the cross C. P. 36-138 x C. P. 27-108. It is likely that it obtained one or both of the two complementary genes present in it from the male parent C. P. 27-108. That is, the complementary gene present in C. P. 43-64, which C. P. 36-138 lacked, definitely came from the pollen parent in the cross that produced variety C. P. 43-64.

In the cross between the two mosaic-susceptible varieties C. P. 43-64 x C. P. 48-106, 92.8 per cent of the progeny were resistant to this disease. It was indicated before that variety C. P. 43-64 carried the genes A-C or B-C. This would indicate that variety C. P. 48-106 carried the third complementary gene, which was not present in variety C. P. 43-64. A mosaic-resistant plant would result from the union of a gamete produced by C. P. 43-64, having the genes AC with a gamete produced by variety C. P. 48-166 having the gene B. The high percentage of resistant plants obtained in the above cross suggests that variety C. P. 28-106 has one of the complementary genes in addition to the gene which C. P. 43-64 lacks.
So, if this latter variety is of the constitution $A\text{-}C\text{-}$, the variety C. P. 48-106 will have the genes $A\text{-}B$ or $B\text{-}C\text{-}$. Likewise, if C. P. 43-64 carries the factors $B\text{-}C\text{-}$, variety C. P. 48-106 would probably carry the genes $A\text{-}B\text{s}$ or $A\text{-}C\text{-}$.

Table 10 shows that the mosaic-susceptible variety Co. 281 was used as a female parent in crosses with each of the susceptible varieties C. P. 38-34 and C. P. 43-74. In each cross, there was a large number of resistant plants among the progeny. This suggests that Co. 281 carries at least one of the complementary genes conditioning resistance to mosaic. It was pointed out before that variety C. P. 38-34, apparently carries the genes AB. As mosaic-resistant plants were obtained in the cross Co. 281 x C. P. 38-34, it is apparent that variety Co. 281 carries the gene C. It was also pointed out that variety C. P. 43-74 carried either the genes BC or $A\text{-}C\text{-}$. If it carried the genes BC, variety Co. 281 would carry the gene A. It was indicated that this latter variety carried also the gene C, thus its constitution would be $A\text{-}C\text{-}$. On the other hand if variety C. P. 43-74 carried the genes $A\text{-}C\text{-}$, variety Co. 281 would then carry the genes $B\text{-}C\text{$. This indicates that the variety of *S. spontaneum* of India which is one of the ancestors of variety Co. 281 carries at least two of the genes conditioning resistance to mosaic which are also present in *S. spontaneum* of Java (Glagah).

Stevenson 1951 (325) in the British West Indies stated that resistance to mosaic disease was associated with the Javan *spontaneum* and the *sinense* chromosomes if the Coimbatore Local *spontaneum* did not carry any resistance gene, and hence this form was useless in breeding for mosaic resistance. Summers et al 1948 (347) reported that *S. spontaneum* Coimbatore, India was immune to mosaic during five years of observations,
and that *S. spontaneum* Coimbatore Local was also immune to this disease during three years of observations in Louisiana and Georgia. That these varieties were immune to mosaic is an indication that they carry the genes conditioning resistance to mosaic. This confirms the results obtained here which indicated that the mosaic-susceptible variety Co. 281, which has chromosomes of *S. spontaneum* India, carries two of the genes conditioning resistance to mosaic.

It was indicated before that variety Co. 214 was immune to mosaic in Louisiana. This variety resulted from backcrossing an *F₁* generation hybrid between *S. officinarum* and *S. spontaneum* to another variety of the former species. This indicates that resistance to mosaic was inherited as a dominant character and in turn the factors conditioning resistance in Co. 214 are dominant factors. As variety Co. 281 was also derived from crosses involving *S. spontaneum* of India, it is apparent that the genes carried by Co. 281 are also dominant ones. This is in agreement with what has been stated earlier, i.e., that resistance to mosaic disease of sugarcane appears to be conditioned by complementary dominant genes.

The mosaic-susceptible variety Imp. 1183 was used as a female parent in crosses with the two susceptible varieties C. P. 43-74 and C. P. 38-34. In every cross, mosaic-resistant plants were obtained among the progeny (table 13). From correspondence with Mr. C. O. Grassi, Botanist of the United States Department of Agriculture, it is inferred that "Mbeya, Imp. 1183" is a variety of *S. spontaneum* L. This variety was susceptible to mosaic at Houma, La. (14). The results obtained here suggest that this susceptible variety carries at least one of the complementary dominant factors conditioning resistance to mosaic disease of
sugarcane.

The results obtained prove that mosaic-susceptible varieties can be used as parents in a breeding program designed for developing mosaic-resistant varieties. Such susceptible parents can be inter-crossed with each other, and a high percentage of mosaic-resistant plants can often be obtained among the progeny, providing that each of the parents have chromosomes of *S. spontaneum* in their inheritance. Such chromosomes carry the dominant complementary genes conditioning resistance to sugarcane mosaic virus. The results also suggest that at least three dominant complementary genes seem to condition the resistance to the strain of sugarcane mosaic virus used in these studies. Another gene may be discovered if crosses involving other varieties are studied, or another strain of the virus is used.

In crosses involving mosaic-resistant x mosaic-resistant varieties, mosaic-susceptible plants were obtained among the progeny of most of the crosses made. Appendix I shows that the percentage of mosaic-susceptible plants ranged from zero per cent in some crosses up to more than 75 per cent in other crosses. This suggests also that resistance to sugarcane mosaic virus is conditioned by complementary dominant genes. The evidence supporting this hypothesis is reached from the following facts:

1. The *F*₁ generation hybrids of the crosses *S. officinarum* x *S. spontaneum* (Glagah) are resistant to mosaic.

2. When the *F*₁ hybrids are backcrossed to *S. officinarum* some resistant progenies occur.

3. A high percentage of susceptible plants occur in progenies of certain resistant x resistant crosses, while a high
percentage of resistant plants was obtained from some susceptible x susceptible crosses.

The percentage of mosaic-susceptible plants obtained in resistant x resistant crosses appears to depend on the behavior of the *spontaneum* chromosomes during meiosis. This is given in more detail in the following paragraphs.

Most of the varieties used in these studies possess some chromosomes of *S. spontaneum* L., in addition to at least a complete complement of *S. officinarum* L. chromosomes. As was shown in the review of literature and as given in appendix II, most of these varieties resulted from crosses between *S. officinarum* and *S. spontaneum* with subsequent backcrossing of the hybrids to different varieties of *S. officinarum*. The varieties resulting from backcrossing were in some cases further backcrossed to this same species (nobilization) or intercrossed with each other. There are also varieties that resulted from crosses between *S. officinarum* and the Chunnee variety of *S. barberi*. In addition there are other varieties that resulted from crosses involving these three different species.

As was stated before, the genes conditioning resistance to mosaic disease of sugarcane are carried on the chromosomes of *S. spontaneum*. It has to be pointed out that some of the varieties, such as U. S. 1694 and C. P. 1165, which are resistant to mosaic, have been reported as being obtained from selfing the variety P.O.J. 213, or as an F₂ generation of P.O.J. 213. These were actually open pollinated crosses, and some of the seedlings obtained probably resulted from pollen contamination (cross fertilization). These varieties apparently carry chromosomes of *S. spontaneum*. This explains the fact that they were reported as immune
to mosaic. Additional evidence is that plants of Kassoer, and of Kassoer seedlings and of *S. spontaneum* were planted in the breeding plots. Furthermore, Abbott 1938 (10) in discussing the results obtained in the red rot studies, was also of the opinion that variety C. P. 1165 had some chromosomes of *S. spontaneum*.

The behavior of the chromosomes during meiosis in the interspecific hybrids of the genus *Saccharum* has been given in the review of the literature. It was also pointed out that the $F_1$ hybrids between *S. officinarum* ($2n = 80$) x *S. spontaneum* ($2n = 112$) had the chromosome number 136. This suggested that the chromosome complement of *S. officinarum* was doubled. When the $F_1$ plants were backcrossed to *S. officinarum*, the resulting hybrids had 148 as the diploid chromosome number instead of the 108 normally expected. In the subsequent backcross the somatic number of chromosomes varied between 106 and 120. This suggested that in the second backcrossing (third mobilization) the chromosome number of *S. officinarum* did not double (67), (70), (71). When the seedlings of the third mobilization were further backcrossed to noble cane, the chromosome number of the resulting seedlings ranged from 94 to 100. This is the sum of the monoploid number of the parents. Others had from 120 to 126 chromosomes, which is the sum of the monoploid chromosome number of the parents. These forms had about seven *S. spontaneum* chromosomes (65).

The number of *S. spontaneum* chromosomes in the $F_1$ hybrids of the cross *S. officinarum* x *S. spontaneum* is 56. During meiosis, these chromosomes pair among themselves (65). Accordingly the seedlings resulting from the second mobilization are expected to have 26 *S. spontaneum* chromosomes. Varieties P.O.J. 2725 and P.O.J. 2878 resulted from the
same cross, E. K. 28 x P.O.J. 2364. Li et al 1948 (222), as reported by Li et al 1951 (221) observed that the number of Glagah (Spontaneum) chromosomes in variety P.O.J. 2725 was about 7, while in variety P.O.J. 2878 they observed the number to be about 21. This suggests irregularity of chromosome behavior during meiosis.

Evidence of influence of irregularities in chromosomes on inheritance of resistance to mosaic can be summed up as follows.

Most of the varieties used in the present studies have some chromosomes of S. spontaneum, in addition to at least a complete complement of S. officinarum chromosomes. During meiosis, some of the S. spontaneum chromosomes may pair with each other at synapsis and consequently would be distributed to opposite poles at anaphase, while those chromosomes that lack homologues would be distributed at random to opposite poles at anaphase, and some of them might lag behind, and would not be included in the nuclei resulting from the first meiotic division. This means the loss of one or more of the S. spontaneum chromosomes. The number of these chromosomes in a gamete would thus vary from 1 to 7 or perhaps more. The genes conditioning resistance to mosaic in current varieties are located on the S. spontaneum chromosomes. Consequently the number and kind of genes present in a gamete would depend on the presence in the gamete of the particular S. spontaneum chromosomes carrying them.

Some crosses of mosaic-resistant x mosaic-resistant parents gave a high percentage of mosaic-susceptible seedlings. If resistance to mosaic is due to complementary genes the tendency of the two parental varieties to lose the same S. spontaneum chromosome (carrying the same complementary gene for resistance) in meiosis will account for the low
percentage of resistant progeny in resistant x resistant crosses. This behavior of *S. spontaneum* chromosomes would also account for the very low percentage of resistant plants in some resistant x susceptible crosses.

It is of interest that Loh and Tseng 1951 (226) have reached more or less the same conclusion regarding the influence of irregularities in chromosomes on the inheritance of certain characters in sugarcane. They were of the opinion that the ability of a parent to transmit its visible characters to its offspring resulted from its capacity to maintain its chromosome balance. This means that such parents had obtained the ability to make pairs of homologous chromosomes in their own way, irrespective of other canes with which it was crossed.

In the present studies, the mosaic-resistant variety P.O.J. 2725 was used as a female parent in 8 crosses with other resistant varieties (Table 14). The percentage of mosaic-susceptible plants varied from 22.0 per cent to 54.9 per cent among the progeny of seven of these crosses. The variety C. P. 29*103 was derived from P.O.J. 2725. It was obtained from the cross P.O.J. 2725 x C. P. 1165. When these varieties were used as female parents in crosses with other resistant varieties, high percentages of mosaic-susceptible seedlings were obtained among the progeny. This suggests that in variety P.O.J. 2725 there is a tendency for one or more of the chromosomes carrying one or more of the complementary genes conditioning resistance to mosaic to be lost during meiosis. Many of the gametes produced by this variety would lack the chromosome or chromosomes carrying this gene or genes. Cytological studies (221), (222) indicated that variety P.O.J. 2725 carries about 7 chromosomes of Glagah (*S. spontaneum*), which is a comparatively low number. The chromosome number of Glagah is 112. It is of interest to note the behavior of one of
the varieties derived from P.O.J. 2878 which is a sister of P.O.J. 2725 but has about 21 *S. spontaneum* chromosomes.

Variety C. P. 34-120 was obtained from the cross Co. 281 x P.O.J. 2878. It was used as a female parent in 11 crosses with other mosaic-resistant varieties (table 5). In three of these crosses, the percentage of susceptible plants among the progeny was less than one per cent, and in five crosses the percentage of susceptible seedlings ranged from 1 to 5 per cent. When C. P. 34-120 was used in crosses with two susceptible varieties a low percentage of susceptible plants was obtained among the progeny. Among 179 crosses studied there were 16 crosses in which the percentage of susceptible plants among the progeny was less than one per cent per cross. Variety C. P. 34-120 was used as a parent in four of these 14 crosses. This indicates that a great number of gametes produced by C. P. 34-120 have most or practically all of the set of the complementary genes conditioning resistance to mosaic. As was stated before, this variety was obtained from the cross Co. 281 x P.O.J. 2878. This latter variety has about 21 *S. spontaneum* chromosomes, which is the highest number in comparison to its sister varieties. Furthermore, variety Co. 281 was obtained from crosses involving *S. spontaneum*. It is more likely then, that some of the *S. spontaneum* chromosomes carrying the genes conditioning resistance would have some homologous ones.

It has to be indicated that the point discussed above is not a general rule. It is not necessarily true that the varieties derived from a parent which transmits a high percentage of mosaic-resistant progeny should in turn transmit also a high percentage of resistance to mosaic. The sugarcane varieties studied are hybrids and highly heterozygous. They produce gametes that are likely to be extremely variable in their
genetic constitution. A mosaic-resistant sugarcane variety would produce gametes that may have one, two, or three of the complementary genes conditioning resistance to mosaic, depending upon the chromosome behavior during meiosis, each variety resulting originally from the union of two gametes. The genetic constitution of the new variety depends upon the genetic constitution of these two gametes (parents). Thus a variety may have none or a pair or more than a pair of homologous chromosomes that carry the complementary genes conditioning resistance to mosaic. This explains how a resistant variety which for convenience may be called "I" would, when used as a parent in crosses, produce a high percentage of mosaic-resistant plants in its progeny. In addition there may be two resistant varieties "Ra" and "Rb" which were derived from a cross involving variety "I", both of which or either are, when used as a parent in a cross would yield a high percentage of mosaic-resistant plants in the progeny. It also explained the condition in which a variety "II" would produce a low percentage of mosaic-resistant plants from crosses involving this variety "I". The resistant variety "IIa" is derived from the cross "II x III", yielding a high percentage of resistant plants. This latter case is explained on the basis that most of the gametes produced by variety "IIa" carry the set of complementary genes conditioning resistance to mosaic. This variety has obtained, in addition to the chromosomes carrying the complementary genes obtained from its parent "II", another set of homologous chromosomes obtained from the other parent "III".

The conclusion reached is that the ability of a variety when used as a parent, to transmit a high or a low percentage of mosaic-resistant plants, is dependent upon the behavior during meiosis of the
S. spontaneum chromosomes which carry the complementary genes conditioning resistance to mosaic. This in turn determines the genetic constitution of the gametes produced by the plant. The gametes can carry one or two or more of the complementary genes. Apparently the frequency of production of each kind of these gametes depends mainly on the behavior of S. spontaneum chromosomes during meiosis.

This conclusion stated above and the hypothesis advanced that resistance to mosaic is conditioned by dominant complementary genes, leads to a discussion of another point. The irregularity of chromosomal behavior during meiosis, and the fact that many of the current sugarcane varieties are not homozygous for all factors conditioning resistance to mosaic, would result in a condition in which many of the gametes produced by certain varieties would not possess all of the complementary factors. If resistance to mosaic is conditioned by three complementary genes A, B and C, the gametes produced by many of these varieties could be of the constitution: A, B, C, AB, AC, BC, ABC, and also gametes that do not carry any of these genes. The plant breeder is interested in obtaining as many resistant plants as possible among the progeny of the crosses made. Although the gametes having one or two of the complementary genes do not carry all the set of these genes conditioning resistance to mosaic, these gametes are of considerable value to the plant breeder. When two resistant varieties are crossed with each other, and when a gamete produced by one parent possessing one of the complementary genes (e.g. A), is united with a gamete produced by the other parent that possesses the other two complementary factors (BC), the zygote resulting from such a union has the complementary set of genes. The seedling that will develop will be resistant to mosaic. This point emphasizes the fact that, when a cross is made
between two resistant varieties, the resistant plants obtained among the progeny are not produced by the union of the gametes each having the set of the complementary factors (ABC). The loss of a chromosome or two carrying one or two of the complementary genes in meiosis and the production of gametes lacking one or two of these genes is compensated if these gametes unite with gametes having the other complementary genes or gene. The result is that a high percentage of resistant plants are obtained from crossing such varieties. However, if both varieties have a tendency to lose the same one or two complementary genes, a cross between these two would result in a high percentage of susceptible plants in their progeny.

This principle is also applicable to the cases in which crosses between resistant x susceptible parent are undertaken. Suppose the susceptible variety carries the genes BC, and the resistant variety carries the genes ABC. If this latter variety produces gametes of the constitution A, AB and AC, and these are united with the gametes produced by the susceptible parent carrying the other complementary genes or gene, mosaic-resistant plants will be obtained. However, if both parents produce many gametes that lack the same complementary gene, a high percentage of susceptible plants would result from crosses between such varieties.

Brandes 1931 (60) was of the opinion that resistance to mosaic in sugarcane or lack of it was not to be assigned to a single pair of allelomorphic factors. He called attention to the fact that within the progenies of certain crosses there were graduated ranges of response to mosaic, extending from extreme susceptibility to complete immunity, which he believed to be attributed to multiple factors. He also expressed the belief that susceptibility to mosaic was a recessive character. With
susceptibility to mosaic a recessive character, if it was conditioned by multiple factors a recessive variety should be homozygous for all the recessive factors conditioning susceptibility to mosaic. In this case the crosses involving susceptible x susceptible varieties will always yield mosaic-susceptible seedlings. The results obtained here do not confirm such an opinion. In every cross between susceptible x susceptible varieties there were always mosaic-resistant plants among the progeny. In certain crosses the percentage of resistant plants among the progeny was more than 75 per cent of the seedlings tested. These results indicate that resistance to mosaic is conditioned by complementary dominant genes. The graduated range of response to mosaic among the progenies of certain crosses would be attributed to the presence of modifying factors which affect the degree of expression of the disease.

Li, Cheng and Leung in 1951 published a paper (221) on genetic analysis of the hybrids obtained in crossing the sugarcane variety P.O.J. 2725 with a wild grass, Miscanthus japonicus. However, in their paper they elected to discuss the inheritance of resistance to mosaic in sugarcane, which was not the subject of their work. They stated that resistance to mosaic from Glagah is controlled by a set of genes scattered on different chromosomes. Furthermore, the degree of resistance, as expressed by the varieties derived from such a cross, was both quantitative, i.e. the more Glagah chromosomes, the higher the resistance, and qualitative, i.e. having more resistant genes on certain chromosomes. They based these statements on the fact that the progeny of the cross P.O.J. 100 x P.O.J. 2364 yielded the varieties P.O.J. 2714, 2725, 2878, and 2883. Furthermore, the number of Glagah chromosomes varies in these hybrids, being lowest in P.O.J. 2725, which has about 7, and highest in P.O.J. 2878, which has
about 21. Summers et al 1948 (347) reported that variety P.O.J. 2725 was
immune to mosaic for a 13-year period of study, and that variety P.O.J.
2878 showed 17 per cent of mosaic in a 10-year period of observation.
This does not confirm the statements made by Li et al (221). The variety
P.O.J. 2725 which had the lowest number of Glagah chromosomes showed
immunity to mosaic, while P.O.J. 2878, which had the highest chromosome
number, showed 13 per cent of mosaic. This does not agree with his
statement that resistance to mosaic was a quantitative character; the
more Glagah chromosomes, the higher the resistance. In addition, the
variety P.O.J. 2725, which had only 7 Glagah chromosomes when it was
crossed with noble varieties, yielded also mosaic-resistant (immune)
varieties. Davis 1929 (116) stated that one per cent of the P.O.J. 2725
x S. C. 12-4 seedlings were susceptible to mosaic. He further stated
that immunity to mosaic might be maintained in seedlings having one-
eighth of "Kassoer blood". Many of the seedlings resulting from the
cross P.O.J. 2725 x S. C. 12-4 would be expected to have less than the
seven Glagah chromosomes possessed by P.O.J. 2725. However, a high
percentage of resistant plants was obtained by Davis in the cross dis-
cussed. This would indicate that many of the seedlings that have less
than 7 Glagah chromosomes showed resistance to mosaic, or in the
terms of the other investigators showed "immunity" to this disease.
It appears that the statements in the literature that the more Glagah
chromosomes, the higher the resistance to mosaic, does not necessarily
hold true. Furthermore, the statement by Li et al that there are some
chromosomes which carry more genes conditioning resistance than the
other chromosomes, has no evidence whatsoever to support it. They
themselves did not present any data to support their opinion.
Many cases have been reported in the literature in which the same phenotypic character might be controlled by distinct gene pairs. Walker 1951 (368) stated that the best example of this phenomenon was illustrated by the work of Briggs and associates with powdery mildew of barley caused by *Erysiphe graminis hordei* (DC.) Marshall. The work done and the conclusions reached on this subject have been treated in detail in the last part of the review of literature. Crosses were made between different varieties of barley, and the reaction of the *F*₁ generation hybrids, *F*₂ generation, and *F*₃ generation plants to physiologic race 3 of the mildew fungus was studied. Stanford and Briggs 1940 (77) concluded that there were seven different genetic factors for mildew resistance (to race 3 only), six dominant and one recessive. The number of factors in a single variety varied from one to three factor pairs. Favert 1949 (165) reported on two additional genetic factors for mildew resistance. He concluded that with these two new factors mentioned, nine genes were already known to be involved in the resistance to mildew race 3 in barley, the largest number of loci related with resistance to a plant disease.

*Saccharum spontaneum* includes different forms that vary in their chromosome number and geographical distribution. The studies reported here dealt with varieties that obtained their resistance to mosaic from *S. spontaneum* of Java known as Glagah. Other varieties were descendants of crosses involving *S. spontaneum* of India. The hypothesis advanced here in regard to the inheritance of resistance to mosaic applies to the varieties that obtained their resistance to mosaic from these two *S. spontaneum* sources. If further studies on the inheritance of mosaic are conducted on other forms or varieties of *S. spontaneum* it will not be amazing if other genetic factors for mosaic resistance are found. Furthermore, the nature
of these genetic factors may not be the same as the complementary do- 
nant genes reported here. As shown in the barley studies, some varieties 
possess a dominant gene for resistance to physiologic race 3 of the powdery 
mildew fungus, while another variety possesses a recessive genetic factor 
for resistance. This would suggest that in studying crosses involving 
other forms of S. spontaneum or S. sinense, resistance to mosaic may be 
found to be inherited as a recessive character. If these studies were made 
and such results were obtained, it will not affect the validity of the conclu-
sions reached from the present studies, since they deal with resistance to 
mosaic in other varieties or forms of S. spontaneum. The importance of 
the present study is that it deals with the varieties which have been used 
as a source for resistance to mosaic in the breeding program in the 
United States and many countries throughout the world. These varieties 
are still in active use in these different places. The efforts of the plant 
breeders in using them resulted in saving the sugar industry in several 
localities and was a great step for obtaining a better and more permanent 
ariculture.

The results obtained in the comparison of the flat inoculation 
technique and the pot inoculation technique are given in tables 30 through 
36. It is observed that in the seven crosses listed in table 34, a much 
higher percentage of susceptible seedlings was obtained by using the 
flat inoculation technique. The differences between the percentages of 
plants showing mosaic symptoms obtained by using these two inoculation 
techniques ranged from 62.3 per cent in one cross to 37.2 per cent in 
another cross. In the pot inoculation technique, the seedlings were in-
oculated when they were in an older stage of development than those 
inoculated in the flats. This suggests that there are probably two types of
resistance to mosaic among these seedlings. In the first type the seedlings would be resistant to a given strain of the mosaic virus at their very early stage of development (in the flat), and remain resistant to this strain throughout their life. In the second type, the seedlings would be susceptible to mosaic when they are inoculated at an early stage of development (in the flat), but would be resistant to this disease if they were inoculated in a later stage of their development (in the pots). Such types of resistance have also been reported to occur in wheat. Certain wheat varieties are resistant in the seedling stage and remain resistant until heading and maturity to a given physiologic form of the black stem rust fungus *Puccinia graminis tritici* Eriks and Henn. In the other type, the plants are susceptible to stem rust in the seedling stage, but resistant in the field. It was also found that the genes conditioning mature plant resistance and those conditioning resistance in the seedling stage were independently inherited.

In the crosses shown in table 34, the seedlings that showed resistance to mosaic when they were inoculated in the flat possess the first type of resistance indicated earlier. On the other hand, the seedlings that showed resistance when they were inoculated in the pots apparently fall into two groups. One group of these seedlings would possess the first type of resistance, while the other group would possess the second type of resistance in that they would be susceptible to mosaic if inoculated at a very early stage (in the flats), while they would be resistant to mosaic, if inoculated in a later stage of their development (in the pots). The percentages of the seedlings possessing this type of resistance in the progeny of a certain cross could be roughly estimated by subtracting the percentage of mosaic-resistant seedlings in the flats from the percentage of resistant
seedlings in the pots.

The results given in table 35 show that a higher percentage of mosaic-susceptible seedlings was obtained by using the flat inoculation technique. The differences between the percentages of plants showing mosaic symptoms by using the two inoculation techniques ranged from 18.7 per cent in one cross to 11.1 per cent in another cross. The differences here are not as great as those in the crosses given in table 34, which ranged from 62.3 per cent in one cross to 32 per cent in another.

The results given in table 36 show that in the progeny of the four crosses studied the percentage of plants showing mosaic symptoms when the flat inoculation technique was used was slightly higher than when the pot inoculation technique was used. The differences between the percentages of plants showing mosaic symptoms by these two techniques ranged from 7.3 per cent in one cross to 0.6 in another cross. In these studies a difference of about 6 or 7 per cent can occur due to chance. It is thus concluded that in these four crosses there was no apparent difference in the results obtained between the flat and pot inoculation techniques. This indicates that apparently all the mosaic-resistant seedlings in the progeny of these four crosses possess only one type of resistance.

In only one cross was the percentage of susceptible seedlings higher when the pot inoculation technique was used than when the flat inoculation technique was used (table 36). However, the difference was only seven per cent, which is likely to have been due to chance variation.

The results obtained in the comparison of the flat inoculation technique and the pot inoculation technique suggests that there are apparently two types of resistance to sugarcane mosaic virus among the
seedlings. These types have been described. They also indicate that they are genetically controlled. Further evidence is as follows: 1) The percentage of the seedlings possessing each type of resistance among the progeny of a given cross differed according to the parentage entering the cross. 2) In a group of crosses (table 36), all the resistant seedlings possessed one type of resistance only. 3) In another cross, some of the resistant seedlings of the progeny of a given cross possessed one type of resistance, while the remaining resistant seedlings possessed the other type of resistance. 4) The crosses mentioned in item 3 can be further divided into three groups. In one group, the majority of the resistant seedlings of the progeny of a given cross possessed resistance to mosaic at a very early stage of their development (in the flats). In a second group, the majority of seedlings of a given cross possessed the resistance of the other type. The third group included crosses which were characterized in that the progeny of a given cross, the percentage of resistant seedlings that possessed one type of resistance was about equal to the percentage of seedlings possessing the other type of resistance.

The question arises concerning the validity of the two inoculation techniques. The different methods of inoculating sugarcane plants with the sugarcane mosaic virus and the various methods attempted for transmitting this virus have been given in great detail in the review of the literature. Bain 1944 (37) found that the higher percentages of infection were obtained when either 100-mesh sand or carborundum was used as an abrasive in inoculating sugarcane seedling with the mosaic virus than by using the needle-prick method of inoculation. Abbott 1949 (12) attempted to inoculate the seedlings in the flats by crushing the
unfolding leaflets of the plants between small flat pieces of wood covered with fine sandpaper and then atomizing the seedlings with the inoculum. He stated that infection comparable to that obtained by other methods was obtained. He also stated that it was difficult to avoid uprooting some seedlings and practically impossible to give uniform treatments to all plants.

The flat inoculation technique used here is a very rapid method and saves a lot of time. Several thousands of seedlings can be inoculated in 20 minutes. On the other hand, with the other method the average technician would inoculate about 3000 seedlings a day. Sometimes as many as 6000 seedlings were inoculated per day during these studies.

In many crosses a higher percentage of mosaic-susceptible seedlings were obtained by using the flat inoculation technique than by using the pot inoculation technique. This indicates that many seedlings were discarded which would be resistant to mosaic if inoculated at a later stage of their development (in the pots).

It is suggested that the pot inoculation technique would be better than the flat technique. In the latter technique many seedlings which would be resistant to mosaic in the field are unnecessarily discarded, thus eliminating many potentially desirable seedlings.

The period of incubation of the mosaic disease varied from four days to a period of seven weeks and perhaps longer. Some of the seedlings did not show the mosaic symptoms when they were transplanted to the field, but later they showed the symptoms of the disease. In such cases, the appearance of mosaic symptoms could have occurred as a result of natural infection in the field and/or as a result of the inoculation
done in the greenhouse. Assuming that the latter case occurred the incubation period lasted probably more than seven weeks. It was also observed that the incubation period differed among the seedlings of the same cross. In addition, some crosses were characterized by the disease having a short period of incubation among the susceptible seedlings. Such seedlings generally showed severe symptoms of the disease. It can be inferred that when the incubation period was very short (4 days) the seedlings did not have tolerance to the disease. Those seedlings showed severe mosaic symptoms. In addition, in the recovery studies, during August 1951, there were two crosses whose susceptible seedlings did not show any recovery of the disease symptoms. It was observed earlier in the greenhouse that the incubation period of the disease was very short in those seedlings. Since they did not show any recovery from the disease symptoms, they apparently did not have tolerance to this disease. It appears, then, that in these two crosses, the failure of the susceptible seedlings to recover in the field was associated with the very short incubation period was associated with tolerance to mosaic. The seedlings that had short incubation periods showed severe mosaic symptoms. It appears that there is a possibility of the presence of some genetic factors that govern the plants tolerance to mosaic.

Recovery of diseased plants of sugarcane from mosaic symptoms is a known phenomenon which has been observed by various investigators (213), (314), (137), (138), (208), (286), (357), (360), (173), (347). Summers et al 1948 (347) pointed out that under certain conditions the ability of a sugarcane variety to recover from mosaic might be significant in reducing the disease. They also stated that probably one of the most important practical aspects of recovery is its possible utility in cane breeding, where
it might be hoped that it would prove to be a heritable character that may be transmitted to desirable seedlings not highly resistant or immune. They were also of the opinion that this character might be greatly accentuated in an occasional seedling and be one of the important factors for resistance.

The studies reported here were undertaken as a first step in a genetical study to find out whether or not the phenomenon of recovery from mosaic symptoms is an inherited character. The information obtained in such a study would probably be of help to the plant breeder.

The seedlings that showed mosaic symptoms in the greenhouse were transplanted to the field in April 1951 and examined in the period between July 4 and July 14 of the same year. It was observed that many seedlings showed recovery from the mosaic symptoms. A seedling was considered recovered when all the leaves of all the stalks of the stool showed recovery from the disease symptoms. Of the 1470 seedlings studied, 4.4 per cent recovered. It was also observed that in certain crosses there was no recovery among the seedlings.

Kunkel 1924 (213) observed two types of recovery from mosaic symptoms. In the first type the diseased shoots of a stool might at times begin to produce healthy leaves. Later, the old diseased leaves died and fell off. Such stools might grow to maturity without showing any further signs of the disease on the leaves. In the second type, none of the diseased shoots actually recovered, but the new shoots produced might be healthy. The diseased shoots remained small and were overgrown by the healthy ones. After a time, the diseased shoots might die and the stool might then remain healthy to maturity. This second type of
recovery described by Kunkel was never observed here. During the examinations conducted in July it was observed that in certain cases some of the stalks showed recovery from mosaic symptoms, while another stalk within the same stool showed the symptoms of this disease. Such stools are called here partially recovered. Furthermore, the diseased stalks of such stools were equal in size, if not larger in some cases than the recovered ones.

A second examination of the seedlings was undertaken in the period between August 24, 1951 and August 25, 1951. The number of recovered seedlings and partially recovered ones was recorded and the results are given in tables 38–41. The observations at this time were in agreement with those made in July in that the second type of recovery described by Kunkel was never observed. In no case did the diseased stalks remain small and become overgrown by the healthy ones; neither did the diseased stalks die, as stated by Kunkel. On the other hand during the August observation, and during an observation made in late October, in which no data were recorded, there were seedlings whose stalks showed recovery from the diseased symptoms. However, there were new shoots and water sprouts which came out late in the season that showed the mosaic symptoms. The occurrence of such new growth was helpful in the classification of the seedlings according to their type of recovery. Such stools were considered partially recovered. Further, it showed that new diseased shoots may come out later in the growing season in apparently-recovered stools, which contradicts Kunkel's statement that the diseased shoots die later in the season in some of the recovered stools. The presence of water sprouts or the occurrence of new shoots that come out later in the
growing season is a known phenomenon observed by the sugarcane breeders and farmers.

The results obtained during the second examination showed that 19.0 per cent of the seedlings studied recovered from the mosaic symptoms. It was observed that no recovery occurred in two out of the eleven major crosses (tables 38 and 39). Furthermore, the percentage of recovered seedlings varied according to the cross. This suggests that the recovery phenomenon is inherited as a genetic character. For example, when variety C. P. 42-13 was used as a male parent in four crosses with four different female varieties, the percentage of recovered seedlings among the progeny varied according to the female parent used (table 38). In the progeny of these crosses, the percentages of recovered seedlings varied from 0.0 in one cross to 22.4 in another cross. It is apparent that the recovery that occurred was an expression of the genotype of the plant in the environment prevailing during these studies. The genotype of each plant is a result of the recombination of the genetic factors obtained from each parent entering the cross. The wide variation in the percentage of recovered plants among the progeny of different crosses suggests that this was a result of the different genetic recombinations occurring from these crosses.

Reaching the conclusion that recovery from mosaic symptoms in sugarcane seedlings is apparently a genetic character, a program has to be carried out for studying its mode of inheritance with the hope that the information obtained will be of value to the plant breeder. An effort has been started in this subject with the hope that this work would be continued. One hundred of the recovered seedlings were picked up at random in November 1951, and planted in the field. The plants that come out will be examined during the 1952 growing season. Among the suggestions for
continuing these genetic studies are: some of these plants come up and which do not show symptoms of the disease will be chosen, and sent to the Breeding Experiment Plot at Grand Isle or to the Canal Point Station. If these plants flower, different crosses will be made and the phenomenon of recovery would be studied among the progeny. It is also of interest to cross some of the recovered seedlings with their sister seedlings that did not show any recovery. In addition, the recovered seedlings would be crossed with diseased seedlings from the crosses in which no recovery was recorded.

East 1931 (137) stated that the sugarcane plant after recovery from mosaic symptoms might remain healthy for several years. He also stated that a plant might recover and be reinfected as many as three times. The results of the studies on the flat and pot inoculation techniques indicated that there were seedlings which were susceptible to mosaic when they were inoculated in the flat, but if such seedlings were inoculated in a later stage of development (in the pots) some of them would be resistant to mosaic. The possibility arises that there may still be another phase of resistance when the plants are in an older stage of development. In other words, even if the seedlings were susceptible to mosaic in the greenhouse, would some of them in a later stage of development, that is, in the field, show resistance to this disease? This possibility is also applied to those seedlings that showed recovery from mosaic symptoms in the field. These seedlings were susceptible to this disease when they were inoculated at an early stage of their development in the greenhouse. Would some of them be resistant to infection in the field? Inoculation of the recovered seedlings with the sugarcane mosaic virus while they are growing in the field would give an indication of an answer to this problem.
The studies on recovery from mosaic disease as reported in the literature were done on the commercial varieties. In such studies, a population of the same genotype of a single variety was studied. The number of varieties studied and reported in the literature is quite small. They were also dealing with cases in which some plants of a certain variety would recover, while others continued to show the symptoms of the disease. In other words, they were dealing with plants which according to their genetic constitution were susceptible to the disease in the field.

The recovered seedlings obtained in the present studies each represent a variety by itself. These seedlings, and those that might be obtained in future years, provide a wealth of different genotypes. Inoculation studies can be made in the field to determine whether or not some of the recovered seedlings are actually resistant to the disease in the field. Such information would contribute to the knowledge of the types of resistance to this disease and would provide interesting information which might call for reconsidering the validity of the techniques used now in the sugarcane breeding program at Louisiana.

At the present time in the sugarcane breeding program conducted by the Louisiana Agricultural Experiment Station and by the United States Department of Agriculture, all the sugarcane seedlings are inoculated in the greenhouse with sugarcane mosaic virus. The seedlings that show the mosaic symptoms are discarded and the remaining symptomless plants are transplanted to the field. From these latter seedlings, the potential commercial varieties are selected. The studies on the recovery from mosaic symptoms showed that in certain crosses there was no recovery in the mosaic infected plants in the field, while in other crosses, there was recovery from such symptoms, which amounted to 51.6 per cent in the
crosses which are known from previous experience to yield a large number
of agronomically acceptable selections from their progeny, and which are
known to produce a considerable percentage of recovered seedlings among
their progeny, the seedlings which show mosaic symptoms in the green-
house would not be discarded. Such seedlings would also be transplanted
to the field and examined for recovery from mosaic symptoms. These re-
covered seedlings would provide the plant breeder with additional material
from which he could select in his effort to produce better sugarcane
varieties, a step in the plant breeder's effort to provide a permanent
agriculture.

II. Inheritance of Resistance to Red Rot

Considering the inheritance of resistance to red rot disease of
sugarcane caused by *Physalospora tucumanensis* Speg., Abbott 1938 (10)
concluded that high susceptibility to this disease is characteristic of *S.
officinarum*. He also stated that among the New Guinea collection of
Brandes and Jeswiet, tentatively classed as *S. officinarum*, there was a
red rot resistant variety, and another one that was moderately resistant.
He was of the opinion that these two varieties might have been of hybrid
origin.

Chunnee, and Hatoonie were the only varieties of *S. barberi*
studied by Abbott (10). He classified them respectively as very susceptible
and moderately resistant to red rot. Chunnee was considered by Barber
1901 (39) and Butler and Hafiz Khan 1913 (84) to be resistant to this
disease. Abbott 1938 (10) was of the opinion that the forms of the red
rot fungus present in the United States, or at least certain forms of them,
were more virulent than the Indian forms.
In regard to *S. robustum*, the reaction of only one variety was reported by Abbott (10). He considered it susceptible to red rot.

Thirteen varieties or collections of *S. spontaneum* were reported by Abbott (10) as moderately resistant to red rot. Furthermore, another variety of this species was observed to be very susceptible to this disease. These varieties included collections from India, Formosa and five collections of Glagah. However, the question arises as to whether all these varieties studied by Abbott were actually moderately resistant. He stated that the large cavities in the stalks of *S. spontaneum* made it difficult to obtain typical red rot symptoms comparable to those in the solid stalk canes. Further, the typical mottling generally characteristic of this disease was absent in the *S. spontaneum* varieties. These statements would suggest that many of these *S. spontaneum* varieties were resistant to red rot and not moderately resistant. The red discoloration of the tissues of sugarcane stalks may result from a number of different causes and it is not necessarily an indication of the presence of the red rot fungus. If Abbott had observed such discoloration, it is not an indication that these varieties were infected by the red rot fungus. In addition, he stated that the typical mottling generally characteristic of this disease was absent. This would suggest that the disease might have been absent. These points suggest that at least some if not all the *S. spontaneum* varieties considered by Abbott as moderately resistant, could be classified as resistant to red rot.

The hybrids resulting from the crosses of *S. officinarum* x *S. spontaneum* were reported by Abbott (10) as very susceptible to red rot. Kassoer, a natural hybrid of these two species, was observed to be also very susceptible to this disease.
Abbott 1938 (10) stated that all of the red rot resistant seedlings that had been encountered among thousands of plants produced at the Canal Point Station, carry at least a trace of *S. spontaneum* inheritance, and while some of them were also traceable to *S. barberi*, many were not. This led him to suspect that some forms of *S. spontaneum* might be resistant, or possess resistance as a recessive factor. It appears, then, that *S. spontaneum* was a source of resistance to red rot in all the resistant varieties used in the present studies. With this point in mind, a discussion will follow on the results obtained on the red rot studies.

Tables 51 and 52 show the results obtained in the red rot studies. Two crosses involved highly resistant × highly resistant parents. In one 40 per cent of the progeny were as resistant as the parents and only 20 per cent were highly susceptible. However, in the second of these crosses only about 10 per cent of the progeny were as resistant as the parents and 50 per cent were highly susceptible.

In three crosses between susceptible × susceptible varieties, there were always resistant seedlings among the progeny, ranging from 22.8 per cent in the progeny of one cross to 26.4 per cent in another. The percentages of these resistant seedlings were higher than those obtained in two of the crosses between resistant × resistant parents. In the crosses between resistant × susceptible parents, the percentage of resistant seedlings among the progeny was about equal to those obtained in susceptible × susceptible crosses, and in certain crosses higher than those obtained in resistant × resistant crosses.

A hypothesis is advanced as an interpretation of the results obtained that resistance in sugarcane to the red rot disease is governed by one or a few genes for resistance from *S. spontaneum*, plus a dominant
inhibitor gene from *S. officinarum*. The inhibitor gene from *S. officinarum*
masks the effect of the factors for resistance from *S. spontaneum*. A
group of modifier factors also influences the degree of the reaction of
the plants to the disease. The irregular chromosomal behavior occurring
in sugarcane varieties during meiosis influences the ratios of resistant to
susceptible plants obtained in the progenies. The various facts supporting
this hypothesis are as follows:

1. All varieties of *S. officinarum* are susceptible to red rot.
Most varieties of *S. spontaneum* tested are resistant or moderately re-
sistant to this disease. Abbott (10) stated that all the C. P. seedlings
that were resistant to red rot have a trace of *S. spontaneum* in their in-
heritance. Resistance can then be traced in the hybrids studied to *S.
spontaneum*.

2. The *F₁* hybrids of the cross *S. officinarum* × *S. spontaneum*
are susceptible to red rot (e.g. Kassoer, Hind’s Special, L 1081 and Toledo).

3. When the *F₁* hybrids were backcrossed to *S. officinarum*, sus-
ceptible seedlings were always obtained (e.g. P.O.J. 2222, P.O.J. 2364,
P.O.J. 2714, P.O.J. 2725 and P.O.J. 2727).

4. When the hybrids with some *S. spontaneum* in their inheri-
tance were intercrossed red rot resistant seedlings were obtained. An
example of that is found in that most C. P. varieties that are resistant
to red rot resulted from crosses between two susceptible parents.

These facts can be further interpreted as follows. *Saccharum*
*spontaneum* carries one or more genes conditioning resistance to red rot.
It is assumed that resistance is inherited as a dominant character. The
number of genes conditioning resistance in *S. spontaneum* is unknown,
but for illustrative purposes it is assumed to be one gene, and is
designated as R. A resistant variety of *S. spontaneum* would have the constitution "RR".

Varieties of *S. officinarum* lack the dominant gene "RR" for resistance, and therefore would possess the recessive alleles "rr". In addition these varieties carry the dominant epistatic gene "I". This gene masks the expression of the gene for resistance "R" of *S. spontaneum*. Varieties of *S. officinarum* would have the constitution "rrII". Varieties of *S. spontaneum* carry the recessive allele "i" which has no effect on the expression of the gene "R". The crosses between *S. officinarum* and *S. spontaneum* would be as follows:

\[
\begin{align*}
S. \text{ officinarum} & \times S. \text{ spontaneum} \\
P_1 & \text{rrII} \times \text{RRii} \\
F_1 & \text{I4Rr}
\end{align*}
\]

The hybrids are susceptible to red rot because of the presence of the gene I which masks the effect of the gene "R" for resistance. The backcrossing of the F₁ hybrids to *S. officinarum* would be as follows:

\[
\begin{align*}
S. \text{ officinarum} & \times F_1 \text{ hybrid} \\
P_1 & \text{rrII} \times \text{RrII} \\
gametes & \text{ri} \quad \text{RI, Ri, rI, ri} \\
\text{Backcross progeny} & \text{RrII, RrII, rrII, rrII}
\end{align*}
\]

The plants obtained from the backcrossing of the hybrids to *S. officinarum* are all susceptible to red rot because of the presence of the epistatic gene I. This factor is obtained from the *S. officinarum* parent. Many of these seedlings obtained from the backcrossing possess the gene "R" for resistance. However they are susceptible as a result of the presence of epistatic I gene.

Some intercrosses of susceptible hybrids produced resistant
plants. These results suggest segregation of the epistatic gene as well as the genes for resistance. Such a cross would be illustrated as follows:

\[
\text{Susceptible hybrid} \times \text{Susceptible hybrid} \\
\text{HRr} \times \text{HRr}
\]

Susceptible progeny: HHRR, HHRr, HRHR, HHRr, Hrr, hr and Hrr
Resistant progeny: iHRr and iHRr

As seen above, the red rot resistant plants do not possess the epistatic factor "I", and they possess the factor "R" for resistance. This occurred due to the segregation of the factors H and Rr. This explains the results obtained here in which red rot resistant plants were obtained among the progeny of susceptible x susceptible plants.

The results obtained in crosses between resistant x susceptible varieties can also be explained by the same hypotheses. The susceptible plants, as shown earlier, have different genotypes and most of them possess the epistatic dominant gene I. The various ratios obtained in these crosses would be attributed to the differences in the genetic constitution of the varieties entering the cross as well as to the irregularity of chromosome behavior during meiosis. As stated before the genes "RR" for resistance are located on the chromosomes of \textit{S. spontaneum}. The irregularity in behavior of \textit{S. spontaneum} chromosomes in the sugarcane varieties used here were explained earlier during the discussion of the mosaic studies.

The results obtained in the cross of the moderately resistant variety C. P. 29-103 with the susceptible variety C. P. 42-10 are given in table 51. It was observed that only 4 resistant seedlings were obtained among the 97 seedlings tested. The variety C. P. 42-10 was obtained from the cross Louisiana Purple x Honey. Louisiana Purple is a noble
cane, i.e., *S. officinarum*. The available information does not indicate whether the male parent "Honey" is another noble variety or is a sorghum variety. If sorghum was the male parent used in the cross, then variety C. P. 42-10 resulted from a contaminant pollen, since it does not look like a sorghum hybrid. The variety C. P. 42-10 shows a trace of mosaic in the field and the results obtained in the mosaic studies (table 24) indicate that this variety has *S. spontaneum* chromosomes in its inheritance. It is inferred that this variety resulted from the fertilization of a Louisiana Purple egg by a contaminant pollen that possessed *S. spontaneum* chromosomes. Thus variety C. P. 42-10 can be considered as an F₁ hybrid of *S. officinarum* and another cane variety. It should then possess one dominant epistatic factor "I" from its female parent and probably also another similar factor (I) from its pollen parent. If the hypotheses advanced earlier are correct, the progeny of the cross C. P. 29-103 x C. P. 42-10 will be susceptible to red rot. The results obtained here showed that only 4 seedlings of the progeny tested were resistant to red rot. These seedlings could be a result of self fertilization, pollen contamination or escapes. These results confirm the hypotheses advanced earlier.

Abbott 1938 (10) reported on the red rot reaction of seedlings obtained from three crosses involving resistant x susceptible varieties. A low percentage of resistant seedlings was obtained among the progeny. He was of the opinion that susceptibility to red rot was inherited as a partially-dominant character, and that the bringing together of two recessive factors resulted in the expression of resistance. Abbott's statement suggests that a resistant variety would be homozygous for the genes for resistance. In this case, a resistant variety would have homologous pairs of chromosomes. The *S. spontaneum* chromosomes
carry these genes, and so they would pair during meiosis and could be distributed regularly to opposite poles during anaphase. The gametes produced from resistant varieties would then carry the recessive gene for resistance, so in a cross of resistant x resistant varieties, all the progeny would be expected to be resistant to mosaic. The results obtained here do not substantiate such a hypothesis. The percentages of resistant seedlings were low in such crosses. For example, in the cross of the two resistant varieties C. P. 36-13 x C. P. 36-105 only 11 per cent of the progeny were resistant to red rot. This can be attributed to the loss of the *S. spontaneum* chromosomes carrying the genes for resistance during meiosis. Many of these gametes produced by such a variety would not possess these genes. This would not occur if the resistant varieties were homozygous for the genes for resistance, as Abbott claims. It is likely to occur if the resistant varieties were heterozygous for such genes, which agrees with the hypotheses advanced earlier and with the results obtained.

The crosses involving susceptible x susceptible parents produced red rot resistant seedlings. This could be attributed to resistance to red rot conditioned by complementary dominant factors. If this is the case, the F₁ hybrids of the cross *S. officinarum x S. spontaneum* should be resistant to red rot. However, these F₁ hybrids were observed to be very susceptible to red rot. Therefore, the possibility that resistance to red rot is conditioned by complementary dominant genes does not hold.

In examining the different seedlings, it was observed that in some cases the internodal tissues were susceptible to red rot, while the nodal tissues were resistant. This suggests that there is more than one type of resistance to this disease in sugarcane stalks. The types of
resistance to this disease observed or suggested by other investigators have been given in the review of the literature. In summary, resistance to lateral spread of the disease in tissues has been called physiological resistance, and the resistance to longitudinal spread of the fungus, was called nodal resistance by certain investigators or morphological resistance by others. The results obtained here showed that in some cases the disease spread longitudinally, producing a more or less narrow strip of diseased tissues running centrally along the internodes while the remaining parts of the internodes showed resistance to the disease. In a few cases it was observed that this lengthwise spread of diseased tissues progressed near the periphery of the stalk. In the cases in which the tissues were susceptible to lateral spread of the fungus, the degree of resistance to the disease varied. In some cases, the entire complement of internodal tissues was badly rotted, and in others, only a part or scattered spots of the internodes were diseased. This suggests that the degree of susceptibility of the internodal tissues varied, and is genetically conditioned. A group of minor modifying factors would explain differences in the degree of expression of the disease.

The degree of resistance of the nodal tissues to red rot also varied. In some cases, a narrow strip of diseased tissue could be seen extending longitudinally, and in other cases the disease spread laterally in the nodal tissues. The degree of resistance to lateral spread in the internodes also varied in the different seedlings. This suggests that this character is also genetically conditioned.

In many seedlings, especially those that have variety F. 31-962 as a parent, the internodal tissues became extensively necrotic, although
the nodal tissues were very resistant to the disease, and no red dis-
coloration whatever showed on them. This suggests that there are
genes conditioning resistance to red rot in the internodal tissues, and
other genes that condition resistance to this disease in the nodal
tissues. Such genes are apparently different (non-allelic).

The seedlings classified in the present studies as resistant
were those that showed resistance to spread of the fungus in the inter-
nodal tissues, which might be termed physiological resistance. In such
seedlings, the disease did not spread enough to reach the nodal tissues.
Therefore, the genes conditioning resistance discussed in these studies
were concerned with the resistance of the internodal tissues to the
disease. As was indicated earlier, the results suggest that the genes
conditioning internodal resistance are different from those involved in
the resistance of nodal tissues. It is obvious that a new technique should
be developed for the study of this latter type of resistance. The inocula-
tion techniques used now are not suitable for such tests. It would be of
interest to attempt the inoculation through the nodes and see the reaction
of the nodal tissues. Such inoculation can be done, for example, by
rubbing a spore suspension of the fungus on the buds, root primordia,
leaf scars and bud scales.

The seedlings that can be considered as commercially resistant
to red rot are those that fall in the categories designated before as re-
sistant and moderately resistant. By comparing the percentages of com-
mercially red rot resistant seedlings obtained in the progeny of the dif-
ferent crosses it appears that in general resistant x resistant crosses
were not superior to moderately resistant x resistant, resistant x
moderately resistant, resistant x susceptible, moderately
resistant x moderately susceptible, resistant x moderately susceptible
or even susceptible x susceptible crosses. It appears, therefore,
that a variety need not be discarded as a potential parent in the breeding
program because it is susceptible to red rot.

The results obtained and the conclusions stated earlier indicate
that if the variety has at least a trace of _S. spontaneum_ in its inheritance,
it did not appear possible to predict the value of a variety as a source
of resistance to red rot from its phenotype. The data also suggest that
because a variety is highly resistant to red rot is not proof that it will
be valuable as a source of resistance. The tendency for the _S._
_spontaneum_ chromosome or chromosomes carrying the gene or genes
for resistance to be lost during meiosis makes it entirely possible that
many of the gametes produced by such a variety will not possess the
gene for resistance.
SUMMARY

A total of 71,769 sugarcane seedlings from 175 crosses were inoculated with sugarcane mosaic virus using carborundum mesh 220 as an abrasive. Both mosaic resistant and susceptible plants were found among the progeny of all types of crosses. The percentages of plants showing mosaic symptoms in the progeny of the crosses involving resistant x resistant, susceptible x susceptible and resistant x susceptible parents ranged from 0.0 to 87.9, 2.6 to 35.6 and 0.8 to 94.5 per cent respectively.

From the data obtained the hypothesis is advanced that inheritance of resistance to mosaic disease of sugarcane is conditioned by dominant complementary genes. A group of minor modifying factors is interpreted as affecting the degree of expression of the disease. Irregularities in behavior during meiosis of S. spontaneum chromosomes in sugarcane varieties also influence the transmission of resistance from the parents to their progenies. The varieties used obtained their resistance to mosaic from Kassoer cane, S. spontaneum of Java (Glagah) and a form of S. spontaneum of India. The results obtained in the susceptible x susceptible crosses suggest that there is at least three complementary genes governing the resistance to mosaic.

The flat inoculation technique, in which the seedlings were sprayed with the mosaic virus, and then clipped off with shears immediately after spraying were compared with the pot technique in which the spindles were rubbed with a mixture of carborundum and the mosaic virus. About 20,282 seedlings obtained from 18 crosses were inoculated. The seedlings in the pots were older in age than those in the flats. In 12
crosses a much higher percentage of mosaic-susceptible seedlings were obtained by using the flat technique than by using the pot technique. In the other crosses there were almost no difference in the results obtained. The results suggest that the pot technique is preferable. Since in the flat techniques, although more rapid, many seedlings which would be resistant to mosaic in the field are unnecessarily discarded, thus eliminating many potentially desirable seedlings. The data also suggest that among the seedlings there are two types of resistance to mosaic, and that they are genetically controlled. The period of incubation of the mosaic disease varied from four days to seven weeks and perhaps more.

Seedlings from different crosses that showed mosaic symptoms in the greenhouse were transplanted to the field and periodic inspections were made. In August 29, 1951, it was found that in 1470 seedlings examined 19 per cent had recovered. A seedling was considered recovered when the mosaic symptoms disappeared from all the leaves of all the stalks. Recovery ranged from zero in a few crosses to 51.6 per cent in one cross. The results suggest that the recovery phenomenon in sugarcane seedlings is a genetic character. There is also a possibility of the presence of genes for tolerance to mosaic. The relation of these results to the techniques used in the sugarcane breeding program in Louisiana was discussed.

Studies were made for testing the reaction of progenies of 14 crosses of sugarcane to the red rot fungus Physalospora tucumanensis Speg. The crosses involved resistant x resistant, resistant x susceptible and susceptible x susceptible parents. Plants varying in their degree of resistance and susceptibility to red rot were obtained among the progenies.
of all types of crosses. The percentage of resistant plants in crosses between susceptible parents were higher than those obtained in certain crosses involving resistant parents. The hypotheses is advanced that resistance in sugarcane to red rot disease is governed by one or a few dominant genetic factors for resistance from *S. spontaneum* plus a dominant inhibitor genetic factor from *S. officinarum*. This inhibitor factor is thought to mask the effect of the factors for resistance from *S. spontaneum*. A group of minor modifying factors also influences the degree of expression of the disease. The irregular chromosomal behavior occurring in sugarcane varieties during meiosis influences the ratios of resistant to susceptible plants obtained in the progenies. The observations made suggest that the factors governing resistance in the nodes are different from those governing the resistance of the internodal tissues.

It is concluded that a sugarcane variety need not be discarded as a potential parent in breeding programs because of its susceptibility to mosaic, and/or red rot. Because it is resistant to one or both of these diseases is not a proof that it will be valuable as a source of resistance in a cane breeding program.


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Appendix I. Summation of the results of mosaic studies.

<table>
<thead>
<tr>
<th>Reaction of parents to mosaic</th>
<th>Number of crosses with indicated mosaic percentages in progenies</th>
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<tbody>
<tr>
<td></td>
<td>0-5%</td>
</tr>
<tr>
<td>R. x R.</td>
<td>26</td>
</tr>
<tr>
<td>R. x Tr.</td>
<td>14</td>
</tr>
<tr>
<td>R. x S.</td>
<td>8</td>
</tr>
<tr>
<td>Tr. x Tr.</td>
<td>-</td>
</tr>
<tr>
<td>Tr. S.</td>
<td>4</td>
</tr>
<tr>
<td>S. x S.</td>
<td>2</td>
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Appendix II. Parentage of most of the varieties used in these studies.

<table>
<thead>
<tr>
<th>Variety</th>
<th>Parentage</th>
</tr>
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<tbody>
<tr>
<td>C. P. 40</td>
<td>Okinawa Tekcha x F. M. #3</td>
</tr>
<tr>
<td>C. P. 670</td>
<td>U. S. 1484</td>
</tr>
<tr>
<td>C. P. 726</td>
<td>U. S. 1694</td>
</tr>
<tr>
<td>C. P. 1165</td>
<td>C. P. 670 (P.O.J. 213 F_2) as female</td>
</tr>
<tr>
<td></td>
<td>Male possibly U. S. 1694</td>
</tr>
<tr>
<td>C. P. 27-22</td>
<td>L. 74 x U. S. 785</td>
</tr>
<tr>
<td>C. P. 27-34</td>
<td>D. 74 x U. S. 1694</td>
</tr>
<tr>
<td>C. P. 27-35</td>
<td>D. 74 x U. S. 1694</td>
</tr>
<tr>
<td>C. P. 27-38</td>
<td>L. 74 x U. S. 1694</td>
</tr>
<tr>
<td>C. P. 27-108</td>
<td>P. O. J. 2364 x C. P. 726</td>
</tr>
<tr>
<td>C. P. 27-139</td>
<td>P. O. J. 2725 x U. S. 1694</td>
</tr>
<tr>
<td>C. P. 27-156</td>
<td>C. P. 40 x U. S. 1639</td>
</tr>
<tr>
<td>C. P. 28-11</td>
<td>Co. 281 x U. S. 1694</td>
</tr>
<tr>
<td>C. P. 28-19</td>
<td>Co. 281 x U. S. 1694</td>
</tr>
<tr>
<td>C. P. 28-44</td>
<td>Co. 281 x C. P. 27-38</td>
</tr>
<tr>
<td>C. P. 29-103</td>
<td>P. O. J. 2725 x C. P. 1165</td>
</tr>
<tr>
<td>C. P. 29-116</td>
<td>P. O. J. 2725 x C. P. 1165</td>
</tr>
<tr>
<td>C. P. 29-120</td>
<td>P. O. J. 2725 x C. P. 1165</td>
</tr>
<tr>
<td>C. P. 29-307</td>
<td>Co. 281 x P. O. J. 2878</td>
</tr>
<tr>
<td>C. P. 29-320</td>
<td>Co. 281 x C. P. 27-34</td>
</tr>
<tr>
<td>C. P. 30-24</td>
<td>C. P. 27-22 x C. P. 1165</td>
</tr>
<tr>
<td>C. P. 31-288</td>
<td>Co. 281 x U. S. 1694</td>
</tr>
<tr>
<td>C. P. 31-432</td>
<td>C. P. 29-307 x P. O. J. 2878</td>
</tr>
<tr>
<td>C. P. 31-511</td>
<td>P. O. J. 2364 x Co. 290 ?? ??</td>
</tr>
<tr>
<td>C. P. 33-224</td>
<td>C. P. 27-139 x C. P. 31-432</td>
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</table>
## Appendix II. Continued.

<table>
<thead>
<tr>
<th>Variety</th>
<th>Parentage</th>
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<tbody>
<tr>
<td>C. P. 33-229</td>
<td>C. P. 27-139 x C. P. 31-432</td>
</tr>
<tr>
<td>C. P. 33-372</td>
<td>C. P. 1165 x C. P. 28-44</td>
</tr>
<tr>
<td>C. P. 34-120</td>
<td>Co. 281 x P. O. J. 2878</td>
</tr>
<tr>
<td>C. P. 36-13</td>
<td>P. O. J. 2725 x Honey Sorghum?</td>
</tr>
<tr>
<td>C. P. 36-105</td>
<td>Co. 281 x C. P. 1165</td>
</tr>
<tr>
<td>C. P. 36-138</td>
<td>Crystalina x 28 NG 251, Imp. 496 (S. robustum)</td>
</tr>
<tr>
<td>C. P. 36-158</td>
<td>P. O. J. 2725 x 28 NG 251</td>
</tr>
<tr>
<td>C. P. 36-187</td>
<td>P. O. J. 2725 x C. P. 1165</td>
</tr>
<tr>
<td>C. P. 36-211</td>
<td>P. O. J. 2725 x Honey</td>
</tr>
<tr>
<td>C. P. 38-12</td>
<td>P. O. J. 2725 x C. P. 1165</td>
</tr>
<tr>
<td>C. P. 38-27</td>
<td>Co. 281 x C. P. 1165</td>
</tr>
<tr>
<td>C. P. 38-34</td>
<td>Co. 421 x C. P. 27-156</td>
</tr>
<tr>
<td>C. P. 38-41</td>
<td>C. P. 29-116 selfed</td>
</tr>
<tr>
<td>C. P. 42-10</td>
<td>La. Purple x Honey</td>
</tr>
<tr>
<td>C. P. 42-13</td>
<td>P. O. J. 2878 x C. P. 28-11</td>
</tr>
<tr>
<td>C. P. 43-64</td>
<td>C. P. 36-138 x C. P. 27-108</td>
</tr>
<tr>
<td>C. P. 43-74</td>
<td>Co. 281 x C. P. 1165</td>
</tr>
<tr>
<td>C. P. 44-55</td>
<td>Co. 281 x C. P. 27-34</td>
</tr>
<tr>
<td>C. P. 44-101</td>
<td>Co. 281 x C. P. 1165</td>
</tr>
<tr>
<td>C. P. 44-126</td>
<td>C. P. 33-229 x C. P. 33-224</td>
</tr>
<tr>
<td>C. P. 44-150</td>
<td>C. P. 36-158 x P. 33-29</td>
</tr>
<tr>
<td>C. P. 44-154</td>
<td>Co. 281 x U. S. 2694</td>
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<tr>
<td>C. P. 44-155</td>
<td>C. P. 33-229 x C. P. 33-224</td>
</tr>
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<td>C. P. 44-156</td>
<td>Co. 281 x C. P. 27-108</td>
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</table>
### Appendix II. Continued.

<table>
<thead>
<tr>
<th>Variety</th>
<th>Parentage</th>
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<tbody>
<tr>
<td>C. P. 45-184</td>
<td>C. P. 29-103 x 1322A-38</td>
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<tr>
<td>C. P. 46-73</td>
<td>C. P. 34-120 x C. P. 30-24</td>
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<td>C. P. 46-193</td>
<td>C. P. 33-229 x C. P. 33-224</td>
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<tr>
<td>C. P. 46-199</td>
<td>Co. 281 x C. P. 1161</td>
</tr>
<tr>
<td>C. P. 46-201</td>
<td>C. P. 33-229 x C. P. 33-224</td>
</tr>
<tr>
<td>C. P. 47-43</td>
<td>C. P. 33-229 x C. P. 27-108</td>
</tr>
<tr>
<td>C. P. 47-68</td>
<td>C. P. 34-120 x C. P. 27-108</td>
</tr>
<tr>
<td>C. P. 47-120</td>
<td>C. P. 34-120 x C. P. 27-108</td>
</tr>
<tr>
<td>C. P. 48-53</td>
<td>C. P. 34-120 x C. P. 36-211</td>
</tr>
<tr>
<td>C. P. 48-106</td>
<td>P. O. J. 2961 x S. C. 12-4</td>
</tr>
<tr>
<td>C. P. 48-116</td>
<td>C. P. 36-105 x C. P. 38-34</td>
</tr>
<tr>
<td>C. P. 48-126</td>
<td>C. P. 36-105 x C. P. 38-34</td>
</tr>
<tr>
<td>C. P. 48-156</td>
<td>C. P. 38-27 x C. P. 33-372</td>
</tr>
<tr>
<td>C. P. 49-9</td>
<td>P. O. J. 2961 x S. C. 12-4</td>
</tr>
<tr>
<td>C. P. 49-49</td>
<td>634-40 x C. P. 36-211</td>
</tr>
<tr>
<td>Co. 206</td>
<td>Ashy Mauritius x <em>S. spontaneum</em> (India)</td>
</tr>
<tr>
<td>Co. 213</td>
<td>Kansar x P. O. J. 213</td>
</tr>
<tr>
<td>Co. 281</td>
<td>P. O. J. 213 x Co. 206</td>
</tr>
<tr>
<td>Co. 285</td>
<td>Striped Mauritius green Sport x Co. 206 or 205</td>
</tr>
<tr>
<td>Co. 356</td>
<td>P. O. J. 2725 x Sorghum Durra Stape</td>
</tr>
<tr>
<td>Co. 421</td>
<td>P. O. J. 2878 x Co. 285</td>
</tr>
<tr>
<td>D. 74</td>
<td>White Transparent</td>
</tr>
<tr>
<td>E. K. 2</td>
<td>Lahina x Fidji</td>
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</table>
Appendix II. Continued

<table>
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<th>Variety</th>
<th>Parentage</th>
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<tr>
<td>E. K. 28</td>
<td>E. K. 2 x P. O. J. 100</td>
</tr>
<tr>
<td>F. 31-490</td>
<td>P. O. J. 2725 x C. P. 27-35</td>
</tr>
<tr>
<td>F. 31-962</td>
<td>Co. 281 x C. P. 27-108</td>
</tr>
<tr>
<td>F. 36-273</td>
<td>F. 31-490 x C. P. 30-24</td>
</tr>
<tr>
<td>F. 36-819</td>
<td>F. 31-962 x P. O. J. 2878</td>
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<tr>
<td>F. 40-96</td>
<td>F. 31-962 x F. 36-273</td>
</tr>
<tr>
<td>H. 32-8560</td>
<td>Co. 213 x P. O. J. 2878</td>
</tr>
<tr>
<td>Imp. 1183</td>
<td>Saccharum spontaneum L.</td>
</tr>
<tr>
<td>Kassoeer</td>
<td>Black Cheribon x S. spontaneum (Java)</td>
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<tr>
<td>28 NG 39-491</td>
<td>From New Guinea, parents unknown</td>
</tr>
<tr>
<td>28 NG 72-514</td>
<td>From New Guinea, parents unknown</td>
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<td>P. 33-29</td>
<td>P. O. J. 2878 x Badila</td>
</tr>
<tr>
<td>P. 33-32</td>
<td>P. O. J. 2878 x Badila 7?</td>
</tr>
<tr>
<td>P. 33-37</td>
<td>Supposedly from P. O. J. 2878 x Badila, but it looks like a self.</td>
</tr>
<tr>
<td>P. O. J. 100</td>
<td>Bandjarmasin Hitam x Loethers</td>
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<tr>
<td>P. O. J. 213</td>
<td>Black Cheribon x Chumee (S. barberi)</td>
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<tr>
<td>P. O. J. 2364</td>
<td>Kassoeer x P. O. J. 100</td>
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<td>P. O. J. 2725</td>
<td>E. K. 28 x P. O. J. 2364</td>
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<td>P. O. J. 2878</td>
<td>P. O. J. 2364 x E. K. 28</td>
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<tr>
<td>S. C. 12-4</td>
<td>Ba 6835 x Ba 4578</td>
</tr>
<tr>
<td>U. S. 785</td>
<td>Kassoeer</td>
</tr>
<tr>
<td>U. S. 1484</td>
<td>P. O. J. 213</td>
</tr>
<tr>
<td>U. S. 1694</td>
<td>P. O. J. 213</td>
</tr>
<tr>
<td>36-46</td>
<td>C. P. 43-64 x C. P. 33-372</td>
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Appendix II. Continued

<table>
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<tr>
<th>Variety</th>
<th>Parentage</th>
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</thead>
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<tr>
<td>88-45</td>
<td>P. O. J. 2725 x C. P. 1165</td>
</tr>
<tr>
<td>634-40</td>
<td>P. O. J. 2725 x C. P. 31-288</td>
</tr>
</tbody>
</table>
Plate I. Mosaic on leaves of hybrid varieties.

Fig. 1 and 2. Seedlings of the 1949-crosses.

Fig. 3 and 4. Seedlings of the 1950-crosses.
Plate II. General view of the seedlings in the greenhouse
Plate II
Plate III. Red rot on different hybrids

Fig. 1. A resistant variety (top) compared to susceptible varieties that show red rot infection in both the nodes and the internodes.

Fig. 2. A resistant variety (top) compared to varieties that showed nodal resistance and internodal susceptibility to red rot.

Fig. 3. Seedlings of the 1948-crosses showing different degrees of susceptibility.
Plate IV. Red rot on different hybrids.

Fig. 1 and 2. Seedlings of the 1949-crosses showing various types of reaction to red rot.
AUTOBIOGRAPHY

Youssef Elsedik Azab was born on July 24, 1923, at Zagazig, Egypt. He graduated from Zagazig Primary School in 1934. In the same year he entered Zagazig Secondary School, and later received the "High School General Certificate" and the "High School Orientation Certificate, Science Division" offered by the Egyptian Ministry of Education. In 1944, he obtained the Bachelor of Science degree from the College of Agriculture, Fuad First University, Egypt. Later, he was appointed instructor in the same college and in November 1946, was sent to undertake six years of graduate study and practical training in the United States of America. In 1947, he started his studies at the University of California, where, in January 1949, he obtained the Master of Science degree in Agronomy. In July of the same year he entered Louisiana State University. He is a member of The American Society of Sugarcane Technologists, The International Society of Sugarcane Technologists, The American Phytopathological Society, The American Society of Agronomy, The Louisiana Academy of Science, and Sigma-Xi honorary research organization and is now a candidate for the Doctor of Philosophy degree.
Candidate: Youssef Azab
Major Field: Botany & Plant Pathology
Title of Thesis: Inheritance of resistance in sugarcane to mosaic and red rot diseases.

Approved:

[Signatures]

Major Professor and Chairman
Dean of the Graduate School

EXAMINING COMMITTEE:

[Signatures]

Date of Examination:

April 15, 1952