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MOSAIC OF SUGARCANE IN THE UNITED STATES, WITH SPECIAL REFERENCE TO

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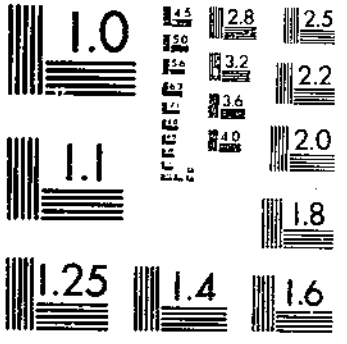

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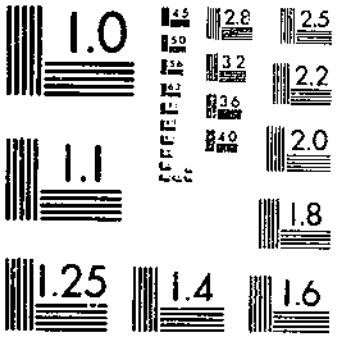

MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS-1963-A

Technical Bulletin No. 955 • 1948

UNITED STATES
DEPARTMENT OF AGRICULTURE
WASHINGTON, D. C.

Mosaic of Sugarcane in the United States, With Special Reference to Strains of the Virus[1]

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[2] The field and greenhouse experiments were performed, for the most part, by E. M. Summers under the general guidance of R. D. Rands, who also supplied most of the photographs. The first section of the paper reviewing previous work and integrating the study with the entire sugarcane mosaic project was prepared by E. W. Brandes.

MOSAIC of sugarcane, a typical virus disease of the leaf-mottling type, was undoubtedly a major cause of the decline of the Louisiana sugar industry—almost to the vanishing point—during the period 1923–26, when sugar production of the State dropped to 47,000 tons. Mosaic has been a serious problem at one time or another in practically every country that produces sugarcane.

The results have exceeded expectations in developing resistant varieties, but there still remains much to be done in incorporating into desirable combinations the breeding material now assembled. Also, more intensive exploration and collection is needed to provide the full natural range of plant characteristics in sugarcane-breeding material.

The production of mosaic-resistant varieties is one of the main objectives in the cooperative varietal improvement program of the Division of Sugar Plant Investigations of the United States Department of Agriculture, the American Sugar Cane League, and the Louisiana Agricultural Experiment Station. In view of the susceptibility of the parent varieties that must be used to obtain commercial qualities it is necessary in obtaining a few worthy of agronomic trial to test many thousands of seedlings for mosaic susceptibility. Many large progenies of individual crosses have been tested. These data have furnished valuable information to the cane breeder, and distinct progress has been made in producing resistant varieties.

This bulletin presents largely data from studies of strains of the mosaic virus, their effect on different varieties of sugarcane, and other phases of the disease, and is summarized below.

SUMMARY

The epidemic spread of sugarcane mosaic in Louisiana, between the time of the discovery of the disease in 1919 and the low point of sugar production for the State in 1926, was the main factor in finally eliminating varieties of *Saccharum officinarum* L. from the canefields of Louisiana and the substitution of species hybrids. The cumulative effects of root rot and red rot during previous years had already set the stage for elimination of the *S. officinarum* varieties.

Temporary relief was provided by the mosaic-tolerant P. O. J.[3] varieties, which were in turn replaced by the more resistant Co. and C. P. varieties. The resistance of presently grown commercial varieties is such that mosaic has become only a minor problem in field practice.

Sugarcane mosaic has a wide host range among both cultivated and wild grasses. Within the genus *Saccharum* the range is from complete susceptibility to apparent immunity. In other genera, mosaic has been observed or experimentally transferred to 38 species of wild and 10 of cultivated grasses. Two new hosts (*Erianthus giganteus* (Walt.) Muhl. and *Panicum reptans* L.) are reported. It is noted that in Louisiana, with possible rare exceptions, mosaic in other grasses does not occur except in proximity to infected sugarcane.

Wide variation in rate of mosaic spread to healthy sugarcane is noted between varieties and from year to year in the same variety. For the varieties and conditions prevailing during these studies the

[3] See *footnote 1 of table 1, p. 27, for designation of abbreviations.*

rate of spread was much greater to adjacent healthy plants in the same row than to those in adjoining rows at a distance of 6 feet. This emphasizes the limited movement of the vectors responsible for most of the secondary spread.

The proved vectors of sugarcane mosaic are *Aphis maidis* Fitch, *Hysteroneura setariae* (Thos.), and *Toxoptera graminum* (Rond.).

The separation of seven strains and three substrains of the virus is demonstrated. They are identified largely by symptom expression on three differential host varieties. A key and chart are presented.

Wide differences in symptom patterns occur on many varieties. A large number of these were tested in extensive inoculation experiments. It was possible to determine two to five distinct patterns of mosaic symptoms on individual varieties. Quantitative differences in the ability of different strains of the virus to produce infection as a result of artificial inoculation were apparent. These differences served to confirm the authenticity of the different strains.

Surveys were made to determine the distribution of the mosaic strains in Louisiana and, to some extent, in other Southern States. Strains B and D were shown to predominate in the sugarcane-for-sugar production area of Louisiana; none of the other strains was collected commonly on the P. O. J. or Co. varieties; strain E was predominant in the rest of Louisiana where sugarcane is grown for sirup production and in adjoining States where small sirup patches consisting mainly of noble canes comprised the total sugarcane acreage.

A hypothesis to account for a succession of virus strains in Louisiana, based on varietal susceptibility and the results of this survey, is offered.

Recovery from mosaic of certain sugarcane varieties is demonstrated. Extensive data are presented upon the different types of recovery with possible explanations and interpretations.

HIGH LIGHTS OF SUGARCANE MOSAIC [4] AND THE CURRENT DEVELOPMENT OF RESISTANT VARIETIES

Theories of Causation

Mosaic was first reported as a sugarcane abnormality by Van Musschenbroek (*53*) [5] in Java in 1892, and thus was among the first noted of the destructive plant diseases later proved to be caused by a virus. In contrast with such virus diseases as tobacco mosaic, which is readily transferred from diseased to healthy plants by artificial means, sugarcane mosaic was not readily transferred by the artificial means then employed. In retrospect, the conclusion reached by early investigators that mosaic was noninfectious is understandable. The requirements for obtaining a high percentage of successful arti-

[4] A comprehensive review of the 1,000 or more papers on sugarcane mosaic that appeared during the world-wide epidemic beginning about 1915 is obviously impracticable, and because much of the great volume of literature is repetitious or extraneous a complete review would be more confusing than instructive. This narrative account seeks to high light the principal research that has advanced knowledge of the disease and to tie together related research in other biological fields that is found to be indispensable to the development of adequate control measures.

[5] Italic numbers in parentheses refer to Literature Cited, p. 121.

ficial infections are exacting. Above all, interference with tests of artificial inoculations by natural infections of control plants through the agency of the abundant but unsuspected insect vector *Aphis maidis* Fitch was confusing. The same circumstances, including careless use of uncontrolled test plants, some of which doubtless were already in the incubation stage of the disease, nullified the efforts of many investigators in other countries at much later dates.

Many unsatisfactory attempts to communicate the disease to healthy plants by inoculation led investigators in Java to conclude that the abnormality was a frequently recurring bud variation. After years of study, dissident investigators in the same country pronounced the disorder genetic, manifesting the hybrid nature of attacked varieties. Roguing and other field sanitation measures were employed, however, to avoid multiplication of the undesirable variant. These practices were followed more or less successfully but at great expenditure of labor, not only in Java but in Formosa, where the disease was also a serious problem.

In the decade 1910–20 many reports of serious damage by mosaic were made in Egypt, Hawaii, and other sugarcane-producing countries in which the disease had been established by the practice of introducing new varieties, and speculation on the nature of the abnormality was revived. In the fresh attack on the problem, mosaic was again ascribed to numerous causes by different investigators. On the basis of field observations of epidemiology and a few insufficiently controlled inoculation tests, some investigators asserted but without proof that it was an infectious disease. Aside from (1) reports of the disease and its rapid spread and destructiveness in new areas, (2) circumstantial evidence on its infectiousness, and (3) some good negative evidence ruling out wrong hypotheses on the cause, no advance in knowledge of mosaic was recorded.

Beginning about 1916 in Puerto Rico and shortly thereafter in Cuba, all manner of claims the reverse of conservative were being made to account for the alarming development and spread of the disease. Mysterious cell inclusions were found in mosaic cane and incautiously were promoted to importance as possible pathogens. Bud variations, segregates in hybrid progenies, bacteria, plasmodia, and other kinds of organisms, perverted enzymes, bad weather, depleted soils, plant exhaustion from excessive ratooning, faulty manuring practices, and poor cultivation, and finally that refuge of the completely destitute, deterioration, or "running out," of varieties all had their advocates. Spread of the disease was attributed to wind-borne infection, poisonous exhalations, pollution of soils, infected cane knives and other tools, unnamed insects, and other means. Some of these pronouncements were made by untrained observers, but they were published in reputable journals and thus added to the confusion. In the welter of published speculation on the cause and spread of mosaic it was inevitable that some ideas closer to the facts should be championed. Lacking proof, they, as well as the less conservative ideas, became confessions of failure. In a review published in 1921 by C. A. Barber (*6*), English mycologist and cane variety expert, the evidence on the cause of the disease was dismissed in these words:

The very complete study to which it [mosaic] has been subjected has knocked away these props one by one, till it is generally conceded that to no single one or combination of them can the disease by any chance be attributed, and there is nothing left to fall back upon but hypothesis.

Proof of Infectiousness

In respect to elucidation of the basic disease phenomena by acceptable scientific methods, mosaic remained befogged by vain evidence and conflicting opinions for 30 years. Even in their elementary traits, virus diseases were obscure and perplexing before 1920 and no clear evidence or indisputable proof of the essential disease characteristics was adduced until Brandes (*7*, *8*, *9*, *22*) in 1919 began a series of studies on the nature and manner of spread of mosaic and on methods of controlling it. These studies served to guide further original research by other investigators, and most subsequent work on mosaic was noticeably improved. Thus directed toward a single purposeful objective, the scientific approach to this complex disease problem eventually brought to be borne upon it the wide range of resources of plant pathology and other plant sciences.

A survey of the mosaic epidemic enveloping the cane areas of Puerto Rico, Cuba, and Louisiana in 1919 led Brandes to believe that conclusive demonstration of the increasingly obvious infectiousness of mosaic could be conducted only at some place far from the reservoirs of infection of the commercial cane areas. In this situation it was decided to conduct disease-transmission tests far removed from the raging epidemic. This precaution, however logical, seems to have been without precedent. The tests were made near Washington, D. C., several hundred miles from any known natural source of infection, and were conducted with extraordinary control of test plants and other conditions of the experiments.

The test plants used for inoculation were known to be from healthy stock grown in local greenhouses for more than 6 years. Cuttings from diseased plants in Puerto Rico were established in an insect-proof "pest" greenhouse at Garrett Park, Md., about 14 miles away. The first critical tests were made by transporting the pedigreed healthy plants from Washington to the pesthouse at Garrett Park, and in all subsequent tests the healthy plants used for inoculation by expressed juice and insects were kept separated from any other source of inoculum by growing the diseased plants in another screened greenhouse. The healthy stock was propagated in a third screened greenhouse.

Under these conditions artificial transmission of mosaic from diseased to healthy plants by a number of different techniques was demonstrated. In the successful experiments 10 to 80 percent of test plants became infected.

A discovery of great significance was that the disease was readily transmitted by the previously unsuspected natural insect vector *Aphis maidis*. Because it had been reported by Van Deventer (*36*) in 1906 as feeding on sugarcane in Java and was known to be practically ubiquitous, this insect was fortunately included among the available insects tested. Up to that time the aphid had not been regarded as a sugarcane pest.

Aphis maidis is a small, inconspicuous insect. Only occasionally does it migrate in large numbers to sugarcane from other preferred grasses. During the infrequent visits to cane it colonizes between the tightly rolled youngest leaves at the apex of the stem and even when abundant is easily overlooked. Many observers have testified that they have searched persistently for *A. maidis* on sugarcane for months without success. For that reason some insect specialists asserted that *A. maidis* could not in any way be considered responsible for the transmission of the disease in the field. With more intensive study, however, others became convinced that the association of the insect with sugarcane, although obscure, is more prevalent than had been supposed and could account for all the observed natural transmission. A brilliant investigation in 1927 by Van Breemen in Java (*31*) on the swarming of *A. maidis* showed that several long, migratory flights of winged females occurred within a year and could account for outbreaks of mosaic remote from an infested area.

It had been pointed out in the earlier research establishing the fact of this specialized relationship between the plant and the insect that sugarcane is a perennial plant and that typically, as cultivated in most countries, the individual stools are vulnerable to attack by the insect for a period of 3 to 10 years; also that a single feeding by a viruliferous aphid will cause infection; that once infected, vegetative propagation by stem cuttings from the stool helps to spread and perpetuate the disease; and, finally, that of all insects tested, *A. maidis* is the only one common to all the numerous countries and areas where the epidemic was raging.

In the series of related studies in Washington 13 species of wild and cultivated grasses were proved susceptible to sugarcane mosaic by inoculation tests with viruliferous aphids. Some of the grasses were common weed pests of sugarcane fields known to harbor the insect vector, a disquieting development because of the potential acceleration of infections and the increased difficulty of controlling them. During 3 years of virus transmission tests in 1919–21 involving thousands of plants, not one case of mosaic developed in the control plants.

One of the significant observations made in this period of the investigation was that infected sugarcane, corn, sorghum, and wild grasses occasionally recovered from mosaic with no association of any obvious change in environmental or other conditions.

Acute Need for Practical Control

Confirmation and extension of the basic facts by others quickly followed the publication by Brandes of his evidence of infectiousness, recovery, natural transmission of the virus by an insect vector, and of the existence of weed hosts. By 1923 the original evidence and confirmatory reports served to allay in some measure the near hysteria that had developed among commercial planters and the professional scientific guardians of the industry. 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 trials of the vexed planters,

however, were destined to be long-continued. Countermeasures were only dimly envisioned by the majority of professional investigators.

As in the questions of the cause and spread of the disease, so also in the important question of control, similar divergence of opinion was manifested and consequently there was confusion compounded on confusion. This somewhat delayed the beginning of recovery and reconstitution of the canefields, a process that by its nature required a long period of years. The technical workers responsible for solving the question of control recognized the urgent need for taking quick steps to alleviate the destruction. Some made suggestions based on conceptions that in later and calmer moments they themselves doubtless would have recognized as inept and futile. In extenuation of some of the precipitate and supposititious recommendations it must be remembered that the decade of 1916–26 was punctuated by a succession of financial failures of many long-established farms and large plantations. In Louisiana toward the end of that period yields of sugarcane had dropped to such pitifully low levels that the banks refused to risk further financing of what they regarded as a permanently collapsed industry. In Brazil a large prize was announced by a State government for the individual who could devise a "cure" for sugarcane mosaic, and the temper of the people most concerned in the drama everywhere could be likened to those whose means of subsistence had been snuffed out in a stock market crash with, unfortunately, the same quota of suicides.

Among the numerous palliatives, stopgaps, and hoped-for controls suggested were roguing, abandoning of ratooning, rotation with legumes, better cultivation, irrigation, potash application to the soil as a therapeutic (*14*), prophylactic inoculations of the cane, attempts to segregate resistant strains of sugarcane from the diseased popular varieties (since cane varieties are clones, this was a revival of the bud-mutation theory in reverse), and the use of immune or resistant varieties. Sugarcane experts in Hawaii, Argentina, and other far countries suggested remedial measures for use in North America. Defeatists, including a few officials responsible for agricultural guidance in the southern United States, drew attention to weather and concomitant adversities of cane farming generally chargeable to the one-crop idea and demanded better use of the sugarcane area by crop diversification.

Refusing that type of guidance, a nucleus of competent authorities among the sugarcane industry leaders in Louisiana and the Louisiana State University became convinced that the industry could be saved and eventually reconstituted by a two-phase substitution of resistant cane varieties suggested by specialists of the United States Department of Agriculture. Plant materials for the initial phase were supplied them in 1921, and they resolutely set about the long complicated task of accomplishing the job.

The program suggested by Department specialists for consideration by the farm leaders contemplated a revolutionary change in character of the plants to be grown for sugar, substituting hybrid canes partly of Temperate Zone origin for the canes of Tropical Zone origin that had been grown exclusively for 150 years. The plan entailed more

or less drastic modification of field and factory practices. The hybrids suggested for the first phase of variety replacement were represented as not fully tested for adaptation to local conditions but were far more tolerant of the mosaic infection than the varieties being grown, and moreover they were immediately available, having been produced (and discarded) in Java many years previously. A small supply of the varieties was on hand, having passed through the required period of quarantine in greenhouses near Washington.

On the promise of similar but better adapted new varieties, already in process of development locally (for the second phase of variety replacement), the group of farm leaders agreed to plant the suggested varieties, to give them the best of care, and, if they appeared to confirm the forecast of superior performance in their disease-saturated fields, to multiply them to hundreds of thousands of tons of seed cane as rapidly as possible. The plant materials supplied by the United States Department of Agriculture in the winter of 1921-22 (*54*) comprised several cuttings each of the varieties P. O. J. 36, 213, and 234. P. O. J. 36 is a cross of Striped Preanger, a tropical variety, with Chunnee, a slender Temperate Zone variety of northern India. P. O. J. 213 and 234 are crosses of Black Cheribon (the old standard cane of Louisiana known there as Louisiana Purple) with Chunnee.

This shipment of a few cane cuttings from Washington proved to be the beginning of the rebirth of an industry. It was not delivered without birth pangs, the first of which were shock and bitter disappointment at the unprepossessing appearance of the slim, high-fiber varieties, which broke the mills on the first run. Two years later, however, the offer of 4-pound packages of seed pieces of P. O. J. 234 brought nearly 2,300 urgent applications, of which all but a very few were filled before the available supply gave out.

It is necessary to go back in time from the period just discussed to 1910 to trace the utilization of ideas and facts in the breeding of mosaic-resistant varieties adapted to particular environments. The first positive advance toward the objective is attributable to observations and research contemporary with the first advances in knowledge of the essential disease phenomena, also beginning in 1919 (*7*, *22*), but it was foreshadowed about 1910 by observations made at the Proefstation Oost Java on the pronounced difference in the tendencies of the several varieties of cane to produce yellow-stripe (mosaic) individuals. Differences in the severity of damage suffered by different varieties also were noted. Fields of the Cheribon × Chunnee hybrids, identical in parentage with P. O. J. 36, 213, and 234, recommended to Louisiana planters by the United States Department of Agriculture in 1919-21, were noted in Java to show the abnormality even up to 100 percent, but injury caused by the disorder in the individual plants was reported to be mild.

A mass of similar data on hybrid varieties of other parentage also was presented but with no evidence of attempts to classify the data or to generalize on the genetic sources of the differences noted in their resistance and susceptibility. At that time the station was engaged in sugarcane breeding on a large scale to develop varieties that could be grown in the lowland valleys and coastal plain of Java without re-

sorting to the expensive production of seed cane in mountain nurseries.

Another mysterious cane disease of unknown cause, sereh, had made it impossible to grow cane on sea-level plantations with cuttings from cane grown there. A satisfactory 1-year crop could be grown, however, with seed cane from the mountain nurseries where sereh did not exist, or was far less destructive. Sereh also was regarded by the Dutch investigators as a manifestation of bad variation. Ratooning was unprofitable because of sereh, but it was discovered that one satisfactory plant-cane crop could be produced in the lowlands, after which it was necessary to renew the seed cane supply from the mountains. Obviously this was too expensive.

Strenuous efforts were made to develop seedling varieties that would relieve the planters of the necessity for maintaining this complicated system of mountain nurseries. The station staff was not seeking new varieties resistant to sereh and mosaic in the accepted meaning of resistance, but "stable" varieties that would not produce these undesirable "mutants". Fortunately, included among the parent varieties used to produce hybrid seedlings was the variety from northern India, Chunnee. This variety, which belongs to the species *Saccharum barberi* Jesw., had, in fact, real resistance to the virus causing mosaic. A measure of resistance was conferred upon its hybrid seedlings bred in Java. Some of these, the P. O. J. 36, 213, and 234 previously mentioned, are the varieties that attracted attention more than a decade later in the New World, where the true nature and value of their resistance to mosaic was recognized (7).

The simple explanation for more abundant and locally concentrated mosaic "mutants" in the highlands of Java was later offered by the findings of Brandes, namely, the more grassy condition of the fields under the higher rainfall conditions, promoted secondary infection by exchange between cane and weeds of the insect vector *Aphis maidis*, which abounded in the susceptible weed hosts. The sporadic occurrence of diseased plants in the drier, less grassy lowlands, chiefly of primary infections (from infected seed pieces) and ordinarily with less secondary spread, was also made clear. However askew the main premise in the earlier studies in Java may have been, it is recognized that important distinctions were made before 1910 in the extent and severity of mosaic present in different varieties of cane.

Although the possibility of using resistant varieties of sugarcane was recognized in 1910 [19][6] as the most satisfactory control measure and was advocated by a number of investigators in several countries, the complexities of the job of providing really acceptable resistant varieties adapted to the wide range of conditions in different localities were underestimated. The several varieties confirmed in the decade under reference as more or less resistant were unfamiliar as com-

[6] In 1919, F. S. Earle mistakenly announced in an unpublished manuscript abstracted by C. O. Townsend (*69*) that the variety Kavangire (Cuba) is immune from mosaic. The variety belongs to the species *Saccharum sinense* Roxb., which in its different varietal forms shows extraordinary tolerance of the mosaic virus and is difficult to infect. The field evidence of immunity was generally accepted as valid by other investigators in 1919, but P. A. Yoder (*71*) later found Kavangire naturally infected.

mercial varieties and were not considered well enough adapted to be regarded as satisfactory answers to the problem. The search for other resistant varieties, moreover, appeared to be from a too restricted point of view, entirely without consideration for the genetics involved in the hybrid commercial cane varieties or for those arising from the equally complex virus.

Perhaps owing to the long practice of cloning sugarcane and the general uniformity of plants in the field, the general tendency at that time was to regard individual cane varieties as more or less dissociated, indivisible entities rather than as complex aggregates, perhaps with resistant and susceptible components traceable to basic sources. Until later, even specialists who published on cane varieties did not classify them genetically, or, with one or two outstanding exceptions (*5*), morphologically, but merely described individual varieties. For the most part, therefore, the direct search for resistant varieties was oversimplified and as unimaginative as a hunt for a lost penny. The known facts about varietal resistance were unrelated.

Steps in Developing Resistant, Adapted Varieties

In one country at least the dimensions of the problem of control by resistant varieties were more fully appreciated. In 1919 justifications were presented to the Government of the United States for adequate appropriations of funds to conduct a project of breeding resistant varieties adapted to conditions in the several American sugar-producing areas, including Puerto Rico. The project was approved, and a beginning was made in that year by surveying parts of Florida for areas that might be found suitable for normal development of sugarcane flowers, the cardinal requisite for breeding. The tropical forms of sugarcane do 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 destined for mosaic resistance breeding (*10*, *24*, *29*). In 1920 additional plantings were made at a site selected on the shore of Lake Okeechobee, near Canal Point, a name designating the lake terminus of a drainage canal that flows eastward 40 miles to the Atlantic Ocean.

Seedlings were produced in the winter of 1919–20 from varieties of the original planting. In these earliest attempts to produce resistant varieties recognition was given to the importance of relationships and classification of parent varieties and to the mass responses of related forms in respect to mosaic resistance or susceptibility (*6*, *22*, *24*). This represented a new departure in theory and practice of breeding sugarcane for disease resistance and eventually led to the second phase of positive advances in coping with mosaic—the broad concept of generic study of host-virus relationships. This study later was to become complicated by the discovery of different strains of the virus in 1934 (*63*), of which there had been prior tokens as early as 1923 (*22*).

The extensive host-virus study began with a project to test the reaction to mosaic virus of the so-called "original" varieties of sugar-

cane and their wild prototypes,[7] a task of such magnitude that after 25 years it is uncompleted. The practical results of the work, however, began flowing as it progressed. The project involved (1) undertaking many plant explorations over a large part of the immense and largely primitive area of the world where sugarcane is indigenous (*18, 28*); (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 practices (*23*) and quarantine facilities (*12*); (4) developing methods for rapid multiplication of sugarcane (*11*); (5) establishing a world reference collection of sugarcane varieties (*15*), which for security against disease or other losses of the valuable material is maintained in the Panama Canal Zone as well as in the United States (the cooperative companion collection at Djambagedeh, Java, is now reported destroyed by war); (6) continually improving techniques for large-scale inoculations (*4*); and many other details.

During the progress of this phase of mosaic study it became apparent that the hoped-for development of commercial cane varieties immune to mosaic probably would have to be abandoned. Representative selections of varieties of the tropical species *Saccharum officinarum* and the Temperate Zone *S. sinense* were proved to be susceptible (*22, 24*); as also were similar ample samplings of varieties and races of the Temperate Zone *S. barberi*, the tropical and temperate *S. spontaneum* L., and the tropical *S. edule* Hassk.; and, finally, representatives of the wild tropical species *S. robustum* Brandes and Jeswiet ex Grassl (*41*). The species of *S. officinarum*, as generally understood by the older botanists, may itself comprise varieties, some of which are complex hybrids with an admixture of the wild grass *Erianthus maximus* Brongn. This grass also was proved to be susceptible to sugarcane mosaic.

The exhaustive research on these large groups would appear to settle the question of possible immunity in sugarcane and its ancestral forms in the negative, but it cannot be maintained that all sugarcane and its wild relatives, some undoubtedly undiscovered, are susceptible. The comprehensive research does indicate that many of the obscure and usually unpromising cane varieties reported as not becoming

[7] The very great number of forms of domesticated chewing cane developed from wild canes in the scattered islands of Melanesia and in southeastern Asia by primitive horticulturists beginning in the Neolithic Age are known as the original varieties. On the mainland they are slender-stemmed and are derived chiefly from the wild *Saccharum spontaneum*. On the islands of Melanesia they are thick-stemmed and are derived chiefly from *S. robustum* (*41*). The numerous and varied forms, or varieties, of wild species, of which more than 100 have been collected by sugarcane specialists of the U. S. Department of Agriculture, are indigenous to the same areas. *S. robustum* is restricted to a few islands of Melanesia, but the range of *S. spontaneum* is greater, extending to eastern Africa. The lack of forms transitional between the domesticated chewing canes and their wild prototypes is easily explainable on the basis of observations by the writers; namely, the inability of the chewing canes to maintain themselves against jungle competition. Their immediate precursors, selected by primitive men, doubtless similarly were unable to compete with wild plants and when they were discarded in favor of more improved domesticated varieties, they became extinct. Together, the chewing canes and wild varieties, numbering in the thousands, constitute the primary reservoir of parent material for breeding, as now developed by the U. S. Department of Agriculture.

infected by inoculation tests, or by observation, may have escaped infection because of faults in the technique used or for other reasons.

The question of resistance to mosaic is one in which there was great promise almost from the beginning of this research. For a short time nothing less than immunity (and eradication of the disease in commercial sugarcane) was regarded as indispensable, because of the sugarcane-wild-host relationship and its potentialities for perpetuating disease losses, however small, in tolerant varieties. With the development of hybrid commercial varieties that not only tolerate the disease without great loss but resist infection as well, and occasionally recover en masse, it became evident that "living with the disease" is practicable.

Without exception the varieties of *S. sinense* take the disease with more difficulty than varieties of *S. officinarum*, and, moreover, the comparative injury is slight (*22*). Varieties of *S. barberi* become infected readily, but the injury is considerably less as a rule than in the case of *S. officinarum*. Some varieties of *S. spontaneum* tested have never shown any obvious sign of the disease, and, as a group, the injury of those found to be susceptible is so slight as to be practically negligible (*28*). In contrast, the varieties of the large wild sugarcane *S. robustum* exhibit pronounced signs of mosaic when infected, and injury, although apparently considerably less than in varieties of *S. officinarum*, is greater than in varieties of the small wild *S. spontaneum* (*16*). The susceptibility of *S. edule* is a matter of indifference, because it does not produce normal flowers and cannot be used in breeding.

Of the large groups studied, the most promising canes for breeding cannot be stated unequivocally, because that depends upon the part of the world for which the breeding for improvement is designed, chiefly upon latitude. In general it has been found advisable to use parent varieties whose geographic centers of origin are the agroclimatic analogues of the commercial areas for which the breeding is conducted. That rule should be followed for at least one of the parents. In view of the fact that it is almost indispensable to resort to *S. officinarum*, a tropical species, for the desired combination of high sucrose and reasonably high cane weight, it is obvious that the rule cannot be followed explicitly in breeding for areas in the Temperate Zone or subtropics. A compromise, however, in which one parent selected is from a latitude equivalent to the commercial area works out reasonably well.

In respect to mosaic resistance the most promising combinations for Louisiana and the Southern States generally are varieties of *S. officinarum* and *S. barberi* or *S. officinarum* and Temperate Zone varieties of *S. spontaneum*. Complex hybrids with *S. officinarum*, *S. barberi*, and *S. spontaneum* are even better and in fact have been used (*59*). Up to the present no success has attended the use of *S. sinense* as a parent. The hybrid progenies with *S. sinense* blood have many undesirable characteristics, including persistent clinging leaf sheaths, which complicate the problem of detrashing now so important with the advent of mechanical harvesting. The knobby nodes of the stem also are undesirable, and we are inclined to agree with the verdict of Dr. J. Jeswiet, the Netherlands' cane variety expert,

who shrugged off the *S. sinense* hybrid seedlings with the comment that they are "just bones."

For Hawaii and Puerto Rico a somewhat modified selection of parents has proved desirable, namely, the substituting of tropical varieties of *S. spontaneum* and the addition of *S. robustum*. One of the parents of a recently released commercial variety now grown in Hawaii is a quarter breed *S. robustum*, and that species may eventually come into wider use in the Tropics, especially where only the mild strains of mosaic exist.

Preparation for and actual breeding and testing of the disease-resistant varieties at Canal Point involved original studies of the development of the sugarcane inflorescence and seed in Florida (*3*), stimulation of blooming by the artificial interference with photoperiod (*26, 57*), cold resistance (*18*), preservation of sugarcane pollen for long-distance shipment by airplane (*58*), use of the reversed seasons north and south of the Equator to cross long-day and short-day varieties of sugarcane by rapid air transport of pollen (*26*), the climatic requirements of the basic breeding types (biotypes) by growth tests of the group replicated at eight stations spaced at intervals between the Equator and 37° N. (*21*), and methods of effective routine testing of progenies for disease reactions (*2*).

Geographic Origin of Epidemic and Its Significance

As the work progressed it became somewhat more diffuse in objectives, but the underlying current was a sustained study of sugarcane mosaic virus, including its geographic point of origin and spread (*17, 25*), similar studies on the host plant (*27*), and the interrelationships of the virus and its host. The evidence on geographic origin is circumstantial, but the weight of many observations inclines one strongly to the conclusion that in the case of sugarcane mosaic the host and virus have a common point of origin. The evidence clearly points out that in the epidemic of the early part of the present century the virus stemmed from Java, carried in infected seed cane. Every outbreak when carefully studied can be traced directly or indirectly to that source and to that method of dissemination.

In the New World there remain scores of small isolated and significantly unimportant valleys where varieties popular a century or more ago are grown exclusively and they are still free of the disease. The more progressive industries that were interested in importing new and better varieties have the disease. In Java, wherever the disease is eliminated from the cane by expensive roguing or other similar means, it dies out in the other surrounding grasses rather quickly, and the same observation has been made in other parts of the civilized world where cane is a modern culture.

In New Guinea and other islands of Melanesia, where the tropical forms of sugarcane are presumed to have been developed as horticultural crops in primitive garden culture, the disease is not abundant on the cane but is occasionally found. Our evidence on the origin of these garden sugarcanes (*27, 28, 41*) shows clearly that they must have reached Java from Melanesia comparatively recently by the unrecorded intervention of man, possibly within the time of recorded history in some other parts of the world. This evidence incidentally

helps to sustain the validity of the renowned "Wallaces Line," which divides the vegetation and forms of life of the Malay Archipelago into Austro-Malayan (including New Guinea) and Indo-Malayan (including Java). The dispersal of cane varieties from the center of origin in Melanesia has been exclusively centrifugal. To Melanesia, most certainly, there has been no backwash or flow inward of varieties, with or without an attendant virus, from the outside world and no need of new varieties in the primitive native economy.

The recent rude awakening of those islands by war may result in starting a two-way traffic of plant propagating materials and eventually in the drifting in of modern improved seedling varieties.

Before that takes place there is opportunity, perhaps, to learn something of value as to the origin of strains of the virus. Whether the virus of sugarcane mosaic was a mixture of strains when it started on its world tour from the place of origin, or whether "biologic forms" may have developed from an originally homogenous strain in response to new environments, is a significant question. A consideration of the sugarcane virus strains constitutes one of the sections of the present contribution on mosaic, which could be profitably amplified by a comparable series of tests with the same differential host varieties used in Louisiana, conducted in widely dispersed areas in the suspected general area of virus origin. The differential host varieties are a group of sugarcane clones selected as suitable for the identification of the several virus strains because of their pronounced differences in symptoms when they become infected with different virus strains. Strong inferences on the area of origin of both sugarcane and mosaic virus may be drawn, but they do not necessarily prove a primitive, constant association of sugarcane and the virus in question. The possibility should be taken into consideration of the existence of grasses in the area not so incompatible with the virus as other grasses outside the area seem to be.

The economic value of the research on sugarcane mosaic to the present time has been considerable in comparison with the cost. A dramatic upcurve in production of sugar per acre followed the development and release of the first group of disease-resistant varieties, and saved from extinction industries with enormous investments in mills, machinery, plantation railroads, and special equipment. In 1940 a careful estimate based on production records from official sources showed that the improved disease-resistant plant material distributed to farmers had conserved capital and augmented national income in the United States by many hundreds of millions of dollars (*19*).

The research on mosaic and its control has not ended with the mere repair of the disease injury to the sugarcane industry. In Louisiana the present disease-resistant varieties of partly Temperate Zone origin have brought with them many accessory benefits. Because of their ability to withstand somewhat lower temperatures in the winter rest period, the practice of summer or early fall planting has become more practicable. This permits better development of the underground parts before cold weather sets in, with the advantage of more uniform, precocious development of the plants the following spring. With varieties of the type now used, there is definite economy also in the quantity of seed cane required, and the average acre yields of sugar are greater and subject to less fluctuation than the tropical

varieties formerly used. The improved tone and tempo of production so easily perceived is the result of added benefits set in motion by and contingent upon the initial benefit. The farmer with a promising crop is the one who gives it good culture; he secures the means whereby he can fertilize his crop; and he arrives at a position that enables him to devote more acreage to legumes and, in general, to better planned soil management and erosion control.

The investigation of sugarcane mosaic and its control has aided in developing a more scientifically mature view of plant introduction, applicable in general, but uniquely applicable in dealing with vegetatively reproduced plants like sugarcane introduced from remote centers of origin. Three points will help to make this clear.

The first is the increasingly obvious need to acquire representative forms of the sugarcane plant comprising the full range of natural variation within the several species and their near relatives. The full range is important not only to insure capturing disease resistance but also all other desirable characteristics needed.

The second is that to obtain the full range of natural variation, the dispersal areas of the plant in nature must be taken into account. The greatest number and diversity of varieties are found near the center of origin, and successively decreasing numbers and variations are found as the edges of the dispersal area are approached (*20*). Sugarcane in America is far beyond the edge of the natural dispersal area in southeastern Asia, and only a few years ago the number of forms of sugarcane that had reached the New World by introduction were essentially limited to one type and all were very susceptible to mosaic. The large and constantly replenished reference collection of wild and cultivated varieties now available in America has greatly improved the assortment of useful characteristics that can be combined in breeding.

Need for Continuous Variety Replacement

The third point of importance is the unending need for new varieties because of changing conditions in the cultivated fields. The constant stream of new hybrid varieties resulting from crosses made at Canal Point and satellite substations (sometimes requiring intershipment of pollen by airplane) has become a necessity recognized by the cane growers. They, as well as the specialists working on field production problems, are coming more and more to the realization that the environment in which the crop is produced is not static but dynamic, presenting in every cultivated field an ever-shifting picture of varying soil composition and texture, weed populations, water relationships, and other changing factors, including the character or abundance of pathogens, such as the strains of mosaic virus.

In contrast, the individual variety or vegetatively propagated clone of sugarcane is static, but, fortunately, in the large resource of parent breeding material gradually being assembled, there are elements that offer the opportunity to mold the variety by breeding to meet the situations as they unfold and to keep ascendancy over the fluctuating environment.

There is no possibility of developing a "wonder" cane as a perfect, ultimate objective of cane breeding. The only wonder is that some varieties so designated in the past lasted as long as they did. These

conceptions of the interrelation between a crop plant and its environment are not so readily demonstrable in cases of plants grown from seeds in which natural seed selection and survival of adapted elements in the "variety" help to cushion it in some degree against the gradual encroachment of unfavorable environmental change. In the case of sugarcane there is reason to foresee the need for comparatively more rapid and, of course, endless turn-over of varieties.

Thus far the required revolution of varieties has been accomplished by a synchronized system involving many interdependent programs of investigation and action. The over-all program is composed of elements tied together in sequence of time in a manner that may be likened to the flow of an assembly line with operators strategically located on the line. To maintain the flow efficiently there is an irreducible minimum of specialists, including ecologists, systematic botanists, gardeners, geneticists, cytologists, plant breeders, physiologists, pathologists, biochemists, and agronomists. The time required from the explorations for new germ plasm in the geographic areas, where such prospecting is profitable, to the emergence of the finished hybrid variety ready for quantity production of sugar on the farms is also a practically irreducible minimum of about 10 years. Just as in the case of assembly lines in general, it would be a mistake to think of this system as a fixed routine. Numerous opportunities are envisioned for improvement by further research; for example, more intensive studies of agro-climatic analogues involving the interference with successful variety interchanges by obscure insect-transmitted diseases like sugarcane mosaic or sugar beet curly top. We are told that Riverside, Calif., is essentially the agro-climatic analogue of Jericho, near the Trans-Jordan border in Palestine. There are good grounds for believing that a given variety of sugar beets adapted to conditions at Jericho would be adapted to conditions at Riverside. The fly in the ointment is the indigenous leafhopper in California that transmits the virus of curly top to sugar beets from the indigenous desert vegetation that harbors the virus. The hypothetical variety adapted to conditions at Jericho would fall down at Riverside. With the shedding of light on these highly specialized relationships of crop ecology, in which sugarcane studies have aided in an intriguing manner, we are led to a better understanding of the potentialities of crop resettlement and adaptation.

Annual Value of Continuous Variety Replacement

The value of the continuing increased production of sugar per acre with continuing substitution of currently adapted disease-resistant varieties, compared with production from the old varieties before the epidemic struck, was recently estimated [8] at more than 20 million dollars annually in Puerto Rico and the southern United States, and similar but larger benefits, difficult to estimate, are assumed throughout the sugarcane industries of the world.

[8] Brandes, E. W. Sugarcane industry restored by disease research and breeding. U. S. Dept. Agr. Res. Achievement Sheet 59 (Pi), Apr. 24, 1946. [Processed.]

HOST RANGE OF THE SUGARCANE MOSAIC VIRUS

The range of wild forms of sugarcane extends from Africa to southeastern Asia and the western islands of the Pacific Ocean. Cultivated forms were first developed near the center of this large area and later were carried to all parts of the equatorial belt and the fringes of the Temperate Zone. Sugarcane mosaic is probably the most cosmopolitan of all the viruses that cause disease of grasses. It occurs naturally on several species of *Saccharum* and also on many other wild and cultivated grasses. In Louisiana occurrence of the disease on genera other than *Saccharum* would seem, in general, to depend on close proximity to mosaic-diseased sugarcane. The virus has been artificially transferred also to additional grass species in which it has not been found occurring naturally.

Sugarcane Species and Varieties

The extensive reference collection of sugarcane varieties maintained by the United States Department of Agriculture at field stations at Houma, La., Cairo, Ga., and Canal Point, Fla., provides data on the relative susceptibility or resistance of a wide range of varieties, representing the five recognized species of *Saccharum* and many interspecific hybrids. At Cairo the varietal nursery antedates the introduction or discovery of mosaic in the United States, while the one at Canal Point was established in 1921 and the one at Houma in 1924. All three have been continued. Additions to the original collections have been made from time to time. Some varieties have been lost, owing to growth failure, and many have been dropped after adequate observation. Each year, records have been made of the diseases that occurred on these varieties.[9]

The plantings at Houma have usually been located in areas exposed to maximum natural spread of mosaic, but only a few of the varieties actually have been subjected to artificial inoculation by the needle-prick method. A brief summary, giving the number of years of observations and the maximum percentage of mosaic-affected plants for each variety, is presented in table 1. While the classification of the varieties in this table may not have a well-defined genetic basis, because of interspecific and intergeneric natural hybridization, it has proved satisfactory for reference in the study of disease resistance and its inheritance. There has been some variation from year to year in the method of taking notes on the incidence of disease. In the earlier years, actual counts and percentage determinations of mosaic were made, but in later years these were either estimated or simply recorded as present or absent.

[9] The writers are indebted to B. A. Belcher, associate agronomist, Division of Sugar Plant Investigations, for most of the mosaic records made at Cairo, Ga.

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TABLE 1.—*Occurrence of mosaic on various species and varieties of sugarcane exposed to natural infection at Houma, La., and Cairo, Ga.*

Variety [1]	Period observed	Maximum incidence of mosaic
	Years	*Percent*
1. Noble varieties (*Saccharum officinarum* L.): [2]		
a. Named varieties:		
Ashy Mauritius	1	100
B. 208	3	100
B. 417	7	100
B. 726	4	100
B. 891	4	100
B. 6032	5	100
B. 6308	6	75
B. 6835	6	100
Ba. 11569	4	37
B. H. 10–12	9	100
Batjan	3	100
Cavangerie	1	100
Creole	2	100
Crystalina	13	100
Cuba 35	9	100
Cuba 553	9	50
D–74	11	100
D–95	7	100
D–109	10	100
D–117	12	100
D–625	5	100
D–663	4	100
D–666	3	86
D–1135	12	100
D. I. 52	11	100
Diamond 10	4	100
E. K. 2	8	50
E. K. 28	11	100
Fiji	8	100
Green Ribbon	11	100
H. 109	7	100
H. 456	2	100
H. M. 14	1	100
H. M. 320	1	100
H. M. 487	1	80
H. M. 603	1	70
H. M. 606	2	100
H. M. 607	1	100
H. M. 613	1	100
H. M. 629	1	100
H. Q. 5	6	100
Haak-kwat-che	10	100
Kam Chaan Che (Rose Bamboo)	11	100
L–256	3	56
L–511	9	57
Lahaina (Muk Che)	2	70
Loethers	2	[3]
Louisiana Purple	12	100
Louisiana Striped	13	100
Manjay	11	100
N. H. 2 (Ti Karamu)	3	100
Otaheite	11	100
P. O. J. 100	12	60

See footnotes at end of table.

TABLE 1.—*Occurrence of mosaic on various species and varieties of sugarcane exposed to natural infection at Houma, La., and Cairo, Ga.*—Continued

Variety [1]	Period observed	Maximum incidence of mosaic
	Years	*Percent*
1. Noble varieties (*Saccharum officinarum* L.)[2]—Con.		
a. Named varieties—Continued		
P. R. 358	9	100
P. S. A. 48	1	[3]
P. S. A. 49	1	[3]
P. S. A. 50	1	[3]
P. S. A. 51	1	0
P. S. A. 52	1	0
P. S. A. 53	1	[3]
P. S. A. 54	1	0
Porvenir 30	1	[3]
Q. S13	6	0
Raratonga 1	5	100
Raratonga 2	5	100
Raratonga 3	5	100
Rutu	2	100
S. C. 12–4	7	100
S. C. 22–21	9	100
S. J. 4	3	70
S. W. 3	9	100
S. W. 111	8	100
S. W. 499	6	100
Shamsara	11	100
Soerat Njamploeng (21–C–3)	6	100
Striped Mauritius	9	100
Striped Tip	7	100
T–77 (Imp. 969)	2	50
Tahiti 3	2	90
Tonga 3	2	100
Tonga 5	1	100
Tonga 6	1	100
Wit Ceram	11	26
Yellow Caledonia	3	80
26–C–270	1	80
75–Lehu	1	30
b. New Caledonia varieties:		
N. C. 5	3	100
N. C. 11	3	100
N. C. 15	3	95
N. C. 17	3	100
N. C. 18	3	80
N. C. 19	3	100
N. C. 20	3	100
N. C. 21	3	100
N. C. 24	3	100
N. C. 25	3	60
N. C. 29	3	70
N. C. 30	3	100
N. C. 31	3	100
N. C. 32	3	100
N. C. 33	3	100
N. C. 39	2	90
N. C. 40	3	100
N. C. 42	2	50

See footnotes at end of table.

TABLE 1.—*Occurrence of mosaic on various species and varieties of sugarcane exposed to natural infection at Houma, La., and Cairo, Ga.*—Continued

Variety [1]	Period observed	Maximum incidence of mosaic
	Years	*Percent*
1. Noble varieties (*Saccharum officinarum* L.)[2]—Con.		
b. New Caledonia varieties—Continued		
N. C. 50	3	100
N. C. 51	2	60
N. C. 53	3	40
N. C. 59	3	100
N. C. 64	3	70
N. C. 74	3	100
N. C. 76	3	90
N. C. 78	3	80
N. C. 80	3	100
N. C. 81	3	70
N. C. 83	3	100
N. C. 90	2	100
N. C. 91	3	100
N. C. 92	3	100
N. C. 93	2	90
N. C. 94	3	100
N. C. 99	3	90
N. C. 104	2	60
N. C. 116	3	100
N. C. 117	3	100
N. C. 130	1	100
c. New Guinea varieties:		
96 N. G. 15 (Badila)	12	100
96 N. G. 22 (Mahona)	3	50
96 N. G. 24 (Goru)	6	60
96 N. G. 24a (Striped Goru)	5	80
14 N. G. 124 (Korpi)	4	100
14 N. G. 190 (Oramboo)	2	100
14 N. G. 241 (Nanemo)	3	50
21 N. G. 1	6	18
21 N. G. 2	6	12
21 N. G. 4	2	50
21 N. G. 5	6	3
21 N. G. 6	4	30
21 N. G. 7	5	100
21 N. G. 10	5	50
21 N. G. 12	7	93
21 N. G. 13	2	60
21 N. G. 14	1	0
21 N. G. 15	6	6
21 N. G. 16	1	80
21 N. G. 17	6	25
21 N. G. 20	5	100
21 N. G. 22	2	80
21 N. G. 23	5	100
21 N. G. 31	2	30
21 N. G. 32	2	30
21 N. G. 33	5	6
21 N. G. 34	2	40
21 N. G. 36	6	80
21 N. G. 37	6	40
21 N. G. 44	6	100

See footnotes at end of table.

TABLE 1.—*Occurrence of mosaic on various species and varieties of sugarcane exposed to natural infection at Houma, La., and Cairo, Ga.*—Continued

Variety [1]	Period observed	Maximum incidence of mosaic
1. Noble varieties (*Saccharum officinarum* L.):[2]—Con.		
c. New Guinea varieties—Continued	*Years*	*Percent*
21 N. G. 49	7	60
21 N. G. 51	5	45
21 N. G. 54	6	100
21 N. G. 55	7	60
21 N. G. 57	5	71
21 N. G. 58	5	100
28 N. G. 11	2	100
28 N. G. 12	6	40
28 N. G. 14	2	30
28 N. G. 15	7	71
28 N. G. 17	7	0
28 N. G. 18	7	100
28 N. G. 20	7	95
28 N. G. 21	7	100
28 N. G. 23	7	80
28 N. G. 25	7	68
28 N. G. 26	7	90
28 N. G. 32	7	50
28 N. G. 34	7	100
28 N. G. 35	7	100
28 N. G. 36	7	100
28 N. G. 37	7	67
28 N. G. 40	7	20
28 N. G. 42	7	40
28 N. G. 45	7	30
28 N. G. 46	7	90
28 N. G. 47	7	100
28 N. G. 51	7	30
28 N. G. 52	7	100
28 N. G. 56	7	75
28 N. G. 59	6	100
28 N. G. 65	7	100
28 N. G. 68	7	80
28 N. G. 72	7	100
28 N. G. 80	7	100
28 N. G. 87	8	100
28 N. G. 89	7	100
28 N. G. 90	7	30
28 N. G. 96	7	100
28 N. G. 97 (Imp. 418)	7	100
28 N. G. 97 (Imp. 503)	7	100
28 N. G. 106 (Imp. 426)	7	12
28 N. G. 106 (Imp. 508)	7	50
28 N. G. 109	7	6
28 N. G. 110	7	30
28 N. G. 202	7	20
28 N. G. 206	1	40
28 N. G. 207	7	40
28 N. G. 208	7	54
28 N. G. 211	7	20
28 N. G. 212	7	30
28 N. G. 220	7	90

See footnote at end of table.

TABLE 1.—*Occurrence of mosaic on various species and varieties of sugarcane exposed to natural infection at Houma, La., and Cairo, Ga.*—Continued

Variety [1]	Period observed	Maximum incidence of mosaic
	Years	*Percent*
1. Noble varieties (*Saccharum officinarum* L.)[2]—Con.		
c. New Guinea varieties—Continued		
28 N. G. 223	2	100
28 N. G. 260	7	80
28 N. G. 261	8	100
28 N. G. 262	7	100
28 N. G. 263	7	80
28 N. G. 265	7	50
28 N. G. 266	7	[3]
28 N. G. 268	7	30
28 N. G. 269	1	80
28 N. G. 284	7	100
28 N. G. 285	7	100
28 N. G. 288	7	10
2. Wild canes (*Saccharum robustum* Brandes and Jeswiet ex Grassl):		
N. H. 1	2	100
28 N. G. 219	2	20
28 N. G. 251	7	100
3. Canes with edible tassels (*Saccharum edule* Hassk.):		
28 N. G. 201	7	30
28 N. G. 286	7	100
4. Indian varieties (*Saccharum barberi* Jeswiet):		
Chunnee	9	100
Hatooni	9	30
Hemja	7	10
5. Chinese varieties (*Saccharum sinense* Roxb.):		
Cayana	9	[3]
Chikucha	8	20
Chukche	9	0
Kavangire	11	[3]
Khera	8	[3]
Kikaigashima	8	[3]
Kinar	9	0
Merthi	12	0
Oshima	13	[3]
Tekcha	13	[3]
Uba	13	65
Yontanzan	8	25
6. Wild canes (*Saccharum spontaneum* L.):		
Aegyptiacum	1	0
59 Amu Darya Selections	4	[3]
U. S. 4513	2	0
U. S. 4514	2	0
U. S. 4515	2	0
U. S. 4516	2	0
U. S. 4517	2	0
60 Amu Darya Selections	4	[3]
U. S. 4518	2	0
U. S. 4519	2	0
U. S. 4520	2	0
U. S. 4521	2	0
U. S. 4522	2	0

See footnotes at end of table.

TABLE 1.—*Occurrence of mosaic on various species and varieties of sugarcane exposed to natural infection at Houma, La., and Cairo, Ga.*—Continued

Variety [1]	Period observed	Maximum incidence of mosaic
	Years	*Percent*
6. Wild canes (*Saccharum spontaneum* L.)—Continued		
U. S. 4523	2	0
U. S. 4524	2	0
Burma	2	0
Coimbatore, India	5	0
Coimbatore Local	3	0
Dacca	3	0
Djatiroto	3	0
Formosa 1	3	0
Formosa 2	3	0
Formosa 3	3	0
Formosa 4	3	0
Gebra Bon	3	0
Kepandjen	3	0
Kloet	3	0
Lahore	1	0
28 N. G. 291	1	0
28 N. G. 292	1	0
Pasoeroean	8	0
Rellagadi	3	0
S. H. 244 (Imp. 956)	1	0
S. H. 249 (Imp. 957)	1	0
Tabonga	8	0
28 N. G. 105 [5]	1	0
7. Interspecific hybrids, selfs, and backcrosses:		
a. Group 1×4:		
Co. 210	8	60
Co. 213	10	50
Co. 232	10	70
Co. 301	1	0
H. 28–4997	6	[3]
H. 28–5009	6	100
H. 28–5019	6	100
H. 28–5055	6	76
H. 28–5114	6	26
H. 28–5118	6	70
P. O. J. 36	13	100
P. O. J. 36–M	13	100
P. O. J. 139	7	18
P. O. J. 213	13	100
P. O. J. 228	13	40
P. O. J. 234	13	100
P. O. J. 501	7	50
P. O. J. 826	13	100
P. O. J. 979	11	100
P. O. J. 1228	2	100
P. O. J. 1335	10	100
P. O. J. 1337	7	100
P. O. J. 1499	10	100
P. O. J. 2379	13	100
P. O. J. 2806	4	[3]
b. Group 1×5:		
C. H. 64–21	9	0

See footnotes at end of table.

TABLE 1.—*Occurrence of mosaic on various species and varieties of sugarcane exposed to natural infection at Houma, La., and Cairo, Ga.*—Continued

Variety [1]	Period observed	Maximum incidence of mosaic
	Years	*Percent*
7. Interspecific hybrids, selfs, and backcrosses—Con.		
c. Group 1×6:		
Co. 214	8	0
C. 107	6	0
Hind's Special	9	0
I–1081	8	0
Kassoer	13	0
P. O. J. 2775	6	4
Toledo	12	0
Tuc. 472	6	0
d. Group 1 × 7c (Kassoer nobilized once):		
P. O. J. 2221	12	95
P. O. J. 2222	9	6
P. O. J. 2354	12	100
P. O. J. 2364	11	[2]
Tjepiring 24	12	0
e. Group 1 × 7d (Kassoer nobilized twice or more):		
Co. 421	3	30
Cuato 307	1	0
E–12	2	60
H. 28–245	6	100
H. 30–2417	1	100
H. 30–4422	1	20
H. 31–156	1	30
H. 31–626	1	50
H. 31–2441	1	0
Mayaguez 28	3	0
Mayaguez 270	2	0
N. M. 5	1	0
N. M. 75	2	0
Naz (Reunion)	4	90
P. 33–1	3	50
P. 33–3	3	10
P. 33–5	3	30
P. 33–8	3	30
P. 33–9	3	0
P. 33–11	2	0
P. 33–12	3	10
P. 33–14	3	100
P. 33–15	3	0
P. 33–16	3	0
P. 33–20	3	0
P. 33–24	3	[3]
P. 33–29	2	[3]
P. 33–31	3	10
P. O. J. 2714	13	[4]
P. O. J. 2722	6	0
P. O. J. 2725	13	0
P. O. J. 2727	13	40
P. O. J. 2802	7	20
P. O. J. 2822	6	56

See footnotes at end of table.

TABLE 1.—*Occurrence of mosaic on various species and varieties of sugarcane exposed to natural infection at Houma, La., and Cairo, Ga.*—Continued

Variety [1]	Period observed	Maximum incidence of mosaic
	Years	*Percent*
7. Interspecific hybrids, selfs, and backcrosses—Con.		
e. Group 1 × 7d (Kassoer nobilized twice or more)—Continued		
P. O. J. 2878	10	17
P. O. J. 2883	4	(3)
P. O. J. 2940	4	(3)
P. S. A. 15	1	0
P. S. A. 19	1	0
P. S. A. 24	1	(3)
P. S. A. 35	1	(3)
P. S. A. 36	1	(3)
P. S. A. 37	1	(3)
Uba Marot	5	0
f. Group 6 × 7c:		
Turkestan hybrids (P. O. J. 2725 × Amu Darya): 1430 seedlings (F)	2	60
g. Group 1 × 2:		
Oramboo × 28 N. G. 251	2	30
8. Complex hybrids:		
a. Inheritance from groups 1, 4, and 6:		
C. P. 807	11	(4)
C. P. 28/11	9	(4)
C. P. 28/19	9	(4)
C. P. 29/103	8	(4)
C. P. 29/116	8	(4)
C. P. 29/120	8	(4)
C. P. 29/320	8	5
Co. 281	12	100
Co. 290	12	100
Co. 313	2	100
F. 29–7	2	0
F. 30/16	1	0
F. 30/20	2	0
F. 30/35	3	(4)
F. 31/436	2	20
F. 31/962	2	(4)
P. O. J. 2747	11	0
P. O. J. 2753	9	80
b. Inheritance from groups 1, 5, and 6:		
H. 31/711	1	0
H. 31/1211	1	100
H. 31/1213	1	0
H. 31/2343	1	0
c. Inheritance from groups 1, 2, and 6:		
32 M. Q. 579	2	0
9. Intergeneric hybrids:		
Saccharum × sorghum:		
Co. 515	4	100
G. 496	4	0
G. 497	4	0

See footnotes at end of table.

TABLE 1.—*Occurrence of mosaic on various species and varieties of sugarcane exposed to natural infection at Houma, La., and Cairo, Ga.*—Continued

Variety [1]	Period observed	Maximum incidence of mosaic
	Years	*Percent*
10. Miscellaneous seedling varieties:		
B-30-13	1	0
B-31-24	2	60
B-32-34	2	100
C. 2443	3	100
C. 2905	3	100
C. H. 64/21	3	0
Co. 270	2	40
Co. 356	2	0
Co. 399	1	0
F. 29/265	1	(4)
F. 31/131	2	0
F. 31/177	2	(4)
F. 31/188	2	0
F. 31/253	1	(4)
F. 31/641	1	0
F. 31/762	1	0
F. 31/831	1	70
F. 31/856	1	0
F. 31/951	1	100
F. 31/985	1	30
F. 33/27	1	20
F. 33/161	1	0
F. C. 305	10	100
F. C. 306	10	100
F. C. 1017	2	0
G. Z. 1188	7	100
H. Q. 409	6	70
H. Q. 429	2	0
H. Q. 813	2	60
Mayaguez 49	5	0
Mayaguez 151	5	0
Mayaguez 255	2	0
P. O. J. 1449	10	100
P. R. 492	10	100
S. C. 14-93	7	100
Tuc. 454	6	0
32-M. Q. 135	2	100
32-M. Q. 478	2	0
32-M. Q. 629	2	100
32-M. Q. 657	2	100
2443-C	8	70
4475-C	8	100
4905-C	9	100
11. *Erianthus* sp.:		
a. *E. maximus* Brongn:		
Fiji 1	2	100
Fiji 2	1	90
Fiji 3	1	80
N. C. 132	3	100
Raiatea 1	3	95

See footnotes at end of table.

TABLE 1.—*Occurrence of mosaic on various species and varieties of sugarcane exposed to natural infection on Houma, La., and Cairo, Ga.*—Continued

Variety [1]	Period observed	Maximum incidence of mosaic
11. *Erianthus* sp.—Continued		
b. *E. arundinaceus* (Retz.) Jeswiet ex K. Heyne: 28 N. G. 7	*Years* 1	*Percent* 0
12. Wild canes (*Narenga porphyrocoma* (Hance) Bor): Shantir Barhni	1	0

[1] Many varieties of sugarcane bred at experiment stations are designated by letters or other abbreviations, usually indicating the place or institution where they originated. The meanings of such designation for varieties mentioned in this article are as follows:

B., Ba., B. H.=Barbados.
C. and C. H.=Cuba.
Co.=Coimbatore (India).
C. P.=Canal Point (Fla.).
D.=Demerara.
D. I.=Dimak Idjoe.
E.=Egypt.
E. K.=Edward Karthaus.
F.=Florida (or Formosa).
F. C.=Fajardo Central.
G.=Coimbatore or Java series.
G. Z.=Java series.
H.=Hawaii.
H. M.=Hebbal, Mysore, India.
H. Q.=Hambledon, Queensland.
I.=Java series.
L.=Louisiana.
M. Q.=Macknade, Queensland.
N. C.=New Caledonia.
N. G.=New Guinea.
N. H.=New Hebrides.
N. M.=Natal.
P.=Seedlings from Philippine seed.
P. O. J.=Proefstation Oost Java.
P. R.=Puerto Rico.
P. S. A.=Philippine Sugar Assoc.
Q.=Queensland.
S. C.=Saint Croix.
S. H.=India.
S. J.=South Johnston.
S. W.=Sempal Wadak.
T.=Trinidad.
Tuc.=Tucuman.
U. S.=U. S. Dept. Agr.

[2] A number of varieties listed as *S. officinarum* are believed to be natural hybrids with admixtures of other species. In several cases what are believed to be the same varieties are listed under different names. These names have been retained because the plants were obtained at different times or from different places.

[3] Present.

[4] Trace.

[5] Probable hybrid between *S. spontaneum* and *S. robustum*.

The noble varieties (*S. officinarum*) in general are very susceptible to mosaic, as pointed out by earlier investigators. Many of them show at all times 100 percent infection under Louisiana conditions. A notable exception, never becoming completely infected, is Wit Ceram (table 1, group 1a). The frequent occurrence of recovery, both at the time of germination and in growing cane, plus a certain degree of resistance, makes some varieties of this group appear more resistant than they actually are. On the other hand, it is commonly accepted that Badila (group 1c), for instance, is much more resistant to mosaic infection than such varieties as Louisiana Purple (Black Cheribon) and Otaheite. Although it has been observed with 100 percent infection, Badila usually has a considerable number of symptom-free shoots in every plot.

The Indian varieties (*S. barberi*) are susceptible to mosaic, but in general they are not so severely injured as varieties of *S. officinarum*. Chunnee (group 4) has become 100 percent infected, but Hatooni and Hemja are more resistant. All have exhibited great vigor, reflecting not only their tolerance of mosaic but also great resistance to root rot and other unfavorable conditions associated with local soils (*55*).

The Chinese varieties (*S. sinense*) were considered immune to mosaic until 1925, when Yoder (*71*) reported the disease present in all but two—Kinar and Merthi—of those listed in table 1, group 5. The percentages of infection are rarely high, and the group as a whole is still considered highly resistant to mosaic. Although showing mosaic in Georgia (*71*), Kavangire, Chukche, and Uba especially have never been observed infected in Louisiana.

With the exception of Amu Darya 59 and 60 (table 1, group 6), varieties of the wild sugarcane (*S. spontaneum*) have passed through several years of heavy mosaic spread without becoming infected, and it seems likely that many varieties in this group may be actually immune to the disease.

The large wild cane (*S. robustum*) (*41*) is represented in table 1, group 2, by three collections, all of which have become infected. Limited observations on certain natural hybrids assignable to this group have established this species as generally susceptible to mosaic.

The seedlings derived from interspecific hybrids, selfs, and backcrosses are listed according to parentage with relation to the groups discussed (table 1). In group 7a, with inheritance of mosaic susceptibility from both parents, the varieties are predominantly susceptible. Groups 7b and 7c, with one parent immune or highly resistant to mosaic, are composed mainly of varieties that have remained mosaic-free for 6 to 13 years. P. O. J. 2775 has shown a small percentage of infection but would be considered commercially resistant and probably would seldom become infected under field conditions. It is known also that Toledo has been occasionally observed with mosaic, both in Puerto Rico and the Philippines (*37*). There are probably other similar unreported cases.

The progressive "nobilization" of *S. spontaneum* (backcrossing with *S. officinarum*), groups 7d and 7e, has resulted in a number of good varieties that are resistant or immune to mosaic. Most of them, however, show varying degrees of susceptibility. This procedure of crossing has proved especially productive in providing commercial seedlings with resistance to other diseases as well as to mosaic.

The *S. spontaneum* varieties, 59 Amu Darya and 60 Amu Darya (group 6), from Turkmen S. S. R., are susceptible to mosaic (*27*). When used as males on P. O. J. 2725 (group 7e), 60 percent of the individuals of the resulting F_1 progenies were susceptible to mosaic. A remarkable amount of recovery occurred in these varieties in their first stubble year, possibly showing a reaction to the disease that might conceivably be a valuable characteristic if incorporated into the genetic make-up of a variety.

The seedling in group 7g, resulting from the cross Oramboo × 28 N. G. 251 (*S. robustum*), proved susceptible as might be expected, since both parents are susceptible.

The complex hybrids with inheritance from three groups or species

of *Saccharum* show all degrees of mosaic susceptibility, from 0 to 100 percent. Included in this group are all the present commercial varieties used in Louisiana, many of which were considered immune for several years but eventually became infected in the field. Of the three Coimbatore (Co.) varieties listed as sorghum crosses (group 9), only one has thus far become infected.

The miscellaneous seedling varieties (group 10) not definitely assignable to any of the groups in table 1, because of incomplete information on parentage, are mostly complex derivatives from crosses between the noble and Indian, or noble and immune wild types. This list includes 43 seedlings from many sources, representing practically every country where any attempt has been made to produce new varieties by hybridization or planting of selfed seed. Their mosaic reactions indicate that some of them probably belong in each of the groups listed in the table. (Many of the varieties in this group were important commercial canes in some cane-growing country or possibly in several different countries prior to the introduction and epidemic spread of mosaic.)

A number of these miscellaneous varieties have never shown mosaic in Louisiana, and many of them may be actually immune to the disease. On the other hand, many of the varieties have shown 100-percent mosaic, although not necessarily during every year of the period exposed. Others, showing intermediate percentages of mosaic, have often exhibited greater damage than some of those of the completely infected group that happened to possess greater tolerance to the disease. Therefore, many of the records presented in table 1 are of more value as a guide to the cane breeder than to the cane planter.

The giant tropical species of *Erianthus* (group 11) are included in table 1 because of their resemblance and close relationship to the genus *Saccharum*, with which generic hybrids have been produced (*27*). As a group they appear to be highly susceptible to mosaic.

Other Grass Hosts

The susceptibility of a number of other grasses to the sugarcane mosaic virus has long been recognized and considered important in dissemination of the disease. A number (usually in or near fields of mosaic cane) have been shown to harbor the sugarcane virus; many others have exhibited symptoms that were probably due to this virus, although not demonstrated by reciprocal cross inoculations; whereas still others not reported diseased in the field have been readily infected in greenhouse experiments. Brandes and Klaphaak (*22*) first demonstrated by cross inoculations that a number of even distantly related grasses were probably as congenial hosts for this virus as sugarcane itself. Since then, Kunkel (*48*), Storey (*62*), Hadden (*42*), and others (*32, 34, 39, 40, 49, 70*) have identified the disease in a number of additional hosts that occur in most of the countries where sugarcane is grown.

A list of grass genera and species found susceptible to, or observed infected with, the sugarcane mosaic virus, including those noted in the writers' studies in Louisiana, is shown in table 2. It comprises 38 wild and 10 cultivated grasses, including ornamentals. The ma-

TABLE 2.—*Grasses reported susceptible to sugarcane mosaic, their reported common names, habit of growth, country, authority for the report, and nature of evidence presented*

Latin name [1]	Synonym under which reported	Common name	Habit of growth	Country	Reported by—	Mosaic observed or experimentally transferred by aphids or hypodermic or needle-prick inoculations
Cultivated grasses						
Andropogon sp.		Wonder forage gress		Hawaii	Kunkel (*48*)	Aphids.
Coix lachryma-jobi L.		Jobs-tears	Annual	Papua	Brandes and Klaphaak (*22*).	Observed.
Miscanthus sinensis Anderss.		Eulalia	Perennial	United States	do	Aphids.
Pennisetum glaucum (L.) R. Br.		Pearl millet	Annual	do	do	Hypodermic.
Sorghum virgatum (Hack.) Stapf.	*Holcus sorghum virgatus* (Hack.) Piper.	Tunis grass	do	Hawaii	Kunkel (*48*)	Observed.
S. vulgare Pers.	*H. sorghum* L.	Sorghum	do	United States [2]	Brandes and Klaphaak (*22*).	Hypodermic.
S. vulgare var. *sudanense* (Piper) Hitchc.	*Andropogon sorghum sudanensis* Piper.	Sudan grass	do	Hawaii [2]	Kunkel (*48*)	Aphids.
S. vulgare var. *technicum* (Koern) Jav.		Broomcorn	do	do	Hadden (*42*)	Observed.
Tripsacum laxum Nash		Guatemala grass	Perennial	do	Kunkel (*48*)	Do.
Zea mays L.		Maize, Indian corn	Annual	United States [2]	Brandes and Klaphaak (*22*).	Aphids.
Wild grasses						
Brachiaria extensa Chase	*Brachiaria platyphylla* Nash.	Brachiaria	do	do [2]	do	Observed.
Digitaria sp.	*Syntherisma debilis* (Desf.) Skeels.	Creeping crabgrass		Hawaii	Lee (*49*)	Do.
Do.	*S. pruriens* (Trin.) Arthur.	Crabgrass		do	Kunkel (*48*)	Do.
Do.	*S. verticillaris* (?) [3]			Puerto Rico	Chardon and Veve (*32*).	(?).
D. horizontalis Willd.			Annual	South Africa	Storey (*62*)	Observed.
D. sanguinalis (L.) Scop.	*S. sanguinalis* Dulac	Crabgrass	do	United States [2]	Brandes and Klaphaak (*22*).	Hypodermic.
Do.	*S. sanguinalis* Lam.	do	do	Puerto Rico	Chardon and Veve (*32*)	Aphids.
D. violascens Link	*S. chinensis* Nees		do	Hawaii	Hadden (*42*)	Do.
Echinochloa colonum (L.) Link		Jungle-rice	do	Puerto Rico [2]	Chardon and Veve (*32*)	Do.
Do.	*Panicum colonum* L.	Wild millet		Australia	Cottrell-Dormer (*34*)	Observed.
E. crusgalli (L.) Beauv.		Barnyard grass	do	United States	Brandes and Klaphaak (*22*).	Aphids.
Eleusine indica (L.) Gaertn.		Goosegrass	do	Hawaii [2]	Kunkel (*48*)	Observed.
Do.		do	do	Puerto Rico	Chardon and Veve (*32*)	Aphids.
Eragrostis ciliaris (L.) R. Br.				South Africa	Storey (*62*)	Observed.
Erianthus giganteus (Walt.) Muhl.		Sugarcane plumegrass	Perennial	Louisiana	Summers [4]	Needle.

Gynerium sagittatum (Aubl.) Beauv		Uva grass	do	Peru	Abbott (*1*)	Observed.
Isachne distichophylla Munro ex Hillebr.		Forest grass		Hawaii	Lee (*49*)	Do.
Lamarckia aurea (L.) Moench	*Achyrodes aureum* Kuntze.	Goldentop	Annual	Iowa	Elmer (*40*)	Aphids.
Narenga porphyrocoma (Hance) Bor	*Saccharum narenga* (Nees) Hack.	Shantir Barhni	Perennial	United States	Brandes and Klaphaak (*22*).	Do.
Panicum purpurascens Raddi	*Panicum barbinode* Trin.	Para grass		Cuba	Walker and Stahl (*70*)	Observed.
P. dichotomiflorum Michx		Fall panicum	Annual [5]	United States [2]	Brandes and Klaphaak (*22*).	Aphids.
Do		do	do	do	Summers [4]	Needle.
P. longijubatum Stapf			do	South Africa	Storey (*62*)	Observed.
P. reptans L			do	Louisiana	Summers [4]	Do.
Paspalum boscianum Flügge		Bull paspalum	do	United States	Brandes and Klaphaak (*22*).	Aphids.
P. conjugatum Bergius	*Paspalum conjugatum* L	Sour paspalum	Perennial	Hawaii	Hadden (*42*)	Observed.
P. fimbriatum H. B. K				Cuba	Walker and Stahl (*70*)	Aphids.
P. virgatum L				do	do	Do.
Pennisetum purpureum Schumach		Napier and elephant grasses.		do	East and Weston (*59*)	Observed. [6]
Phyllostachys pubescens Mazel ex Hous				United States	Brandes and Klaphaak (*22*).	Do. [7]
Setaria aurea Hochst		Rattail grass		Australia	Cottrell-Dormer (*34*)	Do.
S. lutescens (Weigal) F. T. Hubb	*Chaetochloa lutescens* Stuntz.	Yellow bristlegrass	Annual	United States [2]	Brandes and Klaphaak (*22*).	Aphids.
S. magna Griseb	*C. magna* Scribn	Giant bristlegrass	do	do [2]	do	Do.
S. poiretiana (Schult.) Kunth	*Setaria sulcata* Raddi		Perennial	South Africa	Storey (*62*)	Do.
S. verticillata (L.) Beauv	*Chaetochloa verticillata* (L.) Scribn.	Bur bristlegrass	Annual	Hawaii	Kunkel (*48*)	Observed.
Do				do	Lee (*49*)	Aphids.
Sorghum sp		Native sorghum		Australia	Cottrell-Dormer (*34*)	Observed.
S. arundinaceum Stapf			Annual	South Africa	Storey (*62*)	Do.
S. halepense (L.) Pers	*Holcus halepensis* L	Johnson grass	Perennial	Hawaii	Kunkel (*48*)	Do.
Sporobolus sp	*Sporobolus diander* Ritz.			do	Hadden (*42*)	Do.
Trichachne insularia (L.) Nees	*Valota insularis* Chase	Sourgrass	Perennial	do	do	Do.
Urochloa helopus Stapf			Annual	South Africa	Storey (*62*)	Do.

[1] Grass names mostly after Hitchcock (*43*).
[2] Also observed occurring naturally in Louisiana.
[3] This report, attributed by Cook (*33*) to Chardon and Veve (*32*), could not be verified.
[4] Results not previously published.
[5] Perennial in southern Louisiana.
[6] Considered by later writers to be a naturally mottled condition of leaf.
[7] Not definitely called mosaic by Brandes, but is so recorded by Cook (*33*).

jority of these have been reported from only one country, although many species undoubtedly occur rather generally in countries growing sugarcane, indicating that the list is probably far from complete geographically. The basis for listing is mainly observational evidence, further indicating the likelihood that many additional susceptible species exist. Many of the hosts recorded by Brandes and Klaphaak (*22*) and the two new ones added for the United States by the senior writer have been discovered incidental to investigations of other phases of the mosaic problem, rather than as a result of any systematic investigation of the host range of mosaic. The list probably includes most of the grass species that are important factors in epidemiology and control of mosaic in the countries mentioned.

CULTIVATED GRASSES

Of the cultivated species listed in table 2 only corn and sorghum are of any consequence in mosaic spread on sugarcane in Louisiana. Corn has always had a regular place in the crop rotation generally practiced on sugarcane plantations. The usual custom has been to plant corn with a legume at least one summer between successive cane occupations. This has been considered necessary to provide feed for the plantation mules, but the acreage planted to corn for this purpose has been materially reduced in recent years, owing to the rapid replacement of mules by tractors.

Corn is highly susceptible to the sugarcane mosaic virus, as shown in comparative tests by Brandes and Klaphaak (*22*) and Stoneberg (*61*). In the vicinity of diseased cane, cases of mosaic will be evident in very young plants. Individual stalks of mosaic corn have been observed harboring countless thousands of corn aphids (*Aphis maidis*). Since corn is a favored host of this aphid, spread of the disease is apt to be rapid. The stage is thus set for the production of large numbers of viruliferous aphids, which are then available for instigating a mosaic epidemic in adjacent canefields when other contributing factors are favorable.

Sorghum is much less generally grown than corn in the sugarcane area of Louisiana, but all varieties observed were susceptible to mosaic. Sorghum also is a favored host of *Aphis maidis*, and is sometimes also colonized by the rusty plum aphid (*Hysteroneura setariae*). Sorghum functions in much the same way as corn in contributing to mosaic spread in sugarcane, the total effect in that role being less, however, because of its limited culture on sugarcane plantations. Being strictly annuals, neither corn nor sorghum furnishes primary infection early in the growing season because each must first be infected from some other source, which usually is presumed to be sugarcane.

The other cultivated grasses listed as mosaic hosts are so rare if occurring in Louisiana as to be of little or no significance in mosaic spread. Those recorded by Brandes and Klaphaak (*22*) were infected in greenhouse experiments. Sudan grass as a mixture in fields of sorghum has been noted by the senior writer to show unmistakable symptoms of mosaic, and the disease was transferred to sugarcane by inoculation.

Other writers, especially Walker and Stahl (*70*), have minimized the importance of cultivated grasses as hazards in the perpetuation and

spread of sugarcane mosaic. They contend that plantings of corn and sorghum usually can be isolated from cane so as to afford them reasonable protection from mosaic infection and, in turn, eliminate corn and sorghum as sources of viruliferous insect vectors of the disease.

ANNUAL WILD GRASSES

A majority of the wild grasses reported as susceptible to sugarcane mosaic are annuals and as such they can contribute to mosaic spread only after they become infected from mosaic cane or other perennial grasses in the field at the beginning of each growing season. The outstanding examples in this respect in most countries are probably the crabgrasses. Mosaic-diseased specimens of *Digitaria sanguinalis*. were collected in Georgia and Florida, as well as in Louisiana, by Brandes and Klaphaak (*22*) in 1919 and 1920, and this species has been reported from Puerto Rico by Chardon and Veve (*32*) and in Cuba by Walker and Stahl (*70*). It frequently approaches 100 percent mosaic infection in Louisiana canefields and is a favored host of *Aphis maidis*.

Crabgrass probably accounts for a considerable proportion of secondary mosaic spread on sugarcane, following cultivation or hoeing of the fields, which forces a general migration of aphids from the destroyed grass to the cane. 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 other wild grass, perennials included. On the other hand, wherever it is possible to start out with mosaic-free cane for planting and thus avoid foci of primary infection in the cane itself, crabgrass naturally becomes less of a factor in mosaic spread than certain perennial species that have been observed to carry infection from year to year.

Mosaic is also seen frequently in Louisiana on yellow bristlegrass (*Setaria lutescens*) and goosegrass (*Eleusine indica*), but never so generally as on crabgrass. It is found only occasionally on the giant bristlegrass (*Setaria magna*) and rarely on *Echinochloa colonum* and *Panicum reptans*. It is of more general occurrence on *Panicum dichotomiflorum*, which is listed as an annual by Hitchcock (*43*) but functions as a perennial under southern climatic conditions and will be treated as such below. *Setaria magna* has been observed infected with mosaic at a distance of at least a mile from any field of sugarcane. The fact that it is apparently an annual in Louisiana indicates either an unnoticed local perennial-grass source of infection or flights of viruliferous aphids from comparatively distant canefields.

PERENNIAL WILD GRASSES

Sugarcane plumegrass (*Erianthus giganteus*) is found rather commonly infected with mosaic in Louisiana. It is a tall, vigorous perennial and is very common at the edge of swamps or marshes but is also found frequently along ditchbanks and waste areas near canefields. Individual stools have been followed for several years, and in most cases the disease was found to persist in them. A

plantation was visited in 1936 that had not produced sugarcane for at least 10 years, and numerous clumps of this grass in an old orchard showed mosaic. Because it was on so many widely separated clumps, the evidence seemed convincing that it had persisted in this species for that length of time.

During 1939 several swampy areas where this species is abundant were visited. In one 2- or 3-acre area a rather sparse stand intermingled with other grass species and weeds had been visited several years previously and found free of mosaic. At the time of the later observation, however, it was estimated that more than 80 percent of the stools were diseased. This area was more than a mile from any sugarcane and at least 4 or 5 miles from any extensive plantings. The evidence suggested that in this case infection resulted from flights of viruliferous insects from distant canefields. A few mosaic-diseased stools were observed in another similar location, but the rest of the observations on this species in isolated plantings did not show mosaic.

Erianthus giganteus is a favored host of the rusty plum aphid (*Hysteroneura setariae*), which is a vector of sugarcane mosaic (*45*). It is very common to see as many as three or four colonies of this aphid on a single stool, well established on the leaf collar in their mud shelters built for them by ants. Although a limited number of trials with *H. setariae* taken from mosaic *Erianthus* failed to effect a transfer of the disease to sugarcane, circumstantial evidence suggests that such transfer may take place in nature. Transfers have been made to sugarcane by needle-prick inoculations from mosaic plants of this grass. Therefore, considering all insect vectors, it is conceivable that as *E. giganteus* appears capable of keeping the infection indefinitely it may contribute to the perpetuation of mosaic, particularly during years when there is little or no secondary spread. If not taken into account *E. giganteus* could complicate attempts to control mosaic in sugarcane by roguing. Its role in Louisiana could be similar to that of *Setaria poiretiana*, which actually frustrated a well-planned mosaic-eradication program in South Africa, as reported by McClean (*50*).

Fall panicum (*Panicum dichotomiflorum*) usually grows as a perennial under southern Louisiana conditions and would have to be so treated in any mosaic-control program or experiment. Isolated diseased stools have been observed for 3 years in the same location. In dense growths of young sugarcane, it has a growth habit similar to that of sugarcane and may easily be overlooked in weeding or casual inspections. This grass is readily propagated by cuttings and might possibly be distributed with cane cut for planting. It is a common weed in canefields and particularly on neglected ditchbanks, headlands, and fence rows, where its growth is recumbent and is given to rather prodigious branching. In 1920 Brandes (*8*) originally communicated the sugarcane mosaic virus to this species by means of viruliferous aphids, and the senior writer has identified strains B and D of the virus in collections of the grass from canefields.

Panicum dichotomiflorum is, under favorable conditions, undoubtedly a factor in mosaic perpetuation and spread to sugarcane.

This grass under certain conditions (not as yet understood) may act as a symptomless carrier of mosaic. Such an instance was first found

in a field on Alma plantation, where a large stool of the grass growing in a row of Co. 281 showed mosaic. The location was carefully noted, and when the plantation was again visited early in the next growing season the clump was readily located but seemed to be entirely free of mosaic. A quantity of it was obtained and cuttings planted in an insectproof greenhouse at Houma. Several of these sprouted and established themselves and every one exhibited clear-cut mosaic symptoms. Whether this phenomenon is of common occurrence in this species is not known, but circumstantial evidence from other instances suggests that it is not infrequent.

The susceptibility of Johnson grass (*Sorghum halepense*) to sugarcane mosaic, reported by Kunkel (*48*) from Hawaii, has not been confirmed in the United States. Even though it may not actually harbor the disease, this grass is undoubtedly a very potent factor in mosaic dissemination. Johnson grass is abundant in and around canefields 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. The possibility that Johnson grass might be a symptomless carrier is refuted by widespread observations. 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 the disease.

EPIDEMIOLOGY STUDIES

Local Spread of Mosaic in the Field

In Brandes' (*7*) account of the original sugarcane mosaic epidemic in Louisiana he showed that, within a period of 3 years, the disease had spread well over 100 miles from the Mississippi River to the western border of Lafayette Parish. In some instances (presumably by wind carriage of aphids) the disease jumped over barriers of wooded swampland 10 to 30 miles wide. The evidence collected during that epidemic suggested that a healthy field might be peppered with infective *Aphis maidis* from considerable distances. Aphid flights have been mentioned frequently in the later literature and demonstrated repeatedly in Java (*30*, *31*). While such sporadic mass movements of vectors have brought about high initial percentages of mosaic in very susceptible varieties and are doubtless responsible for many cases in varieties more resistant to infection in Louisiana, subsequent spread of mosaic has been suspected of being more local in nature. Davis (*35*) observed in Puerto Rico, for instance, a lateral spread of only 50 feet in 8 months.

If such local spread, as contrasted with long-distance carriage of the disease, should prove to be the rule, it might have an important bearing on control methods. In order to obtain some detailed information, therefore, a study of local mosaic spread on the more important commercial varieties as influenced by seasonal and other factors was conducted over a period of years.

The study was limited to certain plots at the United States Sugar Plant Field Station, near Houma, La., supplemented by observations in other parts of the sugar district. The plots were, for the most part, replicated yield tests, with comparisons of healthy and mosaic cane as

sources of planting material. Thus, 30 to 50 percent of the test, or field, consisted of primary mosaic in these diseased plots. The appearance of secondary mosaic in the healthy plots, or in some cases healthy buffer rows, was recorded in connection with the yield tests. Germination counts were usually made late in April or early in May and a maximum stand count in July or early in August. An additional count in June was usually made in plant-cane (i. e., first-crop) tests to help chart the course of secondary spread into initially healthy plots.

The intensity of secondary spread naturally varied from year to year with the population and relative efficiency of vectors and with weather conditions. Such variations were probably more accurately measured by observations on varieties that possess some degree of natural resistance to mosaic infection rather than on very susceptible varieties, such as Louisiana Purple, that might conceivably have become 90 to 100 percent infected in years of relatively low vector incidence. Of the commercial varieties grown in Louisiana, Co. 281 and Co. 290 were admirably suited to this purpose; Co. 281 is appreciably more susceptible than Co. 290; therefore, together, they furnished an excellent medium for measuring secondary spread from year to year.

SPREAD ON CO. 281

Observations on the rate of mosaic spread into initially healthy plots of Co. 281 were made each year from 1932 to 1938. While these plots were not on strictly comparable soils each year, all were within a fairly small area at the United States Sugar Plant Field Station, and the immediate local surroundings of each test were invariably the same. The spread counts were made on individual rows 16 to 24 feet long, of initially healthy cane, with similar rows of mosaic cane on each end and usually on one or both sides. All counts were made of individual shoots or stalks. This method is considered much more accurate than using the stool as a unit, because frequently only 1 or 2 stalks of the 10 or 15 may become infected. The results of these observations are presented graphically in figure 1, which shows wide divergence in total spread over this particular 7-year period. In 1932 and 1933 there was a very low rate of secondary spread as compared with that in the years immediately following, although it was apparently much higher than during the preceding 4 or 5 years, when this variety had remained practically free of mosaic in all plantings in Louisiana. In 1934 there was a decided increase, the spread being more than four times that of the previous year, although the location of the plots from the standpoint of soil type and vector-breeding areas was supposedly the least conducive to secondary spread of any year reported. Similar plots situated in a more favorable location at the station experienced more than double the spread here shown.

The years 1935 and 1938, with an intermediate rate of spread, should probably be considered about average for the 7-year period, while 1936 must have experienced nearly maximum secondary spread, considering the comparative resistance of the variety involved. The plots during 1936 were on the same piece of land that was used for 1932, and the figures of 71 percent and 7.8 percent, respectively, should be directly comparable. In general, the trends in rate of

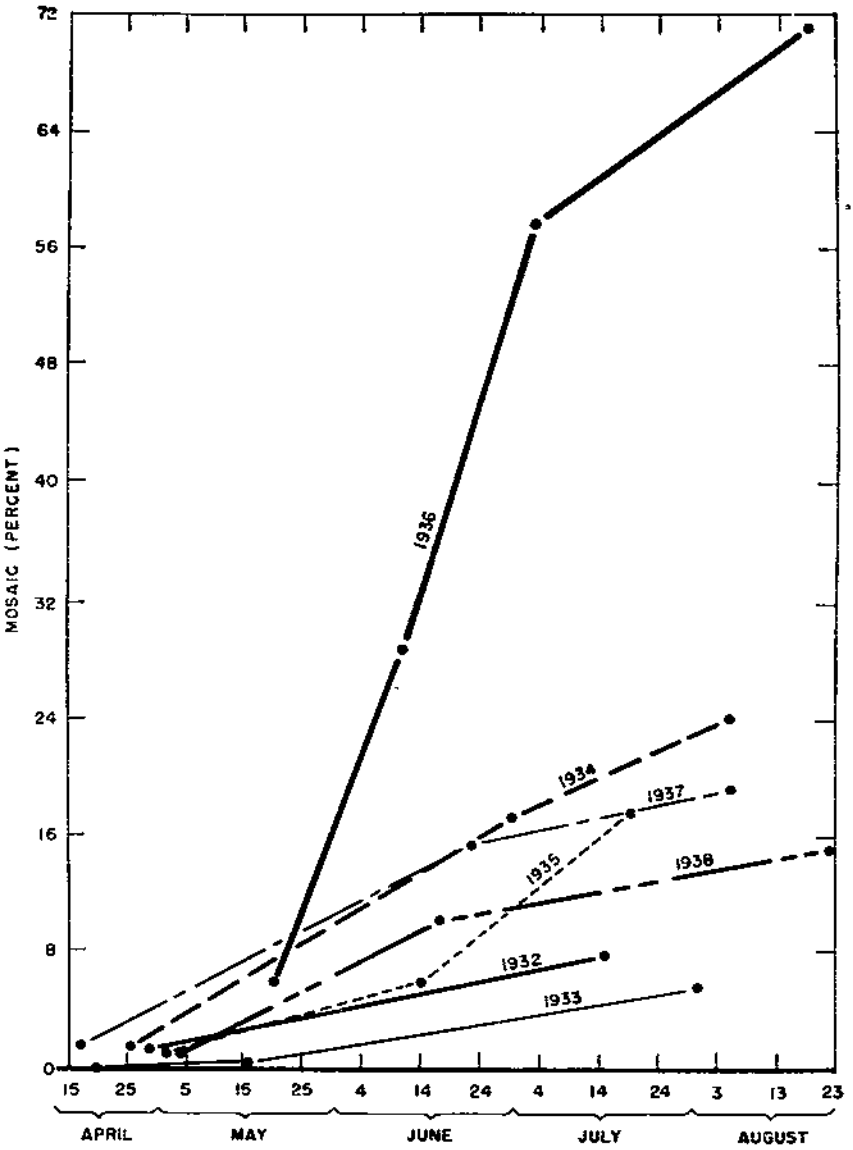

FIGURE 1.—Comparative rate of mosaic spread into healthy plantings of Co. 281 under comparable conditions of exposure during the 7 years 1932–38. The plantings for 1932 and 1936 were on the same area, which presumably was most favorably located for secondary spread. The 1934 planting was least favorably located; the others were intermediate in this respect.

mosaic spread for this period at the station were supported by extensive observations during the same period over the Louisiana sugar belt.

The data reported above covered only the first-year, or plant-cane, crops and are presented separately to show the extreme variability

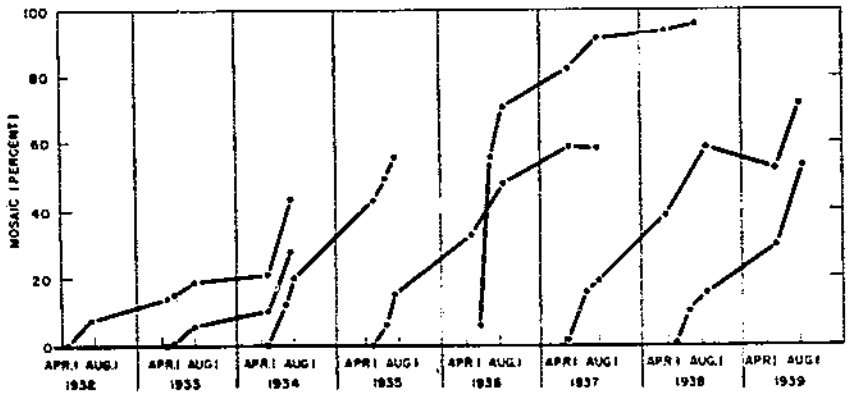

FIGURE 2.—Comparative rate of mosaic spread into healthy plantings of Co. 281 over the 8-year period 1932–39. Records are on new plantings each year and continued on successive stubble crops. The plantings for 1932 and 1936 were on the same area, which presumably was most favorably located for secondary spread. The 1934 planting was least favorably located; the others were intermediate in this respect.

from year to year in rate of mosaic spread into initially healthy plantings of sugarcane. These plots, however, were observed throughout the one or more succeeding stubble, or ratoon, crops, and the additional data on mosaic spread are presented in figure 2. It will be noted that the seasonal differences shown in the plant-cane study are in general evident also in the concurrent stubble crops. Thus the curves for a particular year tend to be nearly parallel, whether the crop be plant-cane or stubble. This is particularly evident for 1934, when very similar trends in rate of spread were obtained for plant-cane and both first and second stubble. An exception is 1936, which, however, is readily explained by the more favorable location of the plant-cane test that year.

SPREAD ON CO. 290

Limited data on mosaic spread in Co. 290, comparable with that reported for Co. 281, are shown in figure 3. The plant-cane crop for 1933 became infected to the extent of only 0.2 percent, and in the stubble crop the following year this percentage increased to 4.5, whereas the 1934 plant-cane crop, presumably in a field more favorably situated for secondary spread, showed a comparatively rapid spread (10.6). This was equaled only at the end of the third crop year in the crop planted in 1935. Unfortunately, no plant-cane test was available for comparable counts in 1936, but, judging by the comparatively low spread in the first stubble plots, Co. 290 appears to accumulate infection more slowly than Co. 281 (compare fig. 2). As pointed out in a later section of this bulletin, commercial experience with roguing confirms this finding.

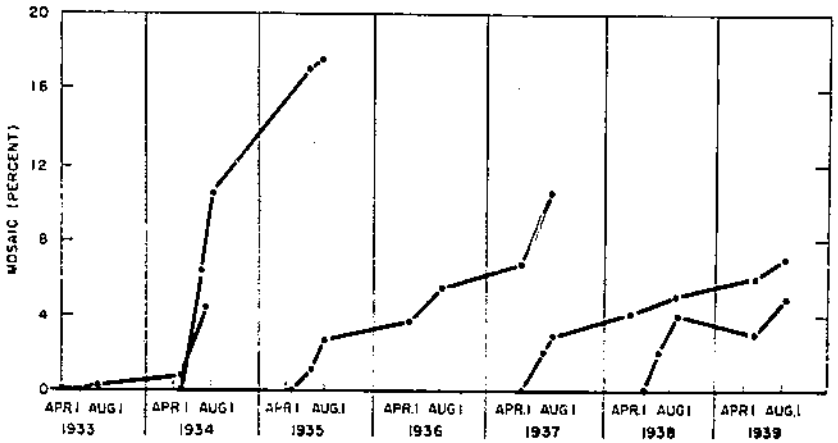

FIGURE 3.—Comparative rate of mosaic spread into healthy plantings of Co. 290 over the 7-year period 1933–39. Notes were made on new plantings each year and continued on successive stubble crops. No new plantings were made for the 1936 season.

EFFECT OF DISTRIBUTION OF INFECTION FOCI ON RATE OF SECONDARY SPREAD

An unusual opportunity was afforded in 1932–34 to obtain information regarding the effect of proximity and distribution of infection foci on the rate of secondary spread into healthy plantings of sugarcane. Identical plantings with 2 varieties, P. O. J. 36–M and Co. 281, were made in 1931 for the purpose of measuring the effect of mosaic on yield of single stools of cane. Each test was 4 rows wide and contained 144 stools of initially healthy cane, alternating in checkerboard fashion with an equal number of mosaic stools spaced 3 feet apart in the row. Each healthy stool was, therefore, adjacent to mosaic stools on either 3 or 4 sides, 3 feet distant in the row and about 6 feet apart between rows. Healthy buffer rows were planted on each side of the tests. An additional test with Co. 281, comparing yields from mosaic and healthy seed cane in paired, single-row replicated plots, 16 feet long, was located in the same field adjacent to the single-stool test of this variety. Opportunity for secondary spread in this row test was presumably the same as that in the others, as far as environment was concerned.

The results of this study are shown in figure 4. From the beginning, the great difference in the rate of mosaic spread on P. O. J. 36–M between the types of plantings was evident—47.4 percent in the single stools as compared with only 9.8 percent for the checkrow in July. These figures increased to 71.2 and 15.9 percent, respectively, in September, and to 87.3 and 46.1 percent in the summer of 1933. A gradual convergence of the two curves after the first year was due principally to two factors, the increase of local foci of infection in the checkrow and the approach of total infection for the variety, i. e., the balance between recovery and infection; this variety seldom if ever becomes 100 percent infected, because of the characteristic counteraction of foliage recovery. This convergence is even more

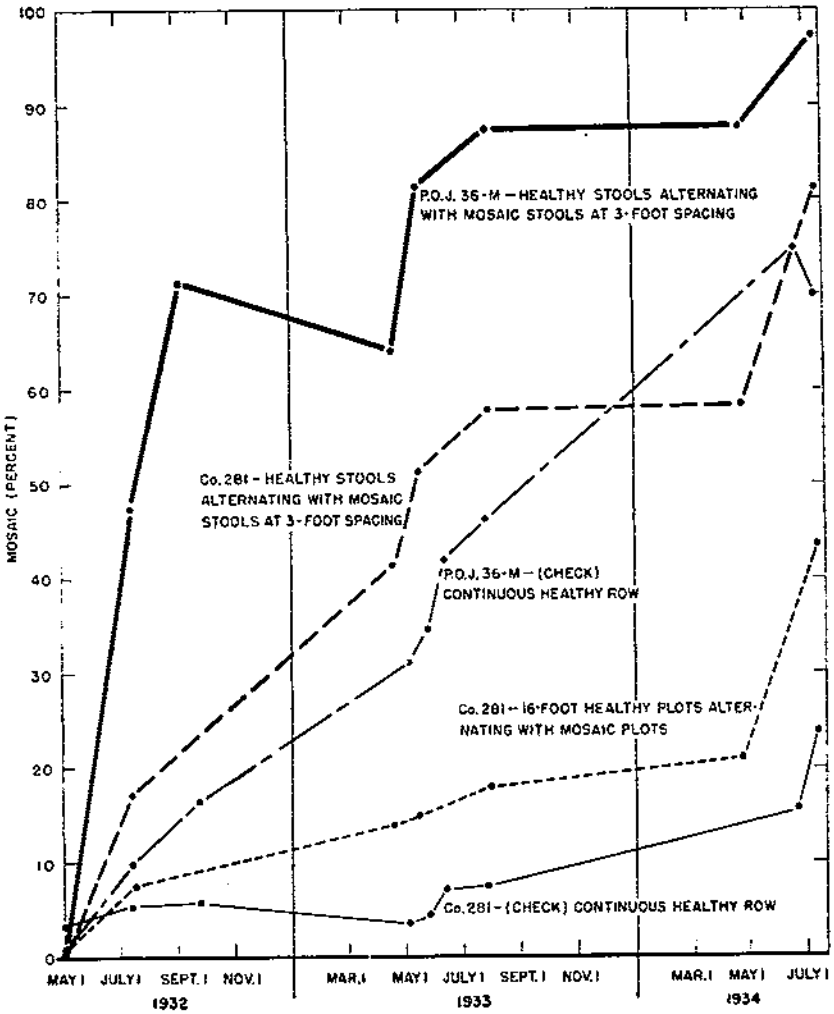

FIGURE 4.—Relation of proximity and distribution of infection foci to rate of mosaic spread into healthy plantings of P. O. J. 36–M and Co. 281 during the years 1932–34. The 3-foot spacing consisted of alternating healthy and mosaic stools, whereas the checkrows were entirely healthy but bordered by mosaic rows (approximately 6 feet distant) on either side; the 16-foot spacing had mosaic cane at both ends and sides of 16-foot healthy plots.

marked the third year, with very little increase in mosaic percentage in the single stools.

A similar development occurred in Co. 281, except that the spread was very much slower, particularly in the checkrow and in spite of an apparent primary infection of about 3.5 percent. This had increased to only 7.5 percent at the end of the second year, but went up to 22 percent the third year. The corresponding figures for the single stools, which started with no infection, were 57.8 and 80.6

percent, respectively. The 16-foot plots showed an intermediate degree of spread, approximating twice that for the checkrow.

Another series of yield tests, for which plantings were made in the fall of 1933, provided an opportunity for obtaining further comparative data on the apparent influence of local infection foci on the rate of secondary spread. Three varieties, P. O. J. 234, Co. 281, and Co. 290, were each represented by both single-stool and 16-foot-plot tests similar to those described above. The results, shown in figure 5, are fully in accord with those discussed above. Only plant-cane results are given here because of the succeeding stubble failure in P. O. J. 234 single-stool tests, which made further results unreliable.

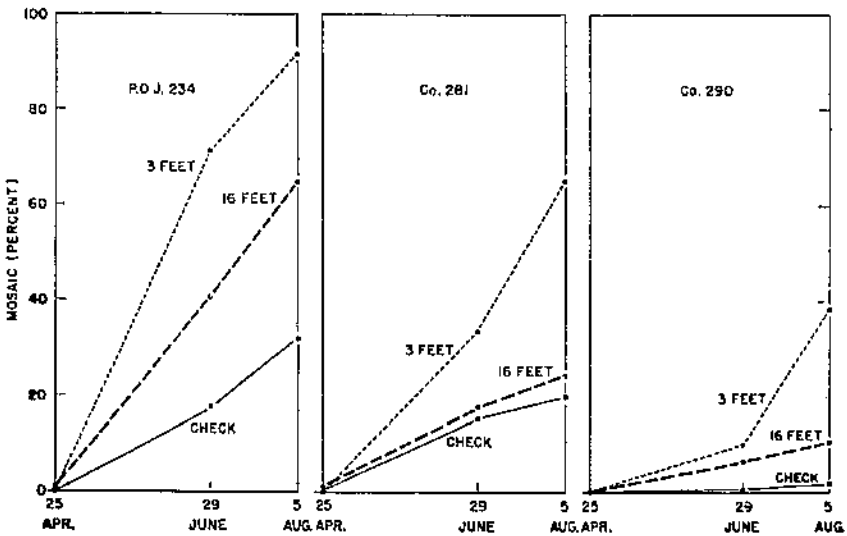

FIGURE 5.—Relation of proximity and distribution of infection foci to rate of mosaic spread in 1934 into healthy plantings of P. O. J. 234, Co. 281, and Co. 290, under conditions of comparatively light secondary spread. The 3-foot spacing consisted of healthy and mosaic stools alternated at that distance, the 16-foot spacing consisted of healthy and mosaic plots alternated in the same row, while the checkrows were entirely healthy but bordered by mosaic rows on either side.

Although the location of these plots was not considered favorable for secondary spread, an unusually heavy spread occurred in 1934. The final results for P. O. J. 234 were 91 percent for the 3-foot spacing, 64.6 percent for the 16-foot, and 31.6 percent for the checks. Corresponding results for Co. 281 were 64.8, 24, and 19.5 percent, and for Co. 290, 38.5, 10.6, and 1.8. These data give, incidentally, a fairly reliable comparison of the relative susceptibility of these three varieties to secondary spread.

The results obtained in 1932 and 1933 on the relation of infection foci to secondary spread prompted the planning of a test in 1934 for the specific purpose of measuring this effect more accurately. Two varieties, Co. 281 and Co. 290, were used in identical tests. Single-stool tests with two spacings, 3 and 4½ feet, were used as well as plots of two lengths, 8 and 16 feet. These were compared with a healthy

checkrow bordered on each side with mosaic rows as sources of inoculum. The location of these plots was considered ideal for obtaining maximum secondary spread.

The results (fig. 6) parallel those described above, particularly for Co. 290, and show that the rate of spread is, to some extent at least, inversely proportional to the distance between the diseased and healthy stools or rows. The secondary spread, however, was too heavy to get such sharp definition in Co. 281, there being a slightly greater spread in the 4½ than in the 3-foot spacings and less in the 8- than in the 16-foot plots and checkrow. The latter had the advantage of about 1.4 percent primary infection.

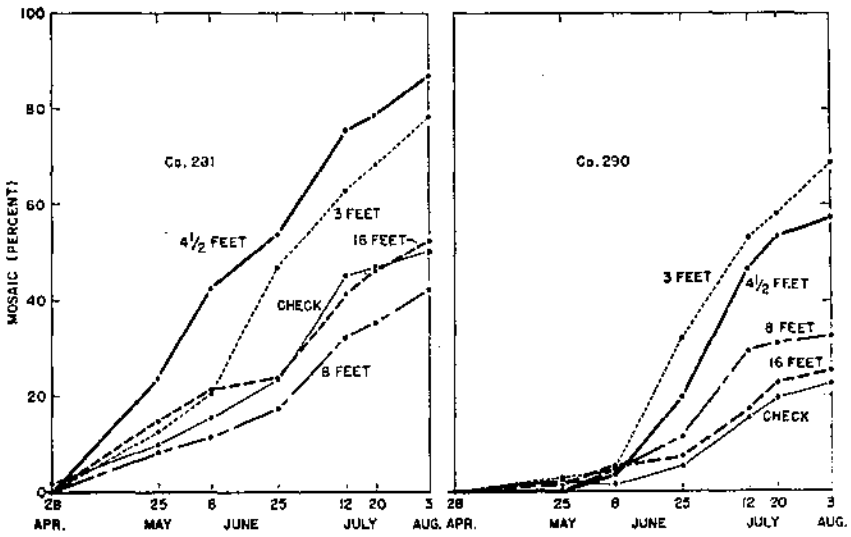

FIGURE 6.—Relation of proximity and distribution of infection foci to rate of mosaic spread into healthy plantings of Co. 281 and Co. 290 in 1934 under conditions of very heavy secondary spread. The 3- and 4½-foot spacings consisted of healthy and mosaic stools alternated at those distances, the 8- and 16-foot spacings consisted of healthy and mosaic plots alternated in the same row, whereas the checks consisted of healthy rows bordered by mosaic rows on either side.

The results presented in figure 6 indicate a very close relation between the number and proximity of infection foci and the extent of mosaic spread to nearby healthy plants of sugarcane. The alternate stools of healthy and mosaic cane at distances of 3 or 4½ feet were apparently close enough to permit movement of viruliferous aphids from mosaic to healthy cane much more freely or generally than occurred in healthy rows 8 or 16 feet long with mosaic cane at either end. This process seemed to be operative even before the plants had made sufficient growth to permit general intermingling of leaves from adjacent stools that might, conceivably, enable wandering aphids to go from one leaf to another on a different plant. In either event, the transfer of viruliferous aphids by ants, as one phase of their mutual-benefit relationship, may be the explanation for the differences noted

in rate of mosaic spread. Even though the vectors produce winged forms capable of flight at certain times, this seems to have been a very minor factor in the local spread of mosaic during the period represented here.

On the basis of these studies it seems logical to assume that, even though extremely high rates of spread were found at times (such as during 1934), the movement of viruliferous vectors was decidedly limited in scope and that proximity of infection foci was the controlling factor in rate of spread. Results from a single locality, of course, may not be representative of other areas in the Louisiana sugar district. Furthermore, possible occurrence of mass flights of viruliferous vectors (not, however, found in this study) would also tend to minimize the significance of this local plant-to-plant spread. Therefore, in the absence of direct information on vector movements, the extensive local spread here observed must have been caused largely by nonviruliferous aphids, whether from a local or a distant source, first colonizing the plants, and then, after becoming infective, transferring the disease from the mosaic to nearby healthy cane. Should these results be confirmed in other areas and years, they would have an important application in roguing for maintenance of mosaic-free areas.

Vectors

Transfer of sugarcane mosaic from diseased to healthy plants was first demonstrated in 1920 by Brandes (*8*), with the corn leaf aphid (*Aphis maidis*) as the vector. His results were confirmed by Kunkel (*48*) in Hawaii, and subsequently by many other investigators. Brandes stated that he did not believe that transmission of sugarcane mosaic was limited to this insect or even to other aphids. It remained the sole proved vector for 15 years, however, until Ingram and Summers (*45*) in 1935 demonstrated the ability of the rusty plum aphid (*Hysteroneura setariae*) to transmit the disease. They later (*46*) established the green bug (*Toxoptera graminum*) as a vector. Tate and Vandenberg (*67*), working in Puerto Rico, showed that *Carolinaia cyperi* Ainslie, a sedge aphid, was another vector. This aphid occurs in Louisiana but has not been observed on sugarcane there (*44*). Under favorable circumstances, any or all of these vectors may become potent factors in mosaic epiphytotics.

Although it is observed more commonly feeding on sugarcane in Louisiana than any of the other proved vectors, *Hysteroneura setariae* may be a relatively less efficient vector because of its habit of feeding on the collar lobe of the cane leaf rather than on the more succulent tissues in the whorl. It is somewhat hardier than the other aphids, as it is in evidence all winter on wild grasses bordering sugarcane fields when the other aphids are not to be found.

It was suggested by Brandes (*8*) and by Ingram, Haley, and Charpentier (*44*) that circumstantial evidence pointed to the sharp-nosed grain leafhopper (*Draeculacephala mollipes* (Say)) as a most likely vector, because of its abundance and activity in sugarcane fields. Numerous trials, however, have failed to confirm these suspicions. Extensive transmission experiments with other insects have always yielded negative results.

STRAINS OF THE MOSAIC VIRUS

Historical

The mosaic symptoms that had been observed on the different commercial varieties in Louisiana, as well as elsewhere, had, in general, shown slight but consistent differences, which seemed readily explainable as due to varietal reactions to a single virus. Thus, the several symptom patterns illustrated by Brandes (*7*) and Kunkel (*48*) were in all probability variations due to the reactions of individual varieties of diverse genetic constitution to a single virus, at least for each locality reported on. It is not unlikely, however, that the viruses concerned in the different countries were not identical.

Widely divergent types of symptoms have long been noted in the Louisiana seedling nurseries. Presumably these were caused by wide genetic differences and consequently by a greater range in host reaction or susceptibility. In the extremely susceptible class many seedlings exhibited very severe mosaic symptoms, their growth was greatly retarded, and they were either killed during the first season of infection or failed to germinate the following spring. Adjacent seedlings from the same cross, on the other hand, have often shown very mild symptoms and no visible growth retardation or other deleterious effect. Until 1932, however, no striking difference or unexplainable variation had been observed between individual plants of the same seedling (or clone), except in extremely rare instances.

The discovery of distinct strains of the virus resulted from studies of divergent patterns on the same clone or variety. In the fall of 1932 a variety, C. P. 28/60, was observed by the senior writer to show two radically different types of mosaic. On one stool a severe pattern, typified by extreme chlorosis and some necrosis, caused marked stunting; on an adjacent stool a mild pattern, barely discernible, had caused little if any growth retardation. Plantings were made, both in the greenhouse and in the field, with cuttings from each of these and from healthy cane of the same variety. With the exception of a few that recovered in the greenhouse, each symptom type was reproduced in the resultant shoots and was maintained through successive vegetative generations. In the field, the plants produced by the cuttings from stools showing the severe symptoms were rather scattered, owing to poor germination, and made a very poor growth the first year, with the first ratoon crop even worse; the other planting produced a good stand of plants with very mild symptoms that made a good growth, comparing favorably with the healthy cane at all stages. During the following year several additional seedlings of the C. P. 31 series were discovered, and these showed two correspondingly divergent types of mosaic.

At about the same time several stools were discovered that showed very severe mosaic symptoms, characterized by extreme chlorosis, severe necrosis, and pronounced stunting. These stools apparently resulted from shoots of a single stalk of cane of Co. 281, which was at one time the most widely planted commercial variety in Louisiana (46.7 percent in 1936). The stools were in a commercial field having a fairly high percentage of ordinary mosaic. Cuttings from stools showing severe symptoms, as well as from adjacent stools showing

ordinary symptoms, were planted in the greenhouse, and the two types have persisted through several vegetative generations, except for some variation in the degree of necrosis in the severe type, due presumably to differences in growing conditions.

Discovery of Differential Varieties

Numerous seedlings showing more than one type of mosaic symptoms have been discovered in connection with the routine progeny testing for mosaic resistance. Each year hundreds of seedlings produced at the Canal Point, Fla., breeding station are grown and studied at the United States Sugar Plant Field Station, near Houma, La. These are the result of a great number of crosses covering a wide range of highly divergent parent material. Because of the extreme genetic variability represented, the mosaic symptoms resulting from either artificial or natural infection may range from very mild to very severe, with almost every conceivable combination of mottling and striping, with or without stunting of growth.

In recent years it has been the practice to inoculate a large proportion of the new seedlings with viruses from two or even three sources, representing the above-mentioned types of mosaic (p. 44), and these often have produced divergent symptoms on the same variety. The extreme rareness of such in the earlier seedling nurseries, that is, prior to 1932, when natural infection alone was relied upon, would appear to have been due to predominance of a single strain or the actual absence of more than one strain of the virus in the station fields. Owing to the facts mentioned above and to the extreme heterogeneity of the sugarcane plant, it will be readily seen that an unusual opportunity was offered for observing a very wide range of reactions to mosaic by sugarcane varieties. Seedlings which showed a severe and distinctive leaf pattern and presence or absence of chlorotic leaf-sheath markings combined with vigor for rapid multiplication, and from which healthy seed could be obtained, seemed most promising for identification and differentiation of strains of the virus. More than 100 such seedlings were selected and have been tested under controlled conditions with virus from various sources for the double purpose of discovering hosts that would differentiate most clearly known strains as well as suspected new strains of the virus.

Comparative Infection Experiments

Materials and Methods

The inoculation technique most suitable for greenhouse work is essentially that described by Matz (*51*), in which the exposed spindle of tightly rolled young leaves is pricked a number of times through a small quantity of juice, freshly expressed from a mosaic cane plant and placed in the axis of the youngest open leaf in such a way as to adhere to and completely encircle the spindle. An alteration of this technique particularly applicable to large-scale work, where hundreds, or even thousands, of individual plants are inoculated in a single test, consists of merely dropping the juice into the open spindle and pricking through it. This method is much more rapid, is adaptable to a wider variety of conditions, and, although the virus is introduced into

slightly older tissues, produces equally high percentages (often 100) of infection without subsequent breaking or falling over of the young leaf at the point of inoculation. The pricking is done with a very small needle or small insect pin, equal success having been achieved with several different types.

For expressing the juice, meat grinders and fruit juice extractors have been used. The fruit juice extractor with its conical shaft is more desirable if a large quantity of juice is required or if there is ample material for grinding. The small meat grinder is more economical of limited supplies of inoculum and can be sterilized in a smaller container.

The grinders employed for obtaining the juice used in these tests were sterilized by boiling. Usually they were placed in water that was already boiling and left for some time after it had again come to a boil, and so also were the containers used for catching the juice and the pipettes, needles, vials, and other equipment. This treatment is considered sufficient, particularly in view of the low thermal death or inactivation point of the virus.

Leaves were always used as a source of juice. In earlier tests the blades only were used, but it was discovered that the more succulent roll of young leaves and sheaths was just as desirable so long as none of the very tender rapidly proliferating growing point was included. When the growing point was included the juice deteriorated more rapidly and could not be kept for any length of time. To make it more amenable to handling with pipettes, the expressed juice was strained through an ordinary 16-mesh screen into a small glass vial and, if not to be used within a very short time, was stored in an ice box. Whenever practical, all early inoculations were repeated on successive days or with one intervening day in order to obtain as high a percentage of takes as possible. Further experience showed this precaution to be unnecessary under most conditions, and it has been largely discontinued. All inoculation experiments were conducted in an aphidproof greenhouse that was fumigated frequently as an additional precaution.

RESULTS OF INOCULATIONS

Observation on two distinct types of mosaic and accompanying effects upon growth of the variety C. P. 28/60 (fig. 7) and their persistence during successive vegetative generations, supported by observations of two similar symptom types upon each of three other seedlings, indicated the existence of at least two causal entities. The further discovery of a severe mosaic pattern on Co. 281, distinctly different from the common pattern on that variety (fig. 8), suggested a third type. The significance of these observed variations was not very obvious at this stage, but the necessity of further detailed observations and of carefully conducted inoculation trials was recognized. Therefore in the fall of 1933 a series of experiments was instituted to compare the varied mosaic collections mentioned on page 45.

EXPERIMENT 4

Since C. P. 28/60 was the first variety to show two distinct types of symptoms, it was selected as a possible differential host. It is a

7. [illegible] C. P. 28-60 [illegible]

[illegible] hybrid with varieties of *Saccharum officinarum* [illegible] Louisiana Purple and D 74 [illegible] *S. barberi* [illegible] Chunnee [illegible] from Java and India [illegible] ancestry. Parallel [illegible] were [illegible] on Louisiana Purple [illegible] very susceptible [illegible] [illegible] This test, which was begun before [illegible] of [illegible] mosaic of Co. 281 was available in the greenhouse [illegible] [illegible] only C. P. 28-60, the three seedlings [illegible] fig. 11, [illegible] of which exhibited two types of symptoms [illegible] mosaic [illegible] the three commercial varieties P. O. J. 2[illegible], P. O. J. 36 M and Co. 281 [illegible] These presumably represented the mosaic population of the station.

The original inoculations were made on November 22 and 23, 1933, and all were repeated on November 21. A summary of the inoculations [illegible] experiment 1 is shown in table 3. A [illegible] produced on Louisiana Purple were typical of mosaic [illegible] observed on this variety, there being no greater variation between plants infected with [illegible] from different sources than between those infected with virus from the same source. The results of the [illegible] C. P. 28-60, however, were of quite a different nature. Mild symptoms were produced by all inoculations with juice from [illegible] of the four seedlings [illegible] C. P. 28-60 [illegible] only mild symptoms, and severe symptoms by the four corresponding lots of juice from plants that had shown severe symptoms [illegible] from P. O. J. 36 M and two lots of Co. 281, one of which had been called [illegible] because it had exhibited a noticeably more severe chlorosis in the field than is usually found in this variety.

TABLE 3.—*Summary of three inoculation tests on two differential hosts, indicating the existence and specificity of strains among certain collections on the sugarcane mosaic virus*

EXPERIMENT 4 [1]

Source of virus			Varieties inoculated				Designation of strain
			C. P. 28/60		Louisiana Purple		
Accession No.	Variety	Associated mosaic symptoms	Number of plants inoculated and infected [2]	Type of symptom	Number of plants inoculated and infected [2]	Type of symptom	
1	C. P. 28/60	Mild	10/9	Mild [3]	5/4	Ordinary [4]	A
2	do	Severe	10/9	Severe [5]	5/4	do	B
7	Seedling 196	Mild	10/8	Mild	5/5	do	A
8	do	Severe	10/1	Severe	5/2	do	B
5	C. P. 31/587	Mild	10/8	Mild	5/5	do	A
6	do	Severe	10/4	Severe	5/0	do	B
3	C. P. 31/589	Mild	10/5	Mild	5/4	do	A
4	do	Severe	10/4	Severe	5/3	do	B
9	P. O. J. 213	Ordinary	10/1	Severe (R) [6]	5/5	do	D
16	P. O. J. 36-M	do	10/3	Severe	5/5	do	B
17	Co. 281	do	10/9	do	5/5	do	B
18	do	Severe (DN) [7]	10/0		5/4	do	B

EXPERIMENT 6 [8]

1	C. P. 28/60	Mild	8/2	Mild	5/4	Ordinary A
1	Louisiana Purple	28/60 (Mild) [9]	8/7	do	5/5	do B
2	C. P. 28/60	Severe	8/5	Severe	5/5	do A
2	Louisiana Purple	28/60 (Severe) [9]	8/7	do	5/5	do B
10	P. O. J. 213	Ordinary	8/2	Severe (R)	5/5	do D
21	Co. 281	do	8/2	Severe	5/5	do B
22	do	Severe	8/5	Severe (R) [10]	5/5	Severe (R) D

EXPERIMENT 9 [11]

1	Louisiana Purple	Ordinary (28/60, Mild) [10]	6/6	Mild	6/6	Ordinary A
2	do	Ordinary (28/60, Severe) [10]	6/6	Severe	6/6	do B
21	do	Ordinary (281, Ordinary) [10]	6/6	Severe (R)	6/6	do D
22	do	Severe (Severe, R) [10]	6/4	do	6/6	Severe (R) C
21	Co. 281	Ordinary	6/6	do	6/6	Ordinary D
22	do	Severe (R)	6/2	do	6/4	Severe (R) C

[1] Original inoculations Nov. 22 and 23, 1933; repeated, Nov. 24.

[2] First figure of the fraction denotes number of plants inoculated; second, number infected.

[3] Mild mottling; little chlorosis; no noticeable stunting; no necrosis.

[4] Mottling and chlorosis intermediate; no necrosis or very marked stunting.

[5] Severe mottling and chlorosis; varying degrees of necrosis; marked stunting.

[6] No mottling but elongated white blotches, often coalescing; some necrosis and blighting or death of the growing point. R—Rosewood, the plantation where first observed.

[7] Delayed necrosis (DN), not very severe; appearing only on older leaves.

[8] Inoculated Jan. 9, 1934; repeated Jan 10.

[9] From experiment 4.

[10] From experiment 6.

[11] Inoculated Mar. 7, 1934; repeated Mar. 8.

FIGURE 8.—Symptoms produced by strain C (right) on the variety Co. 281 by natural infection, as compared with two healthy stalks.

Since no symptoms of any kind were discernible for some time on the C. P. 28/60 plants inoculated with juice from P. O. J. 213, and since 10 of the other 11 virus sources had apparently divided themselves into two natural groups, the logical conclusion seemed to be that there were at least two strains of the sugarcane mosaic virus present. Of these two it appeared that the severe strain identified from P. O. J. 36–M and Co. 281 was probably the mosaic that was common in the canefields, while the mild strain was a much less common one that found congeniality only in occasional seedlings.

As no variety had been observed that exhibited more than two types of symptoms, this hypothesis seemed to be tenable until one of the plants inoculated with juice from P. O. J. 213 began to show some symptoms of a rather unusual character for C. P. 28/60. These consisted of a few elongated, whitish lesions on the leaf, the plant

became stunted, and the growing point was blighted and eventually died. As a result several suckers appeared, most of which showed similar but even more severe lesions and similar blighting of the growing point, which sometimes resulted in the death of the shoot and sometimes in recovery and consequent resumption of growth. Since juice from this same source had produced ordinary symptoms of mosaic on Louisiana Purple, it seemed likely that this was a third type of symptom on C. P. 28/60 and also a third strain of the sugarcane mosaic virus. Table 3 shows that three types of mosaic patterns were differentiated on C. P. 28/60 (fig. 9) and that all of them produced indistinguishable symptoms on Louisiana Purple.

It is of more than passing interest to note, at this point, what was considered to be the very unusual behavior of the single plant of C. P. 28/60 that had been infected with virus from P. O. J. 213 and had exhibited a third and new symptom pattern on C. P. 28/60. Subsequent experience showed its behavior, as outlined separately in table 4, to be typical for this strain of mosaic and that what was considered at the time to be an exception and probably of little consequence was, in reality, of great significance in interpreting differences in the rate of mosaic spread, as discussed on pages 35 to 43.

In the first place, the fact that only 1 plant of 10 became infected and that the symptoms were slow in appearing is significant and in line with later inoculation tests. The next development of significance was the effect of this virus strain upon the growing points of the infected shoots. In some cases the growing points were killed rather early, as in the mother shoot and the first two suckers; whereas in the case of the third sucker, which seemed healthy at first, the growing point was apparently killed and then entirely recovered only to die down again. This wavering tendency may be due, in part at least, to the change in symptoms, whereby the original symptoms, characterized by long whitish lesions, were gradually replaced by mild symptoms. The mild symptoms were apparent in the fourth sucker from the beginning, and no growing-point injury appeared in it.

This stool was repotted and set aside for further observation, but no notes were made for several months. It is not definitely known, therefore, whether it was sucker 3 or 4 that survived, but it was probably the latter. This gradual change of symptoms, either partial or complete, from one type to the other was no accident in this case but is the rule with this virus strain when it is introduced into certain varieties of sugarcane. Repeated subinoculations from such varieties resulted in almost complete elimination of the originally severe symptoms.

EXPERIMENT 6

To check the results obtained in experiment 4, as well as to try out some additional virus sources, inoculations were made on January 9, 1934, and repeated the following day (experiment 6 of table 3). Juice was again obtained from the original stock of mild and severe mosaic plants of C. P. 28/60, as well as from Louisiana Purple plants infected from each in the previous experiments but showing identical symptoms. Since the original collection of P. O. J. 213 was from field material, another sample, with a known mosaic history, was obtained

FIGURE 9.—Three types of mosaic on C. P. 28/60. Stunted stools at left infected by mosaic virus strain B, large stool in center by strain A, and the next three irregular stools by strain D, which killed back some shoots and permitted others to make a fair growth. Stool at extreme right is healthy.

for this second test. Juice from each of the two collections of Co. 281 that were characterized by two widely different symptom patterns, as described on page 44, was also included. The results show reproductions of patterns on C. P. 28/60 characteristic of the original virus sources. Inoculations with P. O. J. 213 juice gave the same results as in the previous test, producing a symptom pattern on C. P. 28/60 readily distinguishable from that produced by either of the original collections from that variety.

The Co. 281 sample (accession 21) that exhibited only ordinary symptoms in the field proved to be harboring the same kind of mosaic as was the P. O. J. 213 (accession 10), while in the previous test the collections of Co. 281 had given the same reaction as the severe mosaic from C. P. 28/60 (accession 2). The other sample (accession 22) of Co. 281 that had exhibited such severe symptoms in the field produced the identical reaction on C. P. 28/60, but on Louisiana Purple it produced a very severe pattern, characterized, as was the Co. 281 in the field, by severe chlorosis and necrosis. This was the first time that symptoms of this sort had been found or produced on this variety, and so the logical conclusion was that a fourth strain of the sugarcane-mosaic virus had been differentiated.

EXPERIMENT 9

The two inoculation experiments 4 and 6 (table 3) gave clear indications that, with the possible exception of accession 18, the virus sources tested to date resolved themselves readily into four groups. Experiment 9 (table 3) then included subtransfers from the variety

TABLE 4.—*Symptom expression* [1] *and effect on growth of primary and secondary shoots of plant No. 6 of C. P. 28/60 inoculated in experiment 4 with virus collection No. 9 (mosaic, P. O. J. 213)*

Part of plant affected	Date examined in 1934					
	Jan. 29 and Feb. 15	Feb. 26	Mar. 12	Mar. 29	May 10	May 18
Primary shoot	Resembles severe mosaic, but plant seems backward (Jan. 29).	Severe (R)	Severe (R), growing point dead for some time.	Old leaves still green, but plant shows no growth.	No growth	No growth.
Secondary shoots:						
No. 1	Severe (R) (Feb. 15).	do	Severe (R), growing point apparently dying.	Growing point recovered, but leaves almost white.	Growing point dead, no growth.	Do.
No. 2		Severe (R), dying.	Dead			
No. 3		Apparently healthy.	Severe (R)	Severe (R), growing point seems to be dead.	Milder pattern, rapid growth.	Growing point dead
No. 4					Mosaic, but not very severe.	Severe (R) streaks, interspersed or scattered over rather mild general pattern.

[1] Severe = mottling and chlorosis intermediate; no necrosis over very marked stunting. Severe (R) = no mottling but elongated white blotches, often coalescing, some necrosis and blighting or death of the growing point. R = Rosewood, the plantation where first observed. Mild = mild mottling, little chlorosis, no noticeable stunting, and no necrosis.

Louisiana Purple, infected with virus from each of the four groups, i. e., the original mild and severe viruses from C. P. 28/60 and the two sources from Co. 281 (accessions 17 and 18), as well as direct transfers from the original material of the last two. The inoculations were performed on March 7, and repeated on March 8, 1934. As in the two previous tests, three distinct symptom patterns were produced on C. P. 28/60: (1) A mild mottling pattern, (2) a severe mottling pattern with necrosis and stunting, and (3) a pattern characterized by long whitish lesions, necrosis, and a lethal tendency on the growing point. Both sources of virus from Co. 281 produced the third type of mosaic on C. P. 28/60, but again produced two very different types on Louisiana Purple; the ordinary mosaic from Co. 281 produced ordinary symptoms for the variety, and the severe mosaic produced a similar severe pattern.

On the strength of these four inoculation experiments a note was published (*63*) in 1934 describing them as four types of the sugarcane mosaic disease. In that note and in similar brief reports in 1935 and 1936 (*64*, *65*) the mosaic types, or strains of the virus, were designated provisionally by numbers 1 to 4, respectively. In a further note (*66*) in 1938, letters (A to D) were substituted for numbers in designating these strains (see last column, table 3), which is in conformity with resolutions of the Committee on Virus Nomenclature of the Sixth International Botanical Congress (*47*). Strain numbers or letters were chosen for convenience in differentiation rather than with any idea of indicating relative prevalence or importance.

EXPERIMENT 10

Experiment 10, in which the four strains of virus A to D were used, was begun on April 5, 1934, and inoculations were again made on April 7 on Co. 281, P. O. J. 36–M, P. O. J. 213, and P. O. J. 234, the chief commercial varieties in Louisiana during the years immediately preceding these experiments. The object was to ascertain whether varietal reactions to the four strains would confirm previous findings. Five plants of each variety were inoculated with each virus strain, and a high percentage (usually 100) of takes was obtained. All reacted as anticipated; that is, they exhibited the same symptoms as Louisiana Purple in the previous tests. Ordinary symptoms typical of mosaic in commercial fields were produced on each variety by the three strains A, B, and D, and a severe pattern by the fourth (C), as it had been found naturally occurring on Co. 281 and had previously been produced by artificial inoculations on Louisiana Purple.

EXPERIMENT 14

Four strains of the sugarcane-mosaic virus having been so readily differentiated on two varieties of cane, it seemed highly probable that the use of more extensive virus sources and additional varieties as hosts would result in the discovery of additional strains. Three new C. P. varieties—29/143, 29/172, and 31/254—that in the field had shown promise of being of value as differential hosts because of the different symptom types observed on them were inoculated with the four strains of the virus. Five plants of each variety, including also C. P. 28/60 and Louisiana Purple as inoculated controls, were infected

with each strain in order to compare their reactions to the known strains.

This test was begun on November 16, 1934, and inoculations were repeated on November 19. The results of experiments 14 and 15 and other later tests have shown this time of the year to be rather unfavorable for beginning inoculation tests, owing presumably to short days and a minimum of sunlight. None of the new varieties proved to be so well adapted to strain differentiation as C. P. 28/60, because they were not only harder to infect but also failed to produce as distinct symptoms from the different strains. They were therefore discarded for use as differential varieties.

EXPERIMENT 15

Examination of about 900 C. P. seedlings, mostly from two complete progenies (Co. 281 × U. S. 1694 and Co. 281 × P. O. J. 2878) of the 1931 series, revealed that mosaic reactions departed somewhat from the usual in nearly 50 percent that had acquired the disease. Several of the diseased seedlings were selected that showed two or more symptom patterns, either separately or in apparent mixtures, as well as a few others that had attracted attention because the type of symptom found was unusual or very clear-cut. Particular attention was given to seedlings with type D symptoms, because seedlings in general seemed to be more congenial to virus strain B.

Juice, extracted from each plant selected, was used to inoculate four plants of C. P. 28/60 and three of Louisiana Purple. Inoculations were performed on November 17 and repeated on November 19, simultaneously with experiment 14. Unfortunately, this proved to be a rather poor season for the test, and the percentage of plants infected (table 5), particularly C. P. 28/60, was unusually low and, in many cases, the symptoms produced were not so readily interpreted as they might have been had growing conditions been more favorable. The experiment does serve to illustrate very clearly, however, that at least three strains of the virus were present in that nursery and that they had produced widely varying effects on many genetically different seedling varieties.

EXPERIMENT 18

Seven varieties of which a quantity of mosaic-free seed was available were selected from those used as mosaic sources in experiments 4 and 15 to be tried for suitability in strain differentiation. These varieties were selected chiefly because they showed at least two distinct types of symptoms in the field. C. P. 31/588, however, was selected because it showed such a perfect type D pattern that it was thought it might be particularly congenial to strain D of the virus. Five plants of each of these seven varieties, as well as C. P. 28/60, were inoculated with each of the four virus strains on January 2, 1935, and the inoculations were repeated 2 days later.

The conditions under which this test was conducted seemed to be ideal, as the plants grew rapidly and high percentages of mosaic, with clear-cut symptoms, were obtained (table 6). Six of the seven varieties showed such promise that they were retained for use in further tests in strain differentiation. C. P. 31/294, for instance,

TABLE 5.—*Identification of mosaic strains by symptom type produced on two differential hosts following inoculation with virus from a series of seedlings showing unusual mosaic patterns (experiment 15)*

Source of virus			Varieties inoculated				Virus strain indicated
			C. P. 28/60		Louisiana Purple		
Accession No.	Seedling No.	Associated symptom type	Number inoculated and infected[1]	Type of symptom	Number inoculated and infected[1]	Type of symptom	
56	C. P. 31/586	Type A	4/0		3/3	Ordinary	A, B, or D.
57	C. P. 31/586	Types A and D (mixed)	4/2	Type A	3/0		A.
58	54	Mild	4/0		3/2	Ordinary	A, B, or D.
59	54	Severe	4/0		3/2	do	
60	79	Type D	4/0		3/0		
61	99	Type A	4/1	Type B	3/3	Ordinary	B.
62	99	Type D	4/0		3/0		
63	99	Types A and D (mixed)	4/1	Type A (recovered).	3/2	Ordinary	A.
64	112	do	4/0		3/2	do	A, B, or D.
65	112	Type D	4/0		3/1	do	A, B, or D.
66	130	Type A	4/1	Type A	3/3	do	A.
67	130	Type B	4/0		3/0		
68	130	Type D	4/0		3/0		
69	C. P. 31/588	do	4/0		3/0		
70	245	Severe	4/0		3/0		
71	245	Types A and B (mixed)	4/2	Types A and B (mixed).	3/3	Ordinary	A and B.
72	C. P. 31/254	Mild	4/2	Type B	3/3	do	B.
73	C. P. 31/254	Severe	4/2	Type D	3/1	do	D.
74	214	Mild	4/3	Type B	3/3	do	B.

75	214	Severe	4/1	Type D	3/1	do	D.
76	C. P. 31/271	Mild	4/4	Type B	3/0	do	A.
77	C. P. 31/271	Severe	4/0		3/2	do	A, B, or D.
78	C. P. 31/590	Mild	4/3	Type A	3/3	do	A.
79	C. P. 31/590	Severe	4/1	Type B	3/2	do	B.
80	C. P. 31/591	Mild	4/4	Type A	3/3	do	A.
81	C. P. 31/591	Severe	4/0		3/0		
82	C. P. 31/294	Mild	4/1	Type A	3/3	Ordinary	A.
83	C. P. 31/294	Severe	4/1	Type B	3/2	do	B.
84	P–76	Mild	4/4	Type B (?)	3/3	do	B (?).
85	P–77	Severe	4/1	Type D	3/0		

[1] First figure of the fraction denotes number of plants inoculated (Nov. 17, 1934; repeated Nov. 19); second, number infected.

TABLE 6.—*Comparison of symptoms produced by 4 strains of the sugarcane-mosaic virus on C. P. 28/60 with those on 7 other seedlings selected as possible differential varieties because of their behavior in the field (experiment 18)*

Variety inoculated	Ratio of inoculated to infected plants [1] and type of mosaic symptoms produced by virus strain—				Totals	Remarks
	A	B	C	D		
C. P. 28/60	4/4—Type A	5/4—Type B [2]	5/3—Type D [2]	5/5—Type D [3]	19/16	
C. P. 31/586	5/5—Type A [4]	5/5—Type B [2]	5/5—Type D [3]	5/5—Type D [3]	20/20	Very congenial to all strains.
C. P. 31/588	5/5—Type A	5/5—Type D [2]	5/0	5/2—Type D [2]	20/12	Congenial to strains A and B, resistant to C and D.
C. P. 31/590	5/5—Type A	5/5—Type B [2]	5/2—Type D [2]	5/5—Type D [2 3]	20/17	Similar to but less congenial than C. P. 31/586.
C. P. 31/271	5/4—Type A	5/4—Type D [2]	5/0	5/1—Type D [2]	20/9	Strains B and D give similar patterns.
C. P. 31/587	5/5—Type A	5/5—Type B	4/3—Type D	5/5—Type D [3]	19/18	Symptoms comparatively indistinct.
C. P. 31/591	5/5—Type A	5/5—Type B [2]	5/2—Type D [2]	5/4—Type D [2]	20/16	Do.
C. P. 31/294	5/5—Type A	5/5—Type B [2]	5/4—Type D [2 3]	5/5—Type D [2 3]	20/19	Symptoms very definite, probably better than C. P. 28/60.
Total	39/38	40/38	39/19	40/32	158/127	

[1] First figure of the fraction denotes number of plants inoculated (Jan. 2, 1935; repeated, Jan. 4); second, number infected.
[2] Necrosis.
[3] Growing-point injury.
[4] One plant apparently recovered later.

has nearly replaced C. P. 28/60, because it shows more general congeniality and gives more definite symptom patterns at most stages. Later tests have shown its value in differentiating further strains. C. P. 31/586 is the outstanding variety thus far tested from the standpoint of congeniality. It is very susceptible to all viruses, but does not give quite so clear-cut symptoms as C. P. 31/294. C. P. 31/588, which exhibited a nice type D pattern in the field, is very susceptible to strains A and B, which are readily separated on the basis of severity of symptoms, but is very resistant to strains C and D. The mosaic seen in the field was probably caused by strain B, which produces a typical type D symptom in the greenhouse.

The one variety that showed the least promise was C. P. 31/271, which gave similar symptoms to strains B and D but no infection with strain C. The other three varieties reacted, in this test, similarly to C. P. 28/60 and C. P. 31/294, but did not give such clear mosaic patterns. In general they were also slower to show symptoms, although this fact is not reflected in the table. They were, however, retained and have since been used in further tests.

The totals at the bottom of table 6 indicate the relative infectivity of the different virus strains. Further tests, shown later, gave the same order of infectivity, although under most conditions strain D has not approached strains A and B so closely as in this test.

EXPERIMENT 23

In a mosaic survey conducted in Louisiana in 1934 to determine the approximate prevalence and distribution of the 4 virus strains previously noted, several collections were observed that did not behave in what was considered an orthodox manner when inoculated to C. P. 28/60. A number of these, which had seemed to be strain D or a mixture of two strains, were selected for further testing. A considerable degree of variation was noted in the effects produced by those collections that had been classified as strain D, particularly those from certain localities in isolated areas near the northern and western edges of the sugar-producing area. The variable collections of strain D were inoculated into 4 plants each of C. P. 31/294 and, insofar as the available plants permitted, C. P. 28/60, along with virus from the stock cultures of strains A, B, C, and D. The results, presented in table 7 show that, of 14 virus sources originally called strain D, 7 did not comply at all closely with the 6 that appeared to fall readily into line based on symptom expression and effect on plant growth. One virus source (No. 177) seemed to be intermediate. While these 7 virus collections produced initial symptoms closely approximating those produced by strain D, the lesions were very sparse or scattered, often only 1 or 2 on a leaf, and they were usually longer and somewhat more clearly defined. The importance of this difference, although rather obvious from the start, was not fully appreciated at the time because of the similarity of the variation of these two types of symptom from those produced by strains A and B. Their difference seemed to be one of degree rather than type.

It soon became evident in experiment 23, however, particularly on C. P. 31/294, that there was a great difference in the effect produced on the growth of the host plant. Strain D and the six collections that seemed identical with it caused a deleterious or lethal effect on the

TABLE 7.—*Deviations in symptom pattern produced on two differential hosts* [1] *by a series of apparent strain-D collections from northern Louisiana—four plants each infected in most instances (experiment 23)*

Virus strain or accession No.	Symptoms produced on variety—		Remarks
	C. P. 31/294	C. P. 28/60	
Controls:			
Strain A	Type A	Type A	
Strain B	Type B, growing-point injury.	Type B, growing-point injury.	
Strain C	Type D, growing-point injury, stunting, and death.	Type D, growing-point injury, stunting, and 1 dead.	
Strain D	Type D, growing-point injury and stunting.	Type D, slow, growing-point injury, stunting, and 2 dead.	
Accession No.:			
113	Type D, growing-point injury, severe stunting, and death.	----------	Typical strain D.
114	do	----------	Do.
117	Type D, growing-point injury and severe stunting.	Type D, growing-point injury, severe stunting, and death.	Do.
121	Mixture of symptoms	----------	
127	Type D, sparse symptoms and vigorous growth.	----------	Atypical strain D.
128	Probably a mixture	----------	
132	Type D, vigorous growth.	----------	Atypical strain D.
136	Type D, sparse symptoms, vigorous growth.	----------	Do.
144	Type D, growing-point injury, all plants died.	Type D, growing-point injury, all plants died.	Typical strain D.
145	do	do	Do.
146	Type D, growing-point injury, stunting, and 2 dead.	Type D, growing-point injury, stunting, and 1 dead.	Do.
151	Type D, sparse symptoms, vigorous growth.	----------	Atypical strain D.
158	do	----------	Do.
162	do	----------	Atypical strain D (recovery tendency).
166	do	----------	Very slow (recovery tendency).
174	Mixture	Mixture	
176	do	do	
177	Type D, growing-point injury and stunting.	Type D, sparse symptoms, good growth.	Atypical strain D.

[1] Four plants inoculated by each virus Apr. 30, 1935.

growing point, which usually was eventually followed by the death of the plant. Suckers were often produced only to be similarly affected. Those not killed were severely stunted. The plants infected by the other virus collections and classified as atypical strain D, on the other hand, showed no marked growth retardation. Although the individual lesions often were so extensive as to produce a rather striking effect, the growth of the plants approximated that of the healthy checks. Some of the plants even showed a tendency to throw off the symptoms entirely by the production of leaves with no lesions. No record of complete recovery, however, was made in this experiment. Therefore, on the basis of this test and experiments 45 and 48, these atypical strain D's have been designated strain E. The results obtained with inoculations from four of the virus collections indicated that they were probably mixtures of two or more strains.

EXPERIMENT 39

Experiment 39 included, as virus sources, the 4 original strains, 2 sources that had produced essentially the same symptoms as strain D but differed in other respects, and a virus obtained from C. P. 807. They were inoculated to 17 cane varieties, some of which had been used in previous tests and some that were used here for the first time. It was hoped that some variety might be found capable of differentiating either DW (13) or GS (18), or both, from strain D.

The DW (13) virus had originally been collected on P. O. J. 213 near Washington, La., and had been classed as strain D. It was found later, however, that cuttings of Co. 281 infected with this virus produced about 45 percent healthy shoots upon germination. Previous to this no germination recovery had been observed in this variety.

The GS (18) virus was collected on Greenwood plantation on Co. 281 and was producing symptoms noticeably more severe than was common in the fields there. Inoculations from it had, as in the previous case, produced type D symptoms on C. P. 28/60 and ordinary symptoms on such varieties as Co. 281, Louisiana Purple, and P. O. J. 234. Later observations on Co. 281 plants, both from inoculations and from the original cuttings, revealed that the older leaves, that is, the fourth or fifth open leaf and older ones, often were characterized by a comparatively mild necrosis. This was much less severe and not so persistent as the necrosis produced by strain C on the same variety.

Thus there were two collections of mosaic that complied with all requirements for strain D, both of which were, however, very different from it and from each other in one respect.

It was thought likely that the virus from C. P. 807 might be different from any of the others, because this variety had, previous to 1932, been considered to be immune to mosaic.

The inoculations reported here, the results of which are shown in table 8, were made on December 30 and 31, 1935. The number of available plants was limited in some varieties, and so there is considerable variation in the number inoculated. Probably the outstanding development in this test was the performance of the virus from C. P. 807, which, contrary to expectations, caused a very mild type of mosaic on every variety. The symptoms were much

TABLE 8.—*Comparison of symptoms produced by 7 sources of the sugarcane mosaic virus on 17 varieties of cane, including several seedlings not previously tried as differential varieties (experiment 39)*

Variety inoculated	Infection ratio [1] and type of mosaic symptoms produced by virus from different sources						
	Strain A	Strain B	Strain C	Strain D	DW (13)[2]	GS (18)[2]	C. P. 807
P. O. J. 36-M	9/9—Ordinary	8/8—Ordinary	9/9—Type C	9/7—Ordinary	8/4—Ordinary	7/7—Ordinary	8/5—Mild
P. O. J. 234	3/3—Ordinary	3/3—Ordinary	3/2—Type C	3/3—Ordinary	3/2—Ordinary	3/3—Ordinary	3/2—Mild
C. P. 28/60	9/7—Type A	7/7—Type B	8/3—Type D	9/4—Type D	8/3—Type D	9/2—Type D	9/5—Mild
C. P. 31/294	10/10—Type A	10/10—Type B	10/9—Type D	10/8—Type D	10/7—Type D	10/3—Type D	10/8—Mild
C. P. 31/254	6/6—Type A	6/6—Type D	6/2—Type D	6/5—Type D	6/5—Type D	6/2—Type D	6/6—Mild
C. P. 31/588	10/9—Type A	10/10—Type D	10/0	10/2—Type D	10/6—Type D	16/0	10/6—Mild
C. P. 31/271	10/9—Type A	10/9—Type D	10/1—Type D	10/1—Type D	10/3—Type D	10/0	10/6—Mild
C. P. 31/593	10/10—Type A	10/10—Type D [3]	10/10—Type D	10/9—Type D	10/8—Type D	10/5—Type D	10/5—Mild
C. P. 31/587	10/10—Type A	10/10—Type B	10/6—Type D	10/9—Type D	8/8—Type B	10/5—Type D	10/10—Mild
C. P. 31/589	10/10—Type A	10/10—Type B	10/7—Type D	10/10—Type D	10/8—Type D	10/1—Type D	10/9—Mild
C. P. 31/591	8/8—Type A	9/8—Type B	9/5—Type D	9/6—Type D	9/7—Type D	9/3—Type D	9/4—Mild
No. A-276	2/0	2/0	2/0	2/0	2/0	2/0	2/2—Mild
No. A-284	6/6—Type A	6/6—Type D	6/1—Type D	6/2—Type D	6/2—Type B	6/1—Type D	6/5—Mild
No. G-76	6/1—Type A	6/2—Type D	6/0	6/0	6/0	6/0	6/1—Mild
No. G-218	2/2—Type A	2/2—Type D	2/0	2/0	2/0	2/0	2/0
No. M-37	1/1—Type A	1/1—Type D	1/0	1/0	2/1—Type D	2/0	2/1—Mild
No. T-30	7/6—Type A	7/6—Type D	7/0	7/2	7/0	7/0	7/1—Mild
Total	119/107	117/108	119/55	120/71	117/64	119/32	120/76
Percentage of infection	90	92	46	59	55	27	63

[1] First figure denotes number of plants inoculated (Dec. 30 and 31, 1935); second, number infected.
[2] DW and GS, respectively, strain D variant from Washington and Greenwood, La., severe, typified by necrosis appearing only on older leaves of infected plants.
[3] Symptoms reported as type B in experiment 18, table 6.

less evident than those produced on similar plants by strain A; in general, they did not appear until 1 to 3 weeks later.

The anticipated differentiation of the DW and GS collections from strain D did not materialize, the slight differences noted here being caused presumably by growing conditions, which were apparently not so desirable as in most previous tests, and to the virus source. In explanation of the source it had been noticed for some time that there was a tendency for strain D to produce not only type D symptoms but also what appeared to be type A symptoms on C. P. 28/60 and C. P. 31-294. The type A symptoms appeared first as an admixture, both patterns being mingled on the same leaf, and then later the type D symptoms on the younger leaves failed entirely. Subinoculations from such plants would usually produce the mild type of symptom. This phenomenon was for some reason more in evidence than usual during this test, not only with strain D but also with DW, which further emphasizes the close relationship of these two viruses. The data obtained here, therefore, could not be used for separating these viruses, even though the reactions were probably typical for the conditions of this test. The DW virus, for instance, was reported as producing type B symptoms on C. P. 31-587 and A 284, while strain D and the GS virus produced type D symptoms. Only about half the number of infections were obtained with the GS virus as with the other two.

Final evidence as to whether the differences in infectivity and symptom behavior noted above have diagnostic value depends on repetition of the inoculations in question, which are reported in subsequent experiments.

EXPERIMENT H

The inoculations reported in table 9, made on March 31, 1936, include an assortment of collections that appeared to be different from those previously identified or that were difficult to classify. They were grouped together more for convenience than for comparison, each presenting its own particular problem.

Acc. 403.—A stool of Co. 290 affected with a severe necrotic condition was called to the senior writer's attention on Southdown plantation. This condition persisted in plants from vegetative cuttings of this material, but no mosaic symptoms were apparent, although they did appear later, apparently as a result of secondary spread. An early attempt to transfer mosaic from this source to D 74 and C. P. 31-294 resulted in a very much delayed case of mosaic in one plant of C. P. 31-294. The symptoms were rather odd, with a tendency for the lesions to be in islands without necrosis. Several months later the symptoms became more severe, resembling those produced by strain B. The results of the present experiment on C. P. 31-294 and C. P. 31-588 indicate that it is probably strain B, but inoculations back to Co. 290 failed to produce anything like the original necrotic symptoms. It seems highly unlikely that the virus obtained and used here was responsible for the symptoms originally observed on Co. 290.

Acc. 407. Two stools of C. P. 28-19, submitted from Rosewood plantation, were severely dwarfed and had stooled out excessively. The leaves were rather mottled, with some resemblance to mosaic.

TABLE 9.—*Strain determinations on miscellaneous and difficultly transferable virus collections (experiment 44)*

Virus collection accession No.	Variety	History and symptoms	Infection ratio [1] and type of symptoms produced on variety—						
			Co. 290	C. P. 31/294	C. P. 31/588	C. P. 28/19	C. P. 807	C. P. 31/587	C. P. 31/589
403	Co. 290	Single stool, mosaic apparently associated with ring mosaic.	20/16—apparently ordinary mosaic.	10/10—Probably type B, but not entirely typical.	10/10—Type D, but milder in early stages.				
407	C. P. 28/19	Severely dwarfed stool, with questionable mosaic.		10/0	10/0	20/0			
409	do	Very mild mosaic, which produced mild symptoms on P. O. J. 234.		10/0	10/0	20/0			
410	Co. 281	Ordinary mosaic, plus sheath discoloration.		10/10—Type D, necrosis.	10/7—Type D, necrosis.				
430	C. P. 807	Severe on C. P. 807, but very mild and slowly developing on other varieties.			10/2—Very mild and slow.		20/0	10/10—Very mild, some even doubtful, at times slow.	10/9—Mild, but distinct; slow, but compatible.
Strain A (control).	C. P. 28/60	Mild, rapidly developing.			10/10—Type A		20/0	10/10—Type A	10/10—Type A+, i. e., very distinct but no necrosis.

[1] First figure of the fraction denotes number of plants inoculated on Mar. 31, 1936; second, number infected.

Inoculations with expressed juice failed to produce any infection except on one plant of P. O. J. 234 inoculated by the leaf-puncture method, which showed mild mosaic that did not appear until about 2 months after inoculation. The symptoms later became very faint, however, and no infection was obtained when this plant was used as a source of inoculum in the present experiment.

Acc. 409.—Mosaic, whenever it occurred on C. P. 28/19, usually produced very severe symptoms, but an occasional stool was found with extremely mild symptoms. One such source was transferred to P. O. J. 234 by the leaf-puncture method and gave a very mild infection similar to that produced by 407. Attempted subtransfers, however, failed in this case, as before.

Acc. 410.—A stool of mosaic Co. 281 that exhibited a marked purplish discoloration of the leaf sheath was observed in the field. No such discoloration had previously been noted in this variety. Inoculations in experiment 44 to C. P. 31/294 and C. P. 31/588 produced typical type D symptoms on both varieties. The reaction was not, however, exactly typical of strain D, in that a rather high percentage of infection was obtained on C. P. 31/588, which had consistently proved highly resistant to infection by strain D. Frequently no infections at all were obtained.

Acc. 430.—The virus originally obtained from C. P. 807 was included here for two reasons: (1) To compare it further with strain A and (2) to attempt to transfer it back to C. P. 807. The transfer failed as expected, because of the extreme rarity of mosaic occurrence on this variety in the field. The comparison with strain A on three other varieties confirmed previous findings that much milder symptoms are produced by the C. P. 807 virus and that in general they are much slower in appearing. The variety C. P. 31/589 seemed to be more compatible to this virus than any other that had been inoculated with it.

EXPERIMENT 45

The objective of experiment 45 was primarily to discover additional and better differential hosts, and secondarily to define more clearly six aberrant virus collections.

A number of seedlings had been selected in the fall of 1935 from the complete progeny nurseries of the C. P. 32 and C. P. 33 series because their mosaic reaction indicated possible utility in strain differentiation. Five of these (A–276, A–284, G–76, M–37, and T–36) were used in experiment 39 (table 8), and all were increased in the field under conditions of minimum spread (only 2 or 3 cases of mosaic were noted in a large planting). By the following summer 14 of them were available in sufficient quantity to be used in the larger scale experiment. These, together with 10 C. P. seedlings, most of which had been used in tests 18 and 39 (tables 6 and 8), and 5 commercial varieties, totaled 29 varieties, and each was inoculated with mosaic from 10 sources.

The virus used for inoculum included accessions 436 and 479, both collected on C. P. 29/291, showing no signs of the characteristic leaf-sheath discoloration usually found accompanying mosaic on this variety, and accession 435 from Co. 281, which upon being germinated produced some healthy shoots and others in which the symptoms

wavered back and forth, almost disappearing at times. Preliminary trials had indicated that accession 436 was similar to strain B, although it seemed a little milder on some hosts. The further fact that it produced no sheath discoloration or stunting of C. P. 29/291, however, indicated that it was different from strain B. Simultaneous trials had shown accessions 435 and 479 to be similar, if not the same. On C. P. 31/294 they produced the type of symptom that has been noted above for certain collections from outlying sugarcane areas of Louisiana, i. e., similar to type D, but larger lesions occurring more sparsely on the leaf and causing little or no growth retardation of the infected plant. This type of symptom is here referred to as type E and will be so treated in subsequent discussions. The remaining viruses used were the original four strains and DW, 410, and GS. The last three seem to be variants of strain D and are discussed on page 63.

The results of this test, the inoculations for which were made August 19 and 20, 1936, are presented in table 10. With the exception of two varieties, P. O. J. 234 and G 76, five plants of each variety were inoculated with mosaic from each source and five were held as uninoculated checks. Occasional plants died before it was possible to get their mosaic reaction, and these were not included in the tabulated figures. Such cases are reflected in the infection ratios. In two varieties, A–148 and T–617, single mosaic plants appeared in the supposedly healthy checks. Both were undoubtedly of primary origin, as they appeared so early in the test. Their appearance would, of course, discount the inoculation results on these two varieties to some extent. Later observations indicate that A–148 may be a symptomless carrier of mosaic at times and that latent virus is sometimes present in apparently healthy plants.

Among the most interesting and significant results of this experiment, although incidental to the main objects of the test, are the totals presented at the bottom and right side of the table. The percentages shown at the bottom are significant, since they give what seems to be an accurate infection index of the different virus sources used. The writers' experience in inoculating thousands of Canal Point seedlings has failed to bring to light a single authentic case in which a seedling was more readily infected with strain D than strain B, these being the only two pure strains that have been widely used for this purpose. A similar relation has been noted in inoculations to mosaic-susceptible commercial varieties. It seems very likely, therefore, that since a total of 29 varieties are involved here, the difference between infection percentages of 78.1 for strain B and 38.1 for strain D is highly significant. It seems improbable that the relatively small differences noted between strains D, DW, GS, and 410 have any significance, but the generally lower percentages noted for strain C and accessions 479 and 435 are undoubtedly indicative of their lower infectivity.

The figure of 61.0 percent for accession 436 further confirms the premise that it is probably not identical with strain B, although ordinarily producing identical symptoms on most of the varieties in this test. The symptoms, however, are frequently much slower in appearing. The extremely high figure of 86.8 percent for strain A and 78.1 percent for strain B, as compared with 38.1 percent for strain D, are in general agreement with those presented in table 8. Despite its easy infectivity strain A has rarely been collected in the field except

in the seedling nurseries where it has been found occurring only on seedlings that show both mild and severe mosaic. It shows about the same relative infectivity on commercial varieties, persisting in pure form on Co. 281 for several years and through several vegetative generations, and there seems no reason why it should not be of general occurrence in the field. There is, of course, a possibility that its advent upon the local scene is comparatively recent or, possibly, that a specificity in vector relations may exist. Investigation of both points is desirable.

The percentage figures in the column at the right of table 10, while of general interest, are less significant for several reasons than those just discussed. In the first place, the mosaic sources used could not in any way be considered representative of the mosaic population that any variety would encounter in the field under usual circumstances. Secondly, the artificial inoculation methods used here disregard all the variables, with the possible exception of protoplasmic resistance, which conceivably to some extent may determine resistance to mosaic under field conditions. This would apply particularly to the varietal preference, if any, of vectors. This is forcibly illustrated by the first five varieties listed in table 10, all of which are or have been important commercial varieties in Louisiana. While the percentages rank them in the order of their susceptibility under field conditions, the actual percentages are very misleading. The differences between the varieties would be relatively much greater under conditions favorable for maximum infection in the field. In other words, a moderately resistant variety gets a relatively much more severe test by artificial inoculation than it would under the severest exposure in the field.

Among the C. P. seedlings, it will be noted that C. P. 31/294, our best differential variety, showed 72 percent infection as compared with 40 percent for C. P. 28/60, although C. P. 31/294 was not infected at all by three viruses in this test. One seedling, C. P. 31/586, gave 81.6 percent infection but did not give such clear-cut symptom patterns as C. P. 31/294. Its congeniality to all virus sources, however, may make it valuable as a differential host, particularly for that group of the virus collections that seem to find so few congenial host varieties.

C. P. 31/588 developed only 40.8 percent mosaic because it showed such high resistance to all virus sources except the strain A and B groups. This resistance amounts almost to immunity in the case of strain C, and usually it shows much less infection by the strain D group than in this test. C. P. 31/590 seems to draw a fairly definite line between the regular strain D group and accessions 479 and 435. Plants, if any, infected by the two viruses show type E symptoms and a very strong recovery tendency, presumably because of the inability of the virus to establish itself. A similar tendency is very common with certain seedlings and often with most of the viruses used.

The three seedlings C. P. 31/587, 31/589, and 31/591 all showed around 50-percent infection. The first two will probably have a limited use as differential varieties, because, as shown in previous tests, they were very congenial to mosaic obtained from C. P. 807, but 31/591 will probably be of no use at all. In the present test there was some evidence of a qualitative difference in the effects produced by strains D and DW, the latter apparently causing more pronounced stunting than the former. Symptom types D and E

were very clearly defined on C. P. 31/589, and it is conceivable that it might prove of value for demonstrating this difference.

The other seedlings used in experiment 45 were selected from the complete progeny nurseries of the C. P. 32 and C. P. 33 series because of their mosaic histories in the field and are here referred to by their original progeny numbers. Most of them showed little promise as differential varieties, and they were generally much lower in percentage of infection than those just discussed. One of the main objectives in selecting this group of seedlings was to obtain varieties that would be congenial to strain D and the group of viruses that seem to be so similar to it. Those selected, therefore, were the ones that showed typical, clear-cut, type-D mosaic symptoms, on the assumption that they had possibly been infected with strain D virus. The inoculum was obtained from field cane, variety Co. 281, in buffer ends of the seedling plots on the experiment station at Houma, and represented largely secondary spread. While strain B is the dominant virus in the section of Louisiana adjacent to Houma, strain D occurs frequently and should have been present in some of the plants. The results of experiment 45 indicate, however, that strain B predominated, because with the possible exception of T–617, which gave better than average infection with the strain D group of viruses, none of the varieties gave a type B reaction with strain B virus. From these results it seems natural to assume, particularly since strain D never has produced type B symptoms, that the selection procedure in the field should be reversed unless it is positively known that strain D virus has been used on the varieties being considered for selection. Brief mention of noteworthy results from the inoculations of this group of seedlings will be made here.

Seedling No. A–148 seemed to show a decided preference for strain A, although symptoms were slower in appearing than with some other viruses. These latter, however, showed a number of inconsistencies, traceable possibly to the fact that primary mosaic appeared in one of five healthy check plants and so may also have been present in some inoculated plants. This, of course, was not unexpected, as the cuttings were grown in the open where a small quantity of secondary spread was observed. Later observations have shown that this variety sometimes harbors mosaic in the absence of visible symptoms. Seedling A–254 also seemed congenial to strain A, since it showed more severe chlorosis from this strain than had ever been observed on any other seedling. On seedling A–276 virus accession 436 seemed to hold the ascendency, both qualitatively and quantitatively. This was particularly noticeable in the comparison with strain B, which seemed to be most like accession 436.

The outstanding development with A 284 is that of 15 plants inoculated with the groups of virus strains A and B 10 became infected, but in the 34 plants inoculated with virus from the other 7 sources not a single infection occurred. This line of demarcation is even sharper than that noted above for C. P. 31 588 and may possibly denote complete immunity to the latter group of strains.

The three seedlings G–76, G–112, and G 284 seem to offer little as differential varieties. The first two particularly showed very low infection percentages and symptoms were slow in developing. In the case of G 284, there seemed to be a decided preference for strains

TABLE 10.—*Comparison of symptoms produced by 10 variants of the sugarcane mosaic virus, including 4 distinct strains, when inoculated on 29 host varieties (experiment 45)*

Variety	Ratio [1] infection and types of symptoms produced by virus					
	Strain A	Strain B	Acc. 436	Acc. 479	Acc. 435	Strain DW
Co. 281	5/5—Ordinary	5/4—Ordinary	5/4—Ordinary	5/1—Ordinary [2]	5/0	5/2—Ordinary
Co. 290	5/5—Ordinary	5/5—Ordinary	5/2—Ordinary [4]	5/0	5/1—Ordinary [5]	5/1—Ordinary [5]
Louisiana Purple	5/5—Ordinary	5/5—Ordinary	5/5—Ordinary	5/3—Ordinary [6]	5/5—Ordinary [6]	5/4—Ordinary
P. O. J. 36-M	5/5—Ordinary	5/5—Ordinary	5/5—Ordinary	5/2—Ordinary [6]	5/4—Ordinary [6]	5/5—Ordinary
P. O. J. 234	3/3—Ordinary	3/2—Ordinary	3/3—Ordinary	3/1—Ordinary	3/2—Ordinary	2/1—Ordinary
C. P. 28/60	5/4—Type A	5/5—Type B [5]	5/4—Type B [5 6]	5/1—Type E [3 9]	5/0	5/2—Type D [3 5 6]
C. P. 31/291	5/5—Type A	5/5—Type B [3 5 10]	5/5—Type B [3 5]	5/1—Type E [3 11]	5/3—Type E [3 11]	5/3—Type D [3 5 12]
C. P. 31/254	4/3—Type A	5/4—Type B [3]	5/2—Type B [3]	5/0	5/0	5/5—Type D [3 9]
C. P. 31/271	5/3—Type A	5/3—Type D [3]	4/1—Not determined	5/0	5/0	5/1—Type D [3 13]
C. P. 31/586	5/5—Type A	5/5—Type B [5]	5/5—Type B [5]	5/3—Type E [3]	5/4—Type E [3]	4/3—Type D [3 12]
C. P. 31/587	5/4—Type A	4/4—Type B	5/3—Type B	4/0	5/1—Type E [5]	5/4—Type D [5]
C. P. 31/588	5/5—Type A	5/5—Type D [3]	5/4—Type D [3]	5/0	5/0	4/2—Type D [3]
C. P. 31/589	5/5—Type A	5/3—Type B [3]	4/1—Type B	5/1—Type E [3 2]	5/4—Type E [3]	5/2—Type D [3]
C. P. 31/590	5/5—Type A	5/5—Type B [3]	5/5—Type B [3]	5/3—Type E [3 9]	5/0	5/4—Type D [3 5]
C. P. 31/591	5/5—Type A	5/4—Type D [3]	5/4—Type D [3]	5/1—Type E [3 9]	5/0	5/3—Type D [3]
A-148	5/5—Type A	5/3—Type D [3]	4/3—Type D [3]	3/0	4/1—Type D [3]	5/2—Not determined
A-254	5/5—Type A	5/3—Type D or B [14]	5/2—Type D or B [14]	5/0	5/0	5/1—Type D [3 12]
A-276	5/2—Type A [6]	5/1—Type D [3 13]	5/3—Type D [3 15]	5/0	5/2—Type E [3 16]	5/1—Type D [9]
A-284	5/4—Type A	5/4—Type D [3]	5/2—Type D [3]	4/0	5/0	5/0
G-76	3/2—Type A	3/1—Type D [3]	3/0	3/0	3/0	2/0
G-112	4/1—Not determined [6 9]	5/0	5/1—Type D [6]	5/0	5/0	5/0
G-284	4/4—Type A [6]	4/2—Type D [3 6]	4/1—Type D [3 6]	5/0	4/0	5/3—Type D [3 9]
K-89	3/2—Type A	4/4—Type D [3 5]	5/3—Type D [3 5]	5/2—Type E [3 9]	5/2—Type E [3 9]	5/1—Not determined [14 5]
K-129	5/4—Type A	5/3—Type D [3]	4/2—Type D [3 9]	5/1—Type D [3 9]	5/3—Type D [3 9]	5/1—Type D [3 9]
M-37	5/5—Type A	5/4—Type D [3]	5/3—Type D [3]	5/1—Not determined	5/2—Type D [3 9]	5/3—Type D [3 13]
T-36	5/2—Type A	5/5—Type D [3 9]	5/2—Type D [3 9]	5/1—Not determined [9]	5/0	5/1—Type D [3]
T-97	5/5—Type A	4/4—Type D	5/4—Type D	5/0	5/0	5/2—Type D [13]
T-116	5/5—Type A	5/5—Type D [3]	5/2—Type D [3]	5/2—Type E [3 9 13]	5/1—Type E [3 9 13]	5/0
T-617	5/5—Type A	5/4—Type B [15]	5/2—Type B [15]	5/4—Type E [3 9 6]	4/3—Type E [3 6]	5/1—Type D [3 12 6]
Totals	136/118	137/107	136/83	137/28	139/38	137/58
Percentage of mosaic	86.8	78.1	61.0	20.4	27.3	42.3

See footnotes at end of table.

TABLE 10.—*Comparison of symptoms produced by 10 variants of the sugarcane mosaic virus, including 4 distinct strains, when inoculated on 29 host varieties (experiment 45)*—Continued

Variety	Ratio [1] infection and types of symptoms produced by virus—Continued				Plants inoculated		Remarks
	Strain D	Acc. 410	Strain GS	Strain C	Total	Percentage of mosaic	
Co. 281	5/4—Ordinary	5/3—Ordinary	5/4—Ordinary	5/2—Type C [3]	50	58.0	Note apparent resistance to Nos. 479 and 435.
Co. 290	5/1—Ordinary	5/2—Ordinary [6]	5/4—Ordinary [6]	5/0	50	42.0	Most congenial to strains A and B.
Louisiana Purple	5/3—Ordinary	5/5—Ordinary [6 7]	5/4—Ordinary	5/4—Type C [6]	50	88.0	Highest percentage of takes.
P. O. J. 36-M	5/3—Ordinary	4/1—Ordinary	5/5—Ordinary	5/2—Type C	49	75.5	
P. O. J. 234	3/2—Ordinary	3/2—Ordinary	3/3—Ordinary	3/2—Type C	29	72.4	
C. P. 28/60	5/2—Type D [3]	5/2—Type D [3 5]	5/0	5/0	50	40.0	Congenial to strains A and B.
C. P. 31/294	5/5—Type D [3 5 12]	5/3—Type D [3 5 12]	5/4—Type D [3 5 12]	5/2—Type D [3 5 12]	50	72.0	Shows 4 very distinct symptom types. Growth of strain A equal to healthy checks.
C. P. 31/254	5/3—Type D [3 9]	5/1—Type D [3]	5/0	5/0	49	36.7	Symptoms indeterminate, low percentage of infection.
C. P. 31/271	5/0	5/0	5/0	5/2—Type D [13]	49	20.4	Very low infection index.
C. P. 31/586	5/4—Type D [3 12]	5/3—Type D [3 12]	5/5—Type D [3 12]	5/3—Type D [3 12]	49	81.6	Very high infection index; shows 4 types, but not quite so clearly as C. P. 31/294.
C. P. 31/587	4/1—Type D	5/3—Type D [3 5]	5/3—Type D [3 5]	5/2—Type D [12]	47	53.2	Similar to above; seemed also to be less stunted by strain D than by DW.
C. P. 31/588	5/1—Type D [3]	5/2—Type D [3 9]	5/1—Type D [3 2]	5/0	49	40.8	Very resistant to all but strains A and B.
C. P. 31/589	5/3—Type D [3]	4/2—Type D [3]	5/4—Type D [3]	5/1—Type D [3]	48	51.2	Did not differentiate type B symptoms very clearly. Types D and E very clear.
C. P. 31/590	5/3—Type D [3 5]	5/3—Type D [3 5]	5/3—Type D [3 5]	5/1—Type D [3]	50	64.0	Uncongenial to Acc. 479 and Acc. 435; symptom clarity fair.
C. P. 31/591	5/1—Type D [3 5]	5/2—Type D [3 13 9]	5/1—Type D [3 13]	5/2—Type D [3 13 7]	50	46.0	Symptoms indeterminate.
A-148	5/2—Type D [3 7]	5/0	4/1—Not determined [9]	3/1—Not determined [9]	43	41.9	Infection slow, congenial only to strain A, symptoms indefinite. Strain A more chlorotic. Check also mosaic.
A-254	5/1—Not determined [9]	5/0	5/0	4/0	49	24.5	Low percentage infection except strain A, which caused more chlorosis on this variety than on any other.
A-276	5/1—Type D [9]	4/0	4/0	5/1—Type D [2]	48	22.9	Seemed congenial to Acc. 436; very low percentage with others.
A-284	5/0	5/0	5/0	5/0	49	20.4	Infected only by strains A and B and Acc. 436.
G-76	3/0	3/0	3/1—Type D [9 5]	2/0	28	14.3	Very low percentage, indefinite symptoms.
G-112	5/0	5/0	5/0	5/0	49	4.1	Lowest number infected, very slow.
G-284	5/1—Type D [3]	5/0	5/1—Not determined [9]	5/0	46	26.1	Seemed congenial to DW and strain A (slow).

K-89	5/0	5/1—Type D [9 14 5]	5/1—Type D [9 5]	5/1—Type D [13 5]	47	36.2	Rather slow, tendency to prefer Acc. 435 and Acc. 479 to D and DW.
K-129	5/3—Type D [3 9]	5/1—Type D [13 9]	5/2—Type D [3 2]	5/1—Type D [3 2]	49	42.9	Symptom patterns indefinite, owing to general recovery tendency.
M-37	5/0	5/3—Type D [3 9]	5/0	5/1—Type D [13]	50	44.0	Symptoms indefinite except for strains A and B and Acc. 436.
T-36	5/1—Type D [3]	5/2—Type D [13]	5/0	5/0	50	28.0	Low infection, except strain B.
T-97	4/1—Type D [13]	5/1—Type D [12 2]	5/0	5/0	48	35.4	Low infection, except strains A and B, and Acc. 436.
T-446	5/3—Type D [3 12 17]	5/3—Type D [3 9 13]	5/1—Type D [9 13]	5/2—Type D [13]	50	48.0	Very congenial to strains A and B. Strain A resembled mild D at first.
T-617	5/4—Type D [3 6 19]	5/2—Not determined	5/2—Type D [3]	5/3—Type D [3 12]	50	60.0	Very congenial to strains A and B. Check also mosaic.
Totals	139/53	138/47	139/50	137/33	1,375		
Percentage of mosaic.	38.1	34.1	36.0	24.1		44.8	

[1] First figure of fraction denotes number of plants inoculated (Aug. 19 and 20, 1936); second, number infected.
[2] Recovered, visible symptoms entirely disappeared.
[3] Necrotic areas characteristically present.
[4] One very slow.
[5] Very slow.
[6] Slow.
[7] One recovered.
[8] Stunted, quite severely.
[9] Recovery tendency.
[10] Actually kills back some shoots.
[11] Normal growth.
[12] Growing point blighted or killed; plant usually remains alive, but makes no further elongation.
[13] Sparse lesions, frequently 1 or 2 on some leaves.
[14] Symptoms not well defined.
[15] Severe.
[16] One very faint.
[17] Less severe than strain B.
[18] Atypical.
[19] Two recovered.

A and DW, although the symptoms were quite slow in appearing, particularly with the former. It is doubtful, however, whether any particular significance can be attached to this observation, as there have been no other indications that DW is any more closely related to strain A than others in the strain D group.

There seemed to be very definite indications that K–89 was more congenial to virus accessions 479 and 435 than to the strain D group. This was evidenced not only by the percentage of infection produced but also by the promptness with which the symptoms appeared. In this latter respect particularly K–89 seemed to favor strain B over strain A. In seedling K–129 the symptoms, with the exception of those produced by strains A and B and accession 436, were very indefinite at most stages, owing to a pronounced recovery tendency. The same holds true for M–37, which is further objectionable because of a natural chlorotic tendency in healthy plants that is confusing in making mosaic readings.

T–36 was outstanding in its preference for strain B. This is shown even more by the celerity with which the symptoms appeared than by the 100-percent infection as compared with a maximum of 40 percent for any other virus. Here, again, is emphasized the difference between strain B and accession 436. There seems to be little worthy of note in regard to either T–97 or T–446, although the early symptoms produced on T–446 by strain A were suggestive of a mild type D. The change to normal type A, however, was quite rapid. T–617 comes nearer to differentiating four clear symptom types as found in C. P. 31/294 than any of the seedlings in this latter group. The symptoms, however, were not always clear-cut and were frequently rather slow to appear. The one case of mosaic that appeared in the supposedly healthy checks, of course, minimizes the importance of these results.

EXPERIMENT 48

As a result of certain field observations and inoculation results, particularly those of experiments 23 (table 7) and 45 (table 10), it seemed highly probable that the virus collections that had been classed as strain D were readily divisible into two general groups. This fact had not been so readily discernible in the original inoculations to C. P. 28/60, but the use of new differential varieties, particularly C. P. 31/294, brought out quite vividly differences that, reexamination of old notes indicate, had existed on C. P. 28/60 but had been considered of minor importance. Those strains that were different from the original strain D were called "atypical strain D" or "northern Louisiana D" because of their origin.

Further collections from Mississippi, Alabama, and Georgia contained many similar viruses, a number of which were noted because of their lack of severity on C. P. 29/291, which in Louisiana is severely stunted by virus strains A, B, and D and characteristically shows severe sheath discoloration. The symptoms produced by these viruses on C. P. 28/60, 31/294, and other varieties were designated as type E in experiment 45.

The present test was designed to evaluate more clearly the differences previously noted and included a few new virus sources that had not been extensively tested before. Strain C also was included because it is indistinguishable from strain D on the varieties (C. P. 31/294, 31/590, and 31/586) used in this test, the results of which are presented in table 11. The inoculations were performed on November 24, 1936.

Accessions 13, 18, 243, and 410 produced symptoms on the three differential hosts in general agreement with the standard, or control, strain D. Nevertheless, all four accessions had produced different symptoms on Co. 281, as indicated under "History" (table 11, column 3).

Virus accession 166 produced type B symptoms on all three varieties, with characteristic intermediate stunting. It had previously been classed as strain D but was thought to belong to the so-called northern Louisiana group (E). The inoculum used here was obtained from the field, however, and it seems likely that the host variety, Louisiana Purple, may have recovered from it and been reinfected with strain B, as it had not been observed at regular intervals.

Accessions 202, 435, and 479 were readily differentiated from all the others in this experiment. On C. P. 31/294 they revealed a decided difference in infection percentage as well as in the type of symptoms produced. Although the symptoms (type E) produced by these three gave the impression of being much more severe, because of the larger lesions and striking red necrotic pattern, they seemed to have caused no growth retardation when compared with the healthy checks, whereas in the plants infected by the strain D group the mother shoots ceased elongation at once and as a rule eventually died.

No infection occurred on C. P. 31/590 with any of the three viruses in this test (accessions 202, 435, and 479), although unusually high percentages were obtained with the others. Some takes, however, were obtained in experiment 45 with accession 479, comparable, in fact, in percentage with those in the regular strain D group. The strange thing here is that better percentages were obtained in the present test with the D group and none at all with the other. Any contention that the viruses used were not comparable or virulent would seem to be refuted by the results obtained on C. P. 31/586, where all three produced 100 percent infection but with type E symptoms, as on C. P. 31/294. The same striking difference in growth was even more apparent on C. P. 31/586, which again seems to have proved itself the most congenial mosaic host variety of all the seedlings tested, particularly when a number of diverse viruses are involved.

The conclusion seems justified that a distinctly different strain of the virus (designated E) and represented in this test by accessions 202, 435, and 479, and more extensively in experiment 23 (table 7), has been differentiated and is readily separable from strain D. It appears also that this strain E is quite widely distributed in Louisiana, Mississippi, Alabama, and Georgia, and may even represent the only mosaic occurring in many isolated or outlying sections of these States.

TABLE 11.—*Differential mosaic symptoms identifying strain E among 8 virus collections, all of which previously had been classified into 2 or more groups of strain D (experiment 48)*

Virus strain or accession No.	Variety and locality	History	Variety inoculated (10 plants each) [1]					
			C. P. 31/294		C. P. 31/590		C. P. 31/586	
			Infected plants	Symptoms	Infected plants	Symptoms	Infected plants	Symptoms
			Number		*Number*		*Number*	
Controls:								
Strain C	Co. 281, Rosewood, La.	Severe chlorosis and necrosis on Co. 281 and other varieties.	9	Type D, necrosis, growing point,[2] stunting.	7	Type D, necrosis, recovery, growing point, stunting.	10	Type D, necrosis, growing point, stunting.
Strain D	do	Ordinary mosaic on Co. 281 and other varieties.	10	Type D, necrosis, growing point, stunting.	8	Type D, necrosis, growing point, stunting.	10	Do.
Virus collections:								
13 (DW)	P. O. J. 213, Washington, La.	Co. 281 shows high percentage germination recovery with this virus.	9	Type D, necrosis, stunting.	8	do	10	Do.
18 (GS)	Co. 281, Greenwood, La.	Causes mild necrosis in older leaves of Co. 281.	9	Type D, necrosis, growing point, stunting, 1 died.	6	Type D, necrosis, recovery, growing point, stunting, 1 died.	10	Do.
243	Co. 281, Poplarville, Miss.	Typical strain D	10	Type D, necrosis, growing point, stunting, 4 died.	9	Type D, necrosis, growing point, stunting.	9	Do.
410	Co. 281, Port Allen, La.	Causes purplish discoloration of sheaths of Co. 281.	10	Type D, necrosis, growing point, stunting.	10	do	10	Do.
166	Louisiana Purple, Cheneyville, La.	Classed originally as strain D, but not quite typical.	9	Type B, some stunting	8	Type B, necrosis, some stunting.	10	Type B, some stunting.
202	29-74, Lottie, La	do	2	Type E, necrosis, recovery	0		10	Type E, necrosis.
435	Co. 281, Meridian, Miss.	High percentage germination recovery and transitory symptoms on Co. 281.	2	Type E, necrosis, slow	0		10	Do.
479	C. P. 29/291, Cairo, Ga.	No stunting or sheath discoloration on C. P. 29/291.	6	Type E, necrosis, recovery slow.	0		10	Do.

[1] Inoculated Nov. 24, 1936.
[2] Growing point temporarily blighted or killed.

EXPERIMENT 49

In experiment 45 it was observed that on the variety C. P. 31/587 the single plant infected with virus strain D was much less stunted than the four plants infected with DW. To check this observation, the present inoculation test was begun on November 24, 1936. Twenty plants were inoculated with each virus and on December 28 all but one in each series showed typical symptoms of type D mosaic. There did not appear to be any significant difference in growth, however, so the height of each individual plant was measured on December 30. The measurements were taken at the last growth ring or the axis of the last fully expanded leaf, but as the plants were not measured at the time of inoculation there is no assurance that they were of equal height to start with. The measurements showed that the plants inoculated with strain D averaged 7.64 inches, while the DW plants averaged 6.62 inches, a difference of more than 1 inch. Another measurement on January 23, 1937, showed a very slight decrease in height for both sets of plants. This is in line with previous observations that elongation of infected plants of many of the C. P. seedlings ceases after they have become infected with one of the viruses of the strain D group.

The results do not seem to indicate any appreciable difference in the extent of stunting produced by the two viruses. It seems more likely, as suggested above, that there was an appreciable difference in the plants originally inoculated and that the difference in size of plants noted in experiment 45 was accidental.

EXPERIMENT 50

Strain B of the sugarcane mosaic virus has seemed to be very constant and stable as compared to strain D, which has seemed to have so many variants and has also shown a very marked tendency to break up or change when continuously cultured on certain C. P. varieties. Although no extensive comparison of different sources of strain B had been made, there was no indication of measurable differences until the testing of virus accession 436. This had been collected, as stated previously, on C. P. 29/291, at Meridian, Miss., and had caused no such stunting or sheath discoloration as is produced on this variety by strains A, B, C, and D. It seemed likely, therefore, that this collection would prove to belong to the group of viruses called, for convenience, northern Louisiana D. Upon being inoculated to a number of differential varieties, however, it produced the same reactions as strain B, except that there was some indication that it might not be quite so virulent on some varieties.

The fact that strain B and accession 436 were so similar except on C. P. 29/291 suggested that a survey of other collections of strain B might reveal other viruses that were different from the original. Three other collections of strain B, from widely separated localities in Louisiana, were selected for comparison with these two viruses. A second series was inoculated with strain B from the same original culture, which had been maintained in the Arlington Farm greenhouses for several years. These cultures, together with their sources and histories, are listed in table 12, which includes the results of the inoculations performed on November 25, 1936, to the two varieties C. P. 31/586 and 31/591.

TABLE 12.—*Comparative infection ratios and mean height of plants of 2 differential varieties inoculated with 6 collections classified as strain B from widely separated localities (experiment 50)*

Virus strain or accession No.	Source, variety, and place	History	Differential varieties inoculated [1]			
			C. P. 31/586		C. P. 31/591	
			Infection ratio	Height of infected plant [2]	Infection ratio	Height of infected plant [2]
				Inches		*Inches*
Strain B	Stock culture	Maintained in Houma, La., greenhouse.	20/20	10.67	17/14	20.77
Strain B (W)	do	Maintained in Washington, D. C., greenhouse; sent to Houma, and planted in field.	20/20	11.06	15/12	16.75
Strain B:						
Acc. 207	Co. 290, Bayou Gouki, La.	Ordinary mosaic selected at random in field.	20/14	17.39	14/5	19.75
Acc. 233	Co. 281, Livonia, La.	Ordinary mosaic selected at random from small field; new land.	20/19	12.38	18/15	17.53
Acc. 288	Co. 281, Reserve, La.	Ordinary mosaic selected at random in field.	20/18	17.63	13/7	21.11
Acc. 436	C. P. 29/291, Meridian, Miss.	Did not cause stunting or sheath discoloration; milder than strain B.	20/20	15.84	18/17	17.70

[1] First figure of the fraction denotes number of plants inoculated (Nov. 25, 1936); second, number infected.
[2] On Mar. 18, 1937.

The original supposition was that there might be a natural division of the viruses on the basis of percentage of infection, degree of stunting, or some other effect produced by the viruses, but the results were not conclusive. The only definite percentage difference was the significantly lower number of infections obtained with accession 207 on both varieties. There is a suggestion of a similar but less marked tendency with accession 288.

Based on growth or height of infected plants there seemed to be, on C. P. 31/586, a division of the six viruses into two groups, with both sources of strain B and accession 233 causing much greater growth retardation than the other three. The average height of infected plants of strain B and accession 233 ranged from 10.67 to 12.38 inches and for the other three viruses 15.84 to 17.39. The average difference was 5.38 inches.

These readings were made at the time the experiment was discontinued on March 18, 1937. Comparisons with earlier measurements (January 28) on two series of each group showed an increase in height in the regular strain B group of less than half that in the other groups. Indications were very strong, therefore, that, had it been possible to carry the experiment for a longer period, these differences would have been further accentuated. This growth effect was not apparent with the other variety, C. P. 31/591. The growth differences were not so great as shown in table 12 and there were no natural divisions into groups.

While it is common for two strains of the mosaic virus to produce identical results on one variety and widely different ones on another, it seems that, while the results above with C. P. 31/586 show a definite trend, it would be necessary to conduct further tests, probably with

additional varieties, including C. P. 29/291, to prove any significant differences between these collections of strain B.

Summary of Different Symptoms Produced on Certain Sugarcane Varieties by Seven Strains of the Mosaic Virus

Strain A produces ordinary mosaic symptoms (i. e., type Ad) on Co. 281 and Louisiana Purple and similar symptoms (i. e., a rather irregular mottling type of chlorosis) on C. P. 31/294 (and other seedling varieties). No necrosis is produced on any variety tested. This virus has a very high index of infectivity (figs. 10, 11, and 12). The type collection of this strain is accession 1.

Strain B produces ordinary mosaic symptoms on Co. 281 and Louisiana Purple (type Ad) and a rather general mottling, featured by severe chlorosis, and a varying extent of necrosis, and marked stunting of C. P. 31/294 (type B). It has a high index of infectivity (fig. 13, cf. figs. 11 and 12). The type collection of this strain is accession 2.

Strain C produces on all varieties infected (including Co. 281 and Louisiana Purple) approximately the same symptoms (type C). These first become evident as isolated (often single), elongated, almost white blotches or islands that coalesce or extend into long, yellowish-white streaks, often running the full length of the leaves (figs. 14 and 15). The streaks may follow the midrib and are usually accompanied by necrosis, often so severe as to produce temporary blighting or death of the growing point. Little or no elongation of infected plants of C. P. 31/294 (and other seedlings) takes place, and prodigious suckering is a common aftermath (type D). It has a very low index of infectivity. Many seedling varieties that are very susceptible to strains A and B appear to be immune to strain C (or nearly so) or show only light transitory infection with it (fig. 16, cf. figs. 11 and 12). The type collection of this strain is accession 22.

Strain D produces ordinary mosaic symptoms on Co. 281 and Louisiana Purple (type Ad) but produces symptoms identical with those described for strain C above (type D) on C. P. 31/294. It has a rather low index of infectivity, about half that of strains A and B but considerably above strain C (fig. 16, cf. figs. 11 and 12). The type collection of this strain is accession 21.

Strain Da differs from strain D only in the fact that a high percentage of germination recovery (type Ac) occurs in cuttings of Co. 281 infected with this strain, whereas none occurs with strain D (fig. 16, cf. figs. 11 and 12). The type collection of this substrain is accession 13 (DW).

Strain Db differs from strain D only in the fact that it produces on Co. 281 a marked necrosis (much milder and entirely different from that produced by strain C) on the older leaves (type Ae) of most infected plants. Younger leaves show no signs of this necrosis (fig. 16, cf. figs. 11 and 12). The type collection of this substrain is accession 18 (GS).

Strain Dc differs from strain D only in the fact that it produces, on Co. 281, scattered, purplish leaf-sheath lesions (type Af). It appears also to cause some growth retardation in this variety (fig. 16,

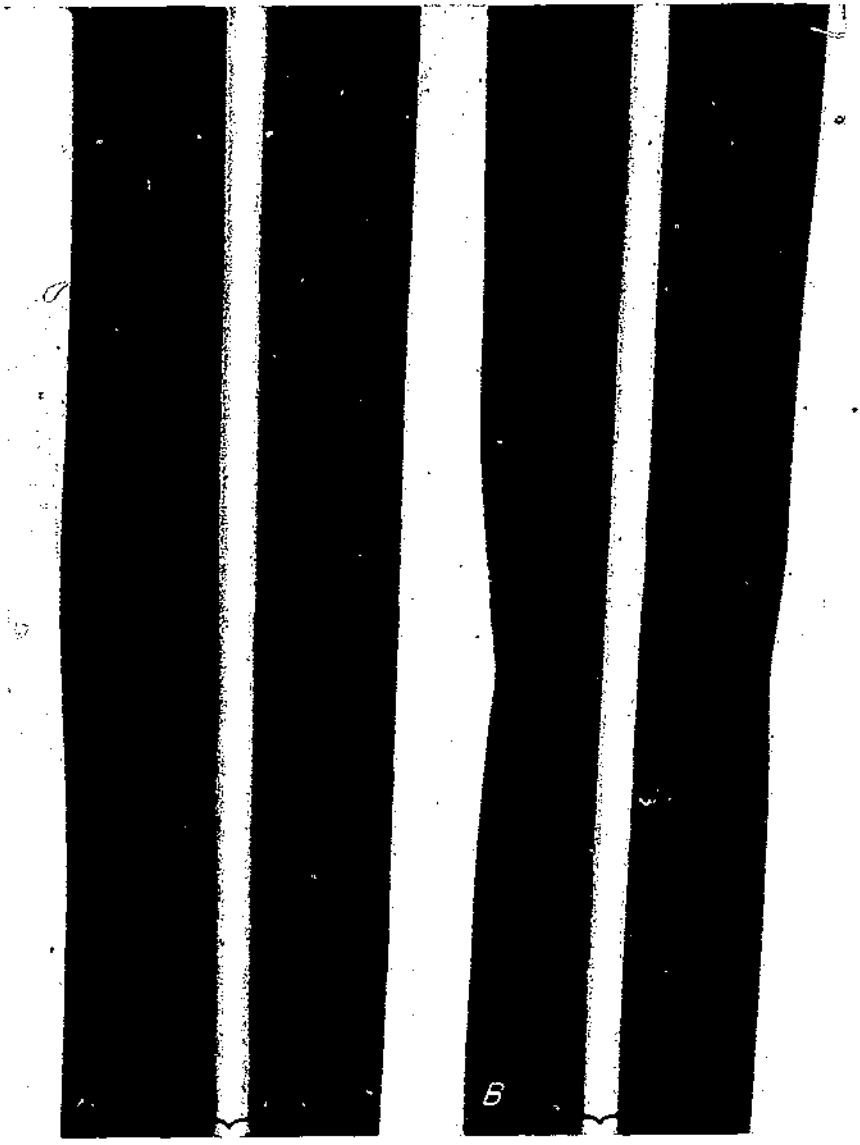

FIGURE 10.—*A*, Healthy leaf; *B*, typical symptoms on leaves of C. P. 28/60 caused by strain A of the sugarcane mosaic virus.

FIGURE 11.—Comparative effect on growth of C. P. 31 294 of eight collections of five strains and two substrains of the sugarcane mosaic virus: *A*, Healthy check; *B*, strain A; *C*, strain E (Acc. 435); *D*, strain E (Acc. 479); *E*, strain B; *F*, strain C; *G*, strain D; *H*, strain Da, and *I*, strain Db at 5 months. Note absence of elongation and occurrence of dead or dying growing point in *F* to *I*. Strain B causes comparable stunting but no killing of growing points. Early suckering is also characteristic of stunted shoots. Growth in plants infected with strains A and E comparable with healthy check.

cf. figs. 11 and 12). The type collection of this substrain is accession 410.

Strain E produces ordinary mosaic symptoms (type Ad) on Louisiana Purple and occasional plants of Co. 281, which, however, is highly resistant to infection by it, a high percentage of infections being transitory (type Ab). Germination recovery is also common in Co. 281. The symptoms produced on C. P. 31 294 resemble in the early stages those produced by strains C and D but differ markedly in that the lesions are fewer in number (often only one or two on a leaf), more clear-cut, and characteristically are accompanied by a pronounced reddening of adjacent tissues (type E). While the macroscopic symptoms are much more formidable than those produced by strain D on C. P. 31 294, there is apparently not the slightest retardation of growth, as compared with that of uninoculated checks, in plants infected with strain E. This strain has a very low index of infectivity, comparable to that of strain C (fig. 17, cf. figs. 11 and 12). The type collection of this strain is accession 202.

Strain F produces very mild symptoms on all varieties infected artificially, the pattern being regular and finely divided, sometimes diffuse and nearly masked (type Aa). It is easily distinguished from strain A, which produces an irregular but very positive pattern. Strain F is characteristically very slow to produce symptoms as com-

pared with strains A and B and has a lower index of infectivity. It was obtained from C. P. 807, long considered immune to mosaic, on which it produces very severe necrosis and stunting (fig. 18, cf. figs. 11 and 12). All efforts to inoculate C. P. 807 artificially have failed, not only with strain F but also with all others. The type collection of this strain is accession 430.

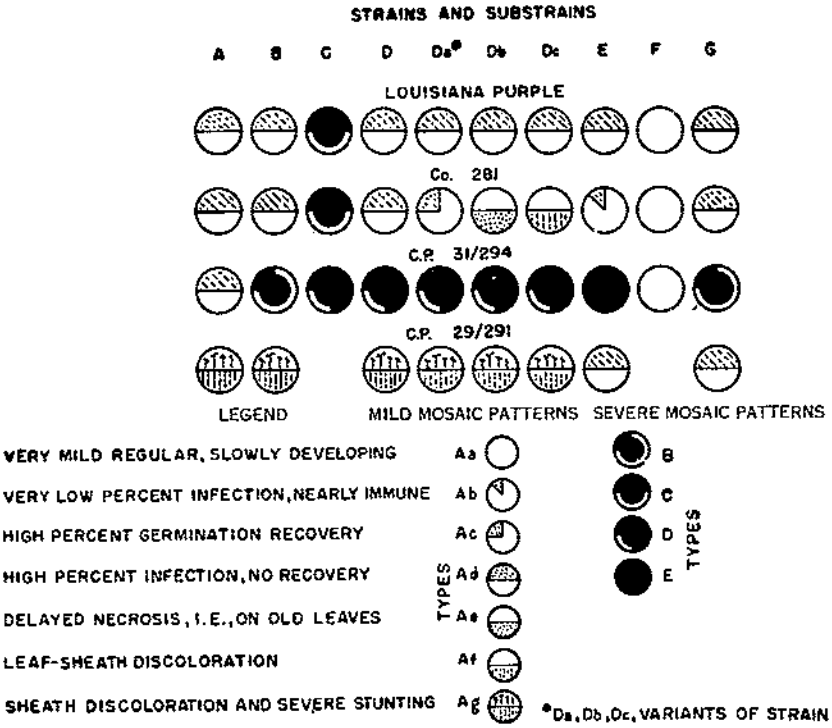

FIGURE 12.—Graphic representation of differential symptoms produced on 4 varieties of sugarcane by 10 strains and substrains of the sugarcane mosaic virus (*Saccharum virus* 1).

Strain G produces symptoms identical with those produced by strain B (types Ad and B), except on C. P. 29/291, from which it was collected. On this variety it produces neither sheath discoloration nor growth retardation (type Ad), both of which are produced by strain B (as well as other strains except F). Strain G has an index of infectivity appreciably lower than that of strain B but much higher than that of the strain D group (fig. 12). The type collection of this strain is accession 436.

The differentiation of the 10 strains and substrains of the sugarcane virus, largely on the basis of symptoms produced on a few varieties, is shown graphically in figure 12. Louisiana Purple is included to illustrate the fact that all but strains C and F are indistinguishable on it. In addition to these two, the substrains Da, Db, and Dc are differentiated on Co. 281. Strain E also becomes conspicuous here

Figure 13. A, H [illegible] of C. P. 31-291. *B*, [illegible] symptoms on leaves [illegible] B [illegible] sugarcane [illegible] virus.

[illegible]

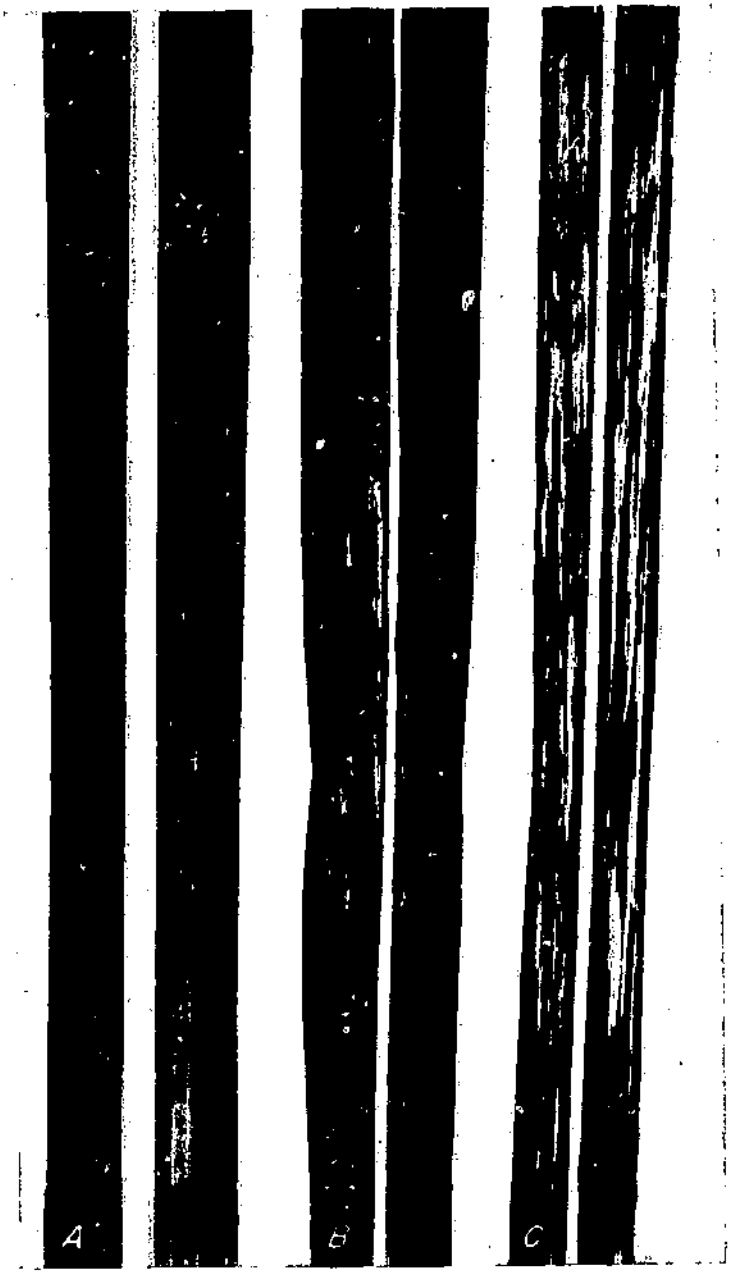

FIGURE 14.—*A*, Healthy leaf of Co. 281; *B* and *C*, typical symptoms produced by virus strain C on artificially infected leaves; white streaks coalesce in *C* and lesions are necrotic in *B*.

FIGURE 15.—Typical symptoms produced by virus strain C on young shoots of P. O. J. 36–M as a result of natural infection. Typical early symptoms are shown in the smaller shoot by the elongated, white, chlorotic areas. Many of these have coalesced in the older shoot, but the linear or striping tendency is still evident, as well as a number of necrotic areas.

FIGURE 16.—A, Healthy leaf of C. P. 31/294; *B*, typical symptoms on leaves caused by strain D (or C) of the sugarcane mosaic virus.

FIGURE 17. *A*, Healthy leaf of C. P. 31-294; *B*, typical symptoms on leaves caused by strain 1 of the sugarcane mosaic virus.

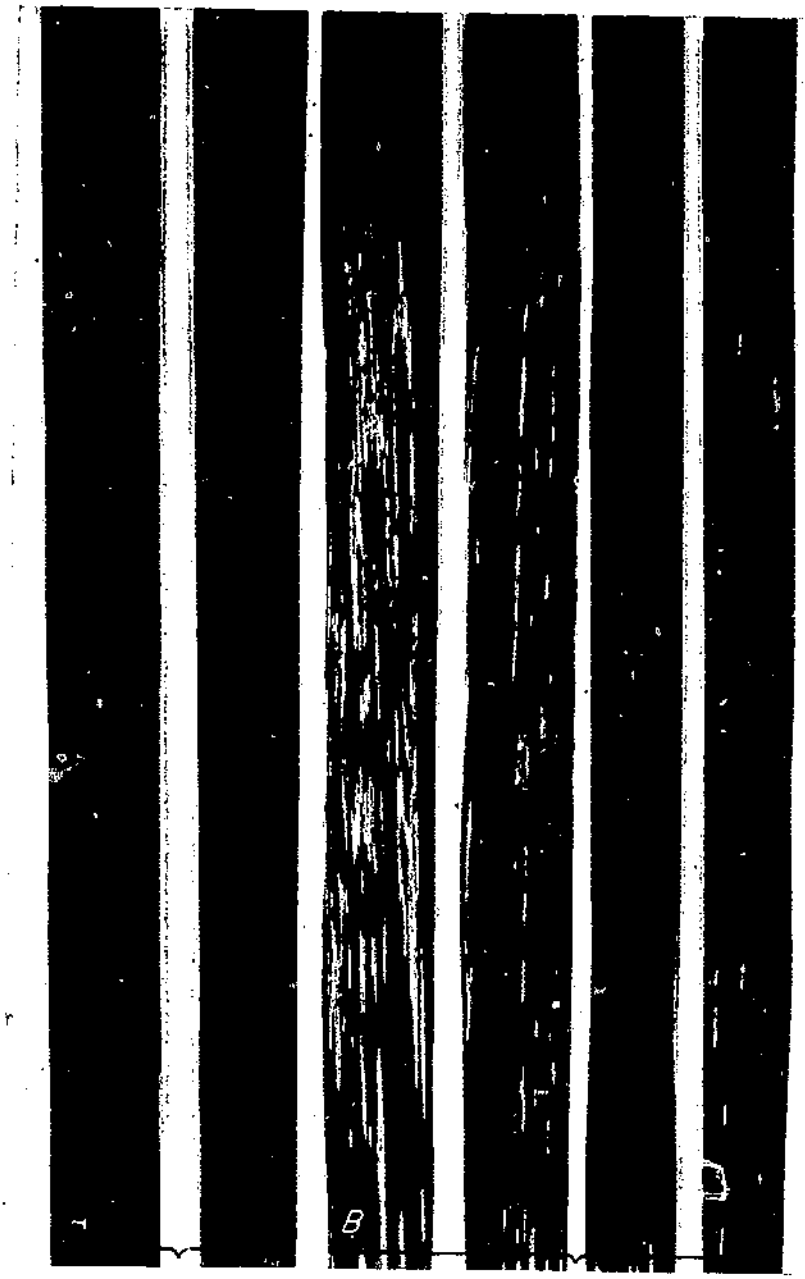

FIGURE 18.—*A*, Healthy leaf; *B*, typical symptoms caused by strain F of the sugarcane mosaic virus on C. P. 807 (natural infection), the variety from which it was obtained. This strain causes only mild symptoms on all varieties artificially infected by it.

because of its inability to produce any appreciable percentage of infection. Five distinct symptom patterns on C. P. 31/294 further emphasize the differences between strain E and the strain D group and also distinguishes strain A from strains B and G. Strains B and G have shown nothing except possible quantitative differences on these three varieties but are adequately differentiated on C. P. 29/291.

KEY FOR IDENTIFICATION OF STRAINS AND SUBSTRAINS OF THE SUGARCANE MOSAIC VIRUS DIFFERENTIATED IN THIS STUDY (COMPARE FIGURE 12)

	Strain designation
Necrotic areas accentuating mosaic mottling on leaves of Co. 281.	
A. Severe necrotic and chlorotic pattern at all stages	C
B. Mild necrotic pattern on older leaves only	Db
Necrosis absent; ordinary mottling pattern of light and normal green on leaves of Co. 281.	
A. Leaf sheaths of Co. 281 develop purplish discoloration	Dc
B. Leaf-sheath discoloration of Co. 281 absent.	
1. Necrotic blotches and stripes on leaves of C. P. 31/294.	
a. Growth of C. P. 31/294 markedly retarded.	
aa. Over-all severe pattern on all infected leaves of C. P. 31/294.	
aaa. Growth of C. P. 29/291 markedly retarded, with leaf sheaths developing a purplish discoloration	B
bbb. Growth of C. P. 29/291 not markedly retarded and discoloration of leaf sheaths absent	G
bb. Localized severe pattern, often along midrib on C. P. 31/294.	
aaa. High percentage of germination recovery by infected cuttings of Co. 281	Da
bbb. No germination recovery in Co. 281	D
b. Growth of C. P. 31/294 not noticeably retarded	E
2. Necrosis absent on C. P. 31/294.	
a. Irregular but distinctive mottling on all varieties; short incubation period and high index of infectivity	A
b. Regular but finely divided and very mild mottling on all varieties; long incubation period and lower index of infectivity	F

REGIONAL AND VARIETAL SURVEY

The differentiation and description of four strains of the sugarcane mosaic virus (*68*) in 1934 naturally raised the question of their possible distribution and relative abundance in Louisiana and other Southern States. Discovery of the extremely severe strain C in commercial fields of Co. 281 and proof of its transmission by aphids (*17*) emphasized its particular hazard to the industry should it become widespread. Surveys of commercial areas were undertaken immediately, therefore, to ascertain the distribution of strain C as accurately as possible, with the object of instituting eradication or control measures if necessary. Strain A, on the other hand, appeared to be so mild that its general occurrence might, conceivably, prove to be a benefit if it were as mild on commercial varieties as on a number of noncommercial seedlings and at the same time accomplished the exclusion of more severe strains. The extent of occurrence of strains B and D was also sought for possible correlation with differences in rate of spread, extent, and severity of mosaic infection of the commercial varieties in different sections of Louisiana. Such data could then be correlated with replicated yield comparisons, beginning with seed cane inoculated with individual strains of the virus.

A comprehensive survey of the sugarcane areas of Louisiana was begun in 1934, supplemented for the western part of the district by a further limited survey in 1939, to determine as nearly as possible the distribution of the known strains of the mosaic virus. Later on, a less extensive survey was conducted in limited areas of the southern parts of Mississippi, Alabama, and Georgia. Additional collections from Louisiana have been made from time to time to augment the original survey and to obtain further light on certain peculiarities of mosaic spread.

FIELD SAMPLING AND INOCULATION TECHNIQUE

An effort was made to sample the mosaic in each important district or locality in Louisiana, to note the variety of cane, crop involved (whether plant or stubble), mosaic percentage in particular fields, distance from other fields, and other pertinent information. One to four samples were taken from one to several representative fields in each locality. Where more than one was taken the samples were obtained from different parts of the field. No systematic survey of mosaic was attempted; for the most part the collections were made in connection with other work, such as field trips to study other phases of mosaic as well as other diseases.

Two methods of obtaining samples were employed. If the samples were to be used for inoculations within a few days' time, typically diseased tops were taken and the samples from each field were tightly wrapped together in heavy oiled paper. This made the package practically airtight and prevented any appreciable loss of moisture. The tops were cut off slightly above the growing point because the tender rapidly dividing tissue of the point is subject to rapid oxidation and deterioration. Such is not true, however, of the tightly rolled spindle and young leaves, which may be kept for 2 or 3 days in the field, even in hot weather if adequately protected from loss of moisture and still be in good condition for use as a source of inoculum. When placed in a refrigerator within a few hours after collection they can often be kept as long as a week. If inoculations were not to be made in a short time or the field trip was to last more than about 3 days, stem cuttings instead of tops of the infected stalks were collected. These were planted in an insectproof greenhouse and the young shoots used as sources of inoculum during the following 6 months. This latter procedure, while limited to the last two-thirds of the growing season after the cane has jointed, permits accumulation of collections until a convenient time for making the actual inoculations into differential hosts for strain identification.

Owing to limited greenhouse space at the United States Sugar Plant Field Station, at Houma, La., and to other demands on the available space, all inoculations in connection with the original survey were made in a large commercial greenhouse (fig. 19) near New Orleans. During the period of these tests it was made as nearly insectproof as possible by the use of large quantities of cheesecloth for screening ventilators and as a partition, and of roofing cement for sealing cracks. The doors were screened with 30-mesh copper-wire cloth. Periodic fumigation kept the house free of aphids, and systematic poisoning of ants prevented introduction and colonization of the plants with fresh

FIGURE 19.—Interior view of greenhouse in which inoculations were made with collections from the strain survey: Center bed, C. P. 28/60, differential host recently inoculated for determination of strains in survey collections; side beds, additional survey collections being maintained in the very susceptible Louisiana Purple variety.

aphids from the exterior. That these precautions were sufficient was established by the continued freedom from mosaic of an adequate number of healthy control plants.

This equipment proved to be ideal for the inoculations made early enough in the fall of 1931 to get adequate symptom expression before the onset of much cool weather. Later inoculations that fall were handicapped by the absence of heating facilities, and many plants were actually killed by one cold snap when the outside temperature dropped to 19° F. No infections at all were obtained from two sets of collections, which, in view of later developments, might have yielded much valuable information had they been saved. Many collections from this greenhouse were transferred to Houma for further study and observation after preliminary identification. All inoculations from later collections were made in the Houma greenhouse.

Since the New Orleans greenhouse was located about 75 miles from Houma, the juice for the earlier inoculations was expressed from the individual tops at Houma, placed in small vials, and frozen at 10° F. until a convenient time for use. Later on it was found more expedient to transport all equipment for grinding, sterilization, etc., to the greenhouse and extract the juice on the spot. This made it possible to use the expressed juice for inoculations at once. Both procedures gave satisfactory results. All juice samples were retained and inoculations repeated 1 or 2 days later. Methods of juice extraction and inoculation were the same as described above in connection with strain-differentiation studies.

Mostly Louisiana Purple and C. P. 28/60 were used for the inoculations. Louisiana Purple was used for three purposes: (1) To compare all collections on a very susceptible nondifferentiating host; (2) to avoid loss of collections through failure to infect the more resistant

C. P. 28/60 or other differential hosts; and (3) to preserve many samples that, because of a shortage of plants, could not be inoculated to C. P. 28/60 at once. C. P. 28/60 was the best differential variety known at the time of this survey and was, therefore, used to determine strains of the mosaic virus in these collections. All plants were sprouted at Houma in 4-inch pots, transferred while very young to the greenhouse by truck, and permitted to grow there until sufficiently large for inoculating.

Three plants of Louisiana Purple and five of C. P. 28/60 were inoculated with each virus sample. After the second inoculation the plants were set in the beds and grown there until final readings had been made. They were kept thrifty by fertilizer applications, cultivation, and regular watering. Readings were made 2 to 3 weeks after inoculation and as frequently thereafter as possible, usually at periods of 3 to 6 weeks, until three or four readings had been made.

As stated on page 87, the original plan was to conduct the survey for strains A, B, C, and D, as no others were recognized at the time. Subsequently, however, largely as a result of this survey, strains E, F, and G were differentiated and their occurrence noted. No attempt was made to separate the substrains of strain D (Da, Db, and Dc) in the survey, owing to the difficulty of identifying them on very young plants. No collections from the Houma station were included in the survey, as they were largely selected for specific purposes and could in no way be considered representative of mosaic in plantation fields.

REGIONAL DISTRIBUTION OF STRAINS

DEFINITION OF SECTIONS

For the purpose of this survey the State of Louisiana was divided into five sections on the basis of locality and, to a somewhat less extent, on local types of sugarcane farming (fig. 20).

The northern section includes most of the State outside of that used for sugarcane production for sugar and consists of small isolated plantings, frequently 10 to 15 miles apart, grown for sirup. This area lies mostly north and west of the sugar district. In 1934 the varieties of sugarcane grown were largely Louisiana Purple and some P. O. J. 213, which is a comparatively recent introduction.

The Red River section represents a sugar-producing area within the northern sirup section and is composed of two fairly extensive mill districts in southern Avoyelles and southeastern Rapides Parishes. This section is widely separated from the main sugar district.

The Mississippi River section includes all the sugar-producing parishes (largely on the west bank) that border that river.

The southeastern section comprises the parishes of Assumption, Lafourche, and Terrebonne and is continuous with the Mississippi River section near Donaldsonville, but by only a small strip of sugarcane land.

The southwestern section, including the parishes of Saint Mary, Iberia, Lafayette, Saint Martin, and parts of Vermilion and Saint Landry, is separated from the southeastern and Mississippi River sections by areas along the Atchafalaya River and by a tidal marsh area that varies in width from at least 10 to as much as 30 or 35

miles. Much of this swampy area is practically impenetrable and is largely overgrown with trees and vines, thus presenting a considerable barrier to natural mosaic spread to or from other sugarcane sections.

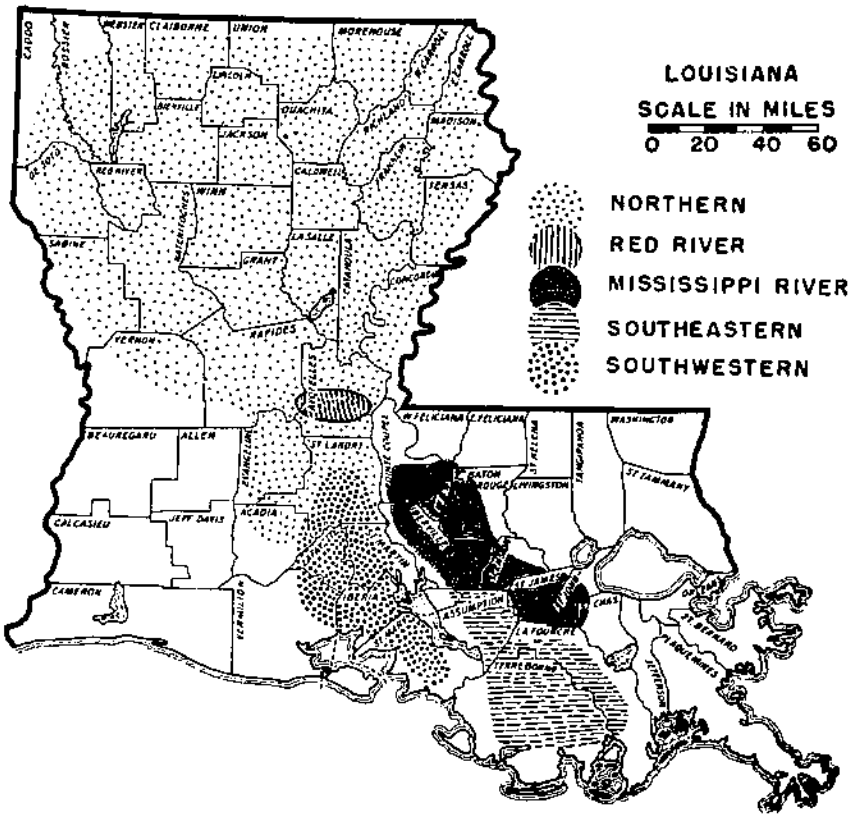

FIGURE 20.—Approximate boundaries of five natural sugarcane-producing districts in Louisiana from which mosaic samples were collected, and each of which proved to have different patterns of mosaic-strain occurrence.

OCCURRENCE OF STRAINS IN THE FIVE SECTIONS

The results of the mosaic virus-strain survey, considered from the regional distribution standpoint, are shown in table 13. Of a total of 278 collections tested, only 137, or 49.3 percent, were identified. On 115 collections (41.4 percent) no infection was obtained, and 26 others (9.4 percent) appeared to be mixtures of two or more strains and could not be definitely assigned to any particular one. Strain G has not as yet been definitely identified from Louisiana, and strain A was identified only from the seedling nurseries at the Houma station, which are not included in this survey.

A total of 51 collections was made in the isolated areas of the northern section, but unfortunately only 15 of them were identified. Of these the great majority proved to be strain E, with only 1 of strain B and 2 of strain D. No infections were obtained with the remaining 36.

TABLE 13.—*Sectional occurrence of strains of the sugarcane mosaic virus in Louisiana*

Section of State	Fields sampled	Mosaic collections								
		Identified as strain—					Total identified	Mixtures	Unidentified	Total tested
		B	C	D	E	F				
	Number	*Number*	*Number*	*Number*	*Number*	*Number*	*Number*	*Number*	*Number*	*Number*
Northern (isolated areas)	27	1	0	2	12	0	15	0	36	51
Red River	38	0	4	19	0	0	23	12	17	52
Mississippi River	48	20	0	20	0	0	40	14	23	77
Southeastern	32	15	2	11	0	1	29	0	20	49
Southwestern	23	6	1	23	0	0	30	0	19	49
Total	168	42	7	75	12	1	137	26	115	278
Total identified—percent		30.7	5.1	54.7	8.8	0.7				
Total tested—percent							49.3	9.3	41.4	

In the larger cane areas of the Red River section, almost totally within the extensive northern section, however, strain D was dominant, to the exclusion of strains B and E. It was identified in 19 collections, and the severe strain C, for which this was the type locality, was identified in 4 of the total of 52 tested. Of the rest, 12 were apparently mixtures of 2 or more strains and the remaining 17 produced no infection when inoculated to differential varieties.

The distribution of strains in the Mississippi River and southeastern sections was very similar, consisting of approximately equal parts of strains B and D. One difference, however, was noted in that 14 collections from the Mississippi River section were classed as mixtures, whereas none from the southeastern section were so classed. Strain C was identified twice in the southeastern section and this was the type section for strain F.

Strain D was dominant in the southwestern section, having been identified from 23 collections, as compared with 6 for strain B and 1 for strain C. The remaining 19 of the 49 collections produced no infection.

DISTRIBUTION OF STRAINS WITHIN LOCALITIES OR FIELDS

During the course of this survey many observations of more than ordinary significance were made upon the distribution of the different strains of the sugarcane mosaic virus within localized areas. In the northern section, for instance, where strain E predominated, it was also collected from the same fields from which strains B and D were identified. Each of these fields, incidentally, was closer to the other sections from which strains B and D were generally obtained than the other fields, which yielded only strain E. The indications are that, had successful identification of the strains present in collections from the more remote parts of this section been possible, strain E would have been found exclusively or nearly so.

The Red River section consists of two definite areas. The first is limited to Rosewood plantation, which yielded, in addition to 4 collections of strain C, including the type collection, 14 of strain D, all of which produced unmistakable, clear-cut symptoms, with no indications of any mixtures. On the other hand, the Bunkie area some 15 miles distant yielded only 5 cases of strain D, but 12 that appeared to be inseparable mixtures of 2 or more strains and could not be definitely identified. What the significance of this fact may be is problematical, but it certainly indicates the desirability of further study of the strain situation in the Bunkie area.

This condition of apparently mixed strains was not, however, necessarily confined to areas where strain D was predominant, as was shown in the upper part of the Mississippi River section, from which most of the 14 cases of mixtures were recorded. They were chiefly from 2 places, of which 1 yielded 9 collections of strain B and only 1 of strain D, and the other 2 and 1, respectively, of the same strains. A few mixtures were recorded from other parts of the same section, but none from either the southeastern or southwestern sections. With the exception of this difference there is a striking similarity in the strain distribution in the Mississippi River and southeastern

sections, indicating that they should be considered as a single unit in this discussion.

From the combined area, 35 collections were strain B and 31 strain D, indicating an almost equal total distribution for these 2 strains in the sugar-producing area east of the Atchafalaya River. Many individual localities or plantations within this region, however, yielded predominantly 1 strain or the other. This was particularly true of strain B, which, for example, was identified from 7 collections from as many fields of Georgia plantation in Lafourche Parish and from 9 of 10 collections from Alma plantation in Pointe Coupee Parish. Alma plantation yielded 1 collection of strain D and a number of the mixtures mentioned above. While there were no large plantations that yielded such large numbers of strain D, in only 1 or 2 cases did a single field yield more than 1 strain. These 2 combined sections, therefore, are the only ones in which there was not 1 strain apparently dominant.

Of the six collections of strain B from the southwestern section, five were from Albania plantation. All other places in this section, with the exception of one field, which yielded one collection each of strains B and C, yielded only strain D.

VARIETAL ASSOCIATION OF STRAINS

Approximately half the 137 identified collections listed in table 13 and segregated by variety in table 14 were from the two varieties Co. 281 and P. O. J. 213. These were emphasized intentionally because of their prominence and because their mosaic was known to be largely or wholly due to secondary spread during the period since 1930. In that year mosaic was practically nonexistent in both varieties wherever grown, while the other P. O. J. varieties and Louisiana Purple were known to have carried a fairly heavy infection at all times since about 1924 and 1920, respectively. Co. 290, still not very generally grown, had but small percentages of mosaic as late as 1934.

The 42 collections of strain B and 75 of strain D make a total of 117 collections (or 85.4 percent) of the 137 identified. Table 14 indicates that all varieties except C. P. 807 harbored both these strains, of which strain D was usually more prevalent. Strains C and F were collected too infrequently to warrant any generalizations here, except possibly that strain C seemed to occur chiefly on Co. 281, while the single collection of strain F was from C. P. 807. Strain E was collected mainly from P. O. J. 213 and Louisiana Purple, because these were the only varieties occurring in the isolated plantings from which this strain was obtained.

SIGNIFICANCE OF UNEQUAL STRAIN DISTRIBUTION

Rather peculiar but quite definite geographic and varietal distributions have been shown for each of the three most common strains (B, D, and E) of the sugarcane mosaic virus in Louisiana. The preliminary survey suggests that, regardless of variety of cane, strains B and D were about equally distributed in the large area east of the Atchafalaya River and adjacent to the Mississippi River; strain D predominated in the sugar-producing areas west of the Atchafalaya almost to the exclusion of strain B; and strain E occurred almost to the

TABLE 14.—*Varietal occurrence of strains of the sugarcane mosaic virus in Louisiana*

Variety showing mosaic in the field	Fields sampled	Mosaic collections								
		Identified as strain—					Total identified	Mixtures	Unidentified	Total tested
		B	C	D	E	F				
	Number	*Number*	*Number*	*Number*	*Number*	*Number*	*Number*	*Number*	*Number*	*Number*
Co. 281	37	11	4	24	0	0	39	10	17	66
Co. 290	12	7	0	4	0	0	11	2	5	18
C. P. 807	1	0	0	0	0	1	1	0	0	1
Louisiana Purple	16	1	0	3	8	0	12	0	19	31
P. O. J. 36	9	1	1	5	0	0	7	1	4	12
P. O. J. 36 M	7	1	1	3	0	0	5	0	3	8
P. O. J. 213	26	8	0	17	2	0	27	5	17	49
P. O. J. 234	19	1	1	8	0	0	10	7	11	28
Others	41	12	0	11	2	0	25	1	39	65
Total	168	42	7	75	12	1	137	26	115	278
Total identified—percent		30.7	5.1	54.7	8.8	0.7				
Total tested—percent							49.3	9.3	41.4	

exclusion of both B and D in the small isolated sirup-cane patches somewhat removed from the main sugar-producing areas. The general occurrence and persistence of strain E in this northern section, where the rate of secondary spread to such varieties as Co. 281, Co. 290, and the P. O. J. canes seemed to be very low, are possibly due to its original infection of and persistence on Louisiana Purple, which is susceptible to all strains. Original introductions of P. O. J. 213 into these areas was probably with mosaic-free seed. The limited occurrence of mosaic on it as compared with that on Louisiana Purple may have been due to the local vector situation, or, more likely, to its greater resistance to infection by this strain.

Single stools of Co. 281 showing strain C were collected in 3 locations, 2 in the southeastern and 1 in the southwestern section in addition to Rosewood plantation in the Red River section, where it was originally found. These 3 stools were the result of an intensive search for this strain, which produces such obvious and severe symptoms on the commercial varieties, rather than as a result of random sampling used for the other strains. Except at Rosewood plantation, therefore, its occurrence was exceedingly slight. Although well over 100 stools of 4 varieties were found infected with it at Rosewood, its abundance was readily traceable to increase of seed cane from a few original infected stools. Since all original distributions of Co. 281 were made with mosaic-free seed, and the 4 strain C localities are widely separated, it seems likely that this strain was not a recent introduction, but was probably coming from some local source of infection. It might conceivably be a recurring mutant of strain D.

Strain F has been identified from C. P. 807 only on Raceland plantation, in the southeastern section, the only place where any appreciable quantity of mosaic has been observed on this variety. A very few mosaic stools were observed elsewhere, but the strain was not determined. The original assumption that all mosaic observed on it was caused by strain F does not seem entirely justified, because some more recent determinations of the virus in C. P. 807 plants, the original source of which was Raceland, have yielded what appears to be strain B.

From present knowledge of the varietal associations of strains B, D, and E, and of the distribution of new varieties of sugarcane in Louisiana during the past 20 years, it seems logical to conclude that those isolated fields giving only strain E represented surviving remnants of the original mosaic epidemic in the State. This strain gives high percentages of infection on such noble varieties as Louisiana Purple and, under favorable conditions for secondary spread such as obtained in that epidemic (7), would rapidly infect a high percentage of the available healthy cane. The western and northern (strain E) areas were found free from mosaic by Brandes (7) in 1919, and became infected subsequently, presumably as a result of the wave of mosaic that spread westward at that time from the Mississippi River but had not as yet crossed the Atchafalaya River, except in isolated plantings readily traceable to imported cuttings.

Mosaic did, however, cross the Atchafalaya River shortly afterward and had become generally distributed before any extensive plantings of the P. O. J. varieties were made, i. e., about 1927. It may be assumed, therefore, that if strain E of the mosaic virus was

the same as it is today, it was prevalent in the first general mosaic invasion of Louisiana sugarcane fields. The possibility that other strains may have been active during this period (1919–27) seems highly unlikely, although strain D may have been gaining some momentum during the latter part of the period, because its general distribution over the entire area used for cane production for sugar seems to coincide exactly with the distribution and general planting of the P. O. J. varieties. Although mosaic-free cuttings of these varieties were brought from Washington to Louisiana in 1922 and 1923, they soon became 100 percent mosaic-diseased (*56*) and were so distributed throughout the sugar district.

Even under the most ideal conditions for secondary spread, it is doubtful whether any of the P. O. J. varieties grown in Louisiana, such as P. O. J. 36, P. O. J. 36–M, P. O. J. 213, and P. O. J. 234, would have become 100 percent diseased with only strain E as a source of infection, because of its very low index of infectivity. A general and fairly complete recovery took place in P. O. J. 213 between 1925 and 1930, so that when it was distributed to the sirup sections of the State it probably was largely mosaic-free; hence, its subsequent low extent of infection with strain E in these areas.

There had been little or no secondary spread for 4 to 6 years previously. Strain B apparently began to be a factor in a new wave of secondary spread which got under way in 1931 and gained momentum until 1936 (see fig. 1, showing incidence of secondary infection in healthy plantings). In that year the heaviest secondary spread in Louisiana in more than a decade was experienced. The ascendancy of strain B was not a matter of distribution on newly released varieties, as must have occurred with strain D, because the varieties released in 1930 and 1931 (C. P. 807, Co. 281, and Co. 290) were practically 100 percent healthy. Both Co. 281 and Co. 290, as well as all the P. O. J. varieties, of which there was considerable healthy cane at that time, are, however, very susceptible to strain B, which has a much higher (nearly double) index of infectivity than strain D (see table 10). This latter fact probably explains the later heavy spread, because both Co. 281 and Co. 290 are more resistant to infection than any of the P. O. J. varieties. The practical absence of strain B in the area west of the Atchafalaya River, noted in 1934, appeared to have continued, for a limited survey in 1939 revealed strain D in practically every sample collected.

DISTRIBUTION IN OTHER STATES

The virus strain survey conducted in Alabama, Georgia, and Mississippi resulted in the identification of 64 of the 77 collections of mosaic from sugarcane tested from those States (table 15). The outstanding development here is the apparent abundance of strain E as compared with strains B and D, not only on Louisiana Purple but also on all varieties with the exception of P. O. J. 213, which accounted for 14 of 17 collections of strain D and 3 of 6 of strain B. Practically all collections of these two strains, except a few made at the United States Sugar Plant Field Laboratory at Cairo, Ga., were from P. O. J. 213.

The procession of virus strains in these three States seems to have

TABLE 15.—*Relative abundance of four strains of the sugarcane mosaic virus in Mississippi, Alabama, and Georgia*

State and variety showing mosaic in the field	Fields sampled	Mosaic collections						
		Identified as strain—				Total identified	Unidentified	Total tested
		B	D	E	G			
Mississippi:	*Number*	*Number*	*Number*	*Number*	*Number*	*Number*	*Number*	*Number*
Co. 281	1	0	0	2	0	2	0	2
C. P. 29/291	2	0	0	2	2	4	1	5
Louisiana Purple	4	0	1	3	0	4	0	4
Otaheite	2	0	0	2	0	2	0	2
P. O. J. 36–M	2	0	0	3	0	3	0	3
P. O. J. 213	6	3	4	0	0	7	1	8
Ribbon	1	0	0	1	0	1	0	1
Total	18	3	5	13	2	23	2	25
Total identified—percent		13. 1	21. 7	56. 5	8. 7			
Alabama:								
Louisiana Purple	7	0	0	7	0	7	1	8
P. O. J. 213	5	10	0	8	0	18	2	20
Total	12	10	0	15	0	25	3	28
Total identified—percent		40. 0	0	60. 0	0			
Georgia:								
C. P. 29/291	2	0	0	2	0	2	1	3
Louisiana Purple	2	0	0	2	0	2	1	3
P. O. J. 234	1	0	0	2	0	2	0	2
Miscellaneous seedlings	2	3	2	5	0	10	2	12
Others	2	0	0	0	0	0	4	4
Total	9	3	2	11	0	16	8	24
Total identified—percent		18. 7	12. 5	68. 7				
Grand total	39	16	7	39	2	64	13	77
Total identified—percent		25. 0	10. 9	61. 0	3. 1			
Total tested—do						83. 1	16. 9	

been analogous to that discussed in the preceding pages for Louisiana, except that here the process at the time of the survey was still in its early stages. This was presumably due to the sugarcane being grown in small isolated patches unfavorable for secondary spread and to less frequent changes of varieties. Louisiana Purple was still very common and for the most part still harbored only strain E. P. O. J. 213, introduced much later than in Louisiana and more resistant to strain E, was furnishing a reservoir of infection for the other two strains which, because of the congeniality of this host, might conceivably predominate with increase in its acreage.

In 1938 Matz (*52*) reported some preliminary strain identifications of mosaic plants intercepted in the Washington quarantine greenhouses. Collections from India, Puerto Rico, and Spain were similar to or identical with the authors' strain B, while four of five collections from Hawaii and other Pacific islands proved less infective and less stable on the standard differential hosts than any of the known American strains.

RECOVERY FROM MOSAIC

Prior to the discovery of strains of the sugarcane mosaic virus, data had been accumulated on the apparent recovery of certain varieties. Some of the P. O. J. varieties had been 100 percent mosaic-infected in 1924 and 1925. During the years 1926 to 1930, a period of apparently low secondary spread in Louisiana, there occurred a widely noted and unexpected disappearance of mosaic symptoms, which in the case of P. O. J. 213 was practically complete throughout the State. Such marked ability to throw off the disease prompted the study of recovery as a factor in field control as well as in breeding for mosaic resistance.

Evidence is here presented showing (1) actual occurrence of permanent recovery; (2) incidence of recovery among the more important commercial varieties; (3) variability of disease transmission by cuttings; and (4) the manner in which a natural diminution and even complete elimination of mosaic may be brought about over large areas by what may be conveniently called the recovery process.

Rather wide variations have been reported from several countries on the extent of recovery of sugarcane from mosaic. These variations may have been due to the particular varieties involved as well as to the probable existence of different strains of the virus. Local environmental factors, including type and prevalence of vectors, undoubtedly also exerted a great influence on the degree of recovery observed. Brandes (*9*), in 1920, reported numerous cases of apparent recovery in corn and also in crabgrass (*Digitaria sanguinalis*) and yellow bristlegrass (*Setaria lutescens*), all three hosts being infected with the virus of sugarcane mosaic. He also observed that occasional mosaic-diseased stools of both sugarcane and sorghum produced tillers with no signs of mosaic.

Kunkel (*48*), reporting in 1924 on his studies of sugarcane mosaic in Hawaii, said:

> It was observed that diseased stools of certain varieties frequently recover. Careful studies show that this may come about in either of two different ways.
>
> The diseased shoots of a stool may at times begin to produce healthy leaves.

Later, the old diseased leaves die and fall off. Such stools may grow to maturity without showing any further signs of disease on the leaves. They show no evidence of having been diseased unless the joints from which the diseased leaves grew have markings characteristic of mosaic. These stools become healthy through the recovery of the terminal buds of their diseased shoots. But the disease may also be overcome in quite a different way. Although none of the diseased shoots actually recover, the new shoots produced may be healthy. The diseased shoots remain small and are overgrown by the healthy ones. After a time, they die and the stool may then remain healthy to maturity. In other instances, a stool may be diseased or partly diseased in the plant crop or in one of the ratoon crops, but after this crop is harvested, it may produce only healthy shoots. Such stools may remain healthy through the next and subsequent crop periods. This is the manner in which many stools were observed to recover in experiments to test the effect of mosaic on yield. . . .

Kunkel's observations resulted in detecting a number of cases of recovery. More detailed observations on Lahaina revealed seven cases of the first type described above.

East (*38*) noted in Cuba that an individual sugarcane plant may recover and be reinfected as many as three times. He suggested that recovery might be the result either of actual death of the infective agent or merely a reduction in virulence, permitting disappearance of symptoms that might reappear under favorable conditions. He favored the latter hypothesis as an explanation of what he considered the first critical evidence of the establishment of an acquired immunity to a definite infective agent in a flowering plant.

Rands and Summers (*56*), in a preliminary paper in 1932, reported observations of numerous instances of apparent recovery from mosaic in several varieties of sugarcane in Louisiana. Field observations during the summer of 1923 on Louisiana Striped cane showed a reduction in mosaic from 63 percent on June 2 to 14 percent on July 21. This was believed to be actual recovery from mosaic rather than a temporary suppression of symptoms, even though a wave of secondary spread increased the mosaic to 86 percent later in the season.

Healthy plants of 3 other noble varieties—Louisiana Purple, L–511, and D–95—were obtained from cuttings of similarly "foliage-recovered" plants. The variety D–74 showed no such tendencies. The newly introduced P. O. J. varieties were 100 percent infected in Louisiana in 1924. By 1930, there was almost no mosaic in P. O. J. 213 and it was greatly reduced in the others. In that year actual observations of individual plants showed 20.2 percent foliage recovery in P. O. J. 36–M and 0.8 percent in P. O. J. 234. Incomplete transmission by diseased cuttings was marked in P. O. J. 36–M and P. O. J. 234, but much less so in the other varieties mentioned, and incomplete transmission by diseased stubbles was much less common than by cuttings.

Importance of Recovery

Under certain conditions the ability of a sugarcane variety to recover from mosaic might be quite significant in reducing losses from the disease. With minimum secondary spread, such as may exist for a period of years in Louisiana, mosaic in certain varieties may be almost completely eliminated by this process. It has been necessary to include in commercial plantings varieties that are susceptible to mosaic because of other desirable qualities that fit them into the local variety program, and any appreciable amount of natural recovery

would be a valuable asset in any efforts at control. When a variety shows a tendency to recover, roguing, as a control measure, is naturally aided to the extent that recovery occurs. Any appreciable amount of recovery in a variety naturally reduces the source of secondary infection by reducing the inoculum of two sources—the cane that recovered and the cane that would have been infected by secondary spread had the supply of the infective agent not been removed.

Probably one of the most important practical aspects of recovery is its possible utility in cane breeding, where it is hoped that it will prove to be a heritable character that may be transmitted to desirable seedlings not highly resistant or immune. It is entirely possible that this character may be greatly accentuated in an occasional seedling and be one of the important factors for resistance.

Recovery Among Commercial Varieties in Louisiana

Observations over the period 1930–32 in Louisiana revealed a strong tendency on the part of a number of commercial varieties gradually to reduce the incidence of mosaic in the field. This was evidently brought about in two 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, common to all grasses, presently showed no further signs of the disease.

Recovery manifested at time of germination

The sprouting of considerable numbers of healthy shoots from supposedly diseased cuttings and ratoons was noted among the newer introductions, P. O. J. 36–M, P. O. J. 213, and P. O. J. 234, as well as the long-cultivated L. 511 and Louisiana Purple, both later discarded as commercial canes.

In order definitely to compare the varieties in respect to this important characteristic, systematic observations were made over a period of 3 years (1930–32) of thousands of plants grown under representative field conditions. Usually the varieties were distributed in replicated single-row plots of 1/412 acre and alternated with plots of healthy cane of the same variety to indicate the prevalence of secondary mosaic infections (i. e., spread by aphids). In conformity with field practice, all experimental plantings were made during the fall preceding the year of observations. Mosaic readings were made as early in the following spring as it was possible to detect leaf symptoms.

The first experiment, begun in the fall of 1929, was about one-third of a mile from Bayou Black in a rear black-land field of the United States Sugar Plant Field Station, near Houma, La. The seed cane had been selected from stools inspected only at harvesttime for the presence or absence of mosaic. In May 1930 evidence of recovery, shown by the sprouting of healthy shoots from the diseased seed, was noted. The occurrence of mosaic sprouts in the healthy plots was noted also. Further readings were made in August and December. Then in the spring of 1931, when the stubble from the previous crop

sprouted, the observations were repeated on the ratoons and were continued in 1932 on the second ratoons.

In the meantime, new plant-cane tests that included additional varieties were put down each year and in most cases were followed through at least to the first ratoon crop. These tests were of necessity located on light soil near the bayou, where, unfortunately, as subsequent observations proved, heavy secondary mosaic spread by aphids complicated the interpretation of findings on the ratoons. All records for this light soil area were on plants 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, and thus were called pedigreed in these tests. The healthy seed of Louisiana Purple was selected in 1930 and grown the next year before being used in the test. The relative extent of recovery indicated by the early spring observations for each of the 3 years is presented in table 16, and later seasonal readings of mosaic are shown in figure 21.

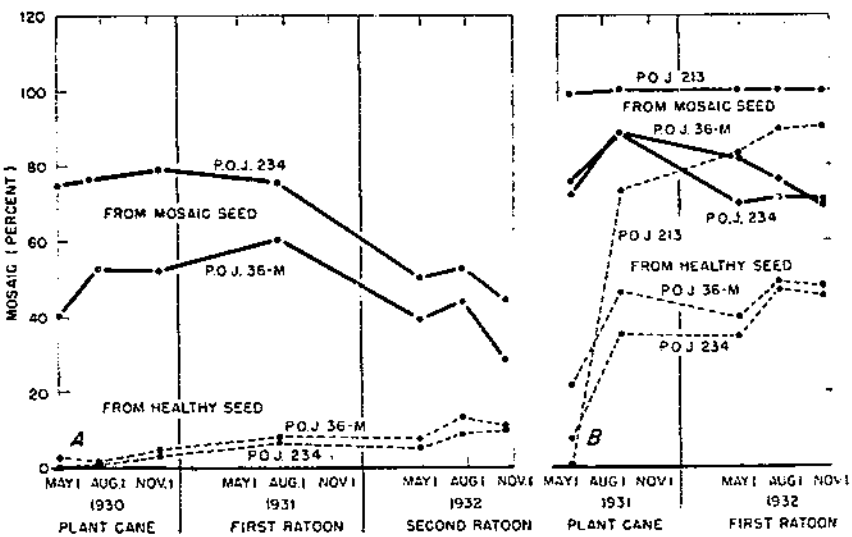

FIGURE 21.—Comparative mosaic incidence in plantings of healthy and mosaic seed of three varieties of sugarcane on areas subject (*A*) to light and (*B*) to heavy secondary infection.

Four plantings of pedigreed mosaic seed of P. O. J. 36 M for a period of years produced 28 to 59 percent healthy shoots. That more mosaic appeared in the first ratoon than in the plant-cane crop was obviously the result of greater secondary spread during the last two growing seasons. In the single case where second ratoons were observed, however, a decided reduction in mosaic percentage over the previous crop was noted. A very slight reduction in the healthy plot also occurred, which probably meant that there was much less secondary spread in that particular field.

The behavior of P. O. J. 234 was similar to that of P. O. J. 36–M, except that a smaller percentage of healthy plants was produced.

TABLE 16.—*Comparative germination recovery from mosaic shown by primary shoots in plant and successive ratoon crops of several varieties grown from pedigreed seed cane planted in localities subject to light and heavy secondary spread*

BLACK-LAND TEST (SLIGHT SECONDARY SPREAD)

Variety	Year	Crop	Young shoots from—			
			Mosaic seed		Healthy seed (controls)	
			Total	Healthy (recovered)	Total	Mosaic (secondary spread)
			Number	*Percent*	*Number*	*Percent*
P. O. J. 36–M	1930	Plant cane	210	59. 4	207	2. 3
	1931	First ratoon [1]	1, 379	39. 0	1, 341	8. 1
	1932	Second ratoon	1, 044	60. 5	1, 250	7. 9
P. O. J. 36–M [2]	1930	Plant cane	196	50. 9	202	2. 7
	1931	First ratoon [1]	1, 756	40. 2	1, 947	15. 6
P. O. J. 234	1930	Plant cane	242	24. 8	234	0
	1931	First ratoon [1]	1, 260	24. 1	1, 432	6. 1
	1932	Second ratoon	1, 062	49. 5	1, 709	4. 9

LIGHT-LAND TEST NEAR BAYOU (HEAVY SECONDARY SPREAD)

Co. 281	1932	Plant cane	1, 004	0. 9	1, 127	1. 7
P. O. J. 36–M	1931	do	357	28. 3	476	14. 7
	1932	First ratoon	1, 059	18. 6	1, 351	37. 6
P. O. J. 36–M	1932	Plant cane	442	59. 5	490	. 4
P. O. J. 213	1931	do	140	. 7	100	1. 0
	1932	First ratoon	371	0	427	83. 1
P. O. J. 213	1932	Plant cane	297	. 3	459	. 2
P. O. J. 234	1931	do	232	24. 2	282	7. 4
	1932	First ratoon	970	30. 3	1, 363	33. 8
Louisiana Purple	1932	Plant cane	47	4. 3	63	12. 7

[1] Mosaic readings delayed until July; all others in April.
[2] Nonpedigreed seed.

Two plantings in successive years gave almost identical results, slightly less than 25 percent of healthy shoots. There was little change in the first ratoon crop, but the percentage of healthy plants was doubled in the one second ratoon. The three remaining varieties showed practically no recovery at the time of germination. Louisiana Purple, based on the smallest population observed, produced less than 5 percent recovery, which was much higher than Co. 281 and P. O. J. 213.

The data in table 16 from pedigreed healthy seed show that in only one case (P. O. J. 234) were all the germinating sprouts from this supposedly healthy seed free from mosaic at the first observation. In the remaining varieties, the percentage varied from 0.2 to 14.7 per-

cent. Two possible explanations might be offered: (1) The plants used for seed might have become infected the previous fall but too late for symptoms to be manifested before harvesting; and (2) natural spread might have occurred in spring before the cane was large enough for observing. The first possibility seems to be refuted by tests in an insectproof greenhouse, later reported. This leaves secondary spread as the most plausible explanation, although it is still difficult to account for as high as 14.7 percent mosaic in the 1931 plots of healthy P. O. J. 36–M (fig. 21). These plots were located closer to the bayou bank, however, where wild grass hosts bearing mosaic and its vector (*Aphis maidis*) were plentiful.

INFLUENCE OF DORMANCY OR UNFAVORABLE WINTER CONDITIONS ON EXTENT OF RECOVERY

The fact that so much recovery was manifested during germination in the field (table 16) led to some conjecture as to whether it might have been caused by environmental factors. One question concerned the possible influence of the adversities encountered during the winter months in Louisiana, when the cuttings are subjected to periods of warm weather, which tend to initiate growth, followed at irregular intervals by combinations of wet and cold (often freezing) weather, which stop all growth and freeze back everything above the surface at least once every winter.

In order to determine the effect of such factors, a planting of four varieties—Co. 281, P. O. J. 36–M, P. O. J. 213, and P. O. J. 234—was made on September 4, 1931, with mosaic seed selected in the field just prior to planting. The following fall and winter were unusually mild, and four mosaic readings were made before March 9, 1932, on which date a killing freeze destroyed all cane, including the terminal buds, above the surface of the ground. This was the only killing frost during the winter, and the cane came up again very quickly, representing in fact a ratoon growth, since the terminal buds had been killed. Further readings were made in May and September.

The results (table 17) show that no recovery occurred in Co. 281. At the first reading, less than 6 weeks after planting, 31.5 percent of the P. O. J. 36–M was healthy. The readings for the next 2 months were 50.4 and 45.0 percent healthy, with practically no change in population. The percentage of healthy cane jumped to 69.4 on February 10, and approximately this percentage was found at both readings taken on the new shoots after the freeze. P. O. J. 213 showed no signs of recovery at the first reading, on October 13, 1931; a few cases became apparent by November 16, but none were evident on February 10, 1932. On May 11, 2 months after the frost, 5.9 percent healthy cane was counted and this was increased to 7.3 percent by September 9, presumably because of a lesser natural mortality from crowding among the healthy stalks.

The cuttings of P. O. J. 234 produced 11.3 percent of healthy plants, which figure remained almost constant until the freeze. An additional 10 percent recovered upon germination of the young stubble after the freeze, giving a percentage of 21.2 on May 11. The percentage was practically the same in September.

The fact that there was about as much recovery in P. O. J. 36–M as in the average of the tests shown in table 16 demonstrates that this

TABLE 17.—*Percentage of healthy primary shoots produced by field-run mosaic seed cane, with subsequent readings to show the combined effect of recovery in the field, tillering, and natural mortality (due to crowding) on the subsequent proportion of healthy stalks*

[Planted Sept. 4, 1931—all growth frozen back Mar. 9, 1932]

Variety	1931									1932								
	October 13			November 16			December 11			February 10			May 11			September 9		
	Stalks observed	Healthy shoots		Stalks observed	Healthy shoots		Stalks observed	Healthy shoots		Stalks observed	Healthy shoots		Stalks observed	Healthy shoots		Stalks observed	Healthy shoots	
		Total	Proportion		Total	Proportion		Total	Proportion		Total	Proportion		Total	Proportion		Total	Proportion
	Number	*Number*	*Percent*	*Number*	*Number*	*Percent*	*Number*	*Number*	*Percent*	*Number*	*Number*	*Percent*	*Number*	*Number*	*Percent*	*Number*	*Number*	*Percent*
P. O. J. 234	194	22	11.3	205	22	10.7	269	33	12.3	277	32	11.6	728	154	21.2	272	60	22.1
P. O. J. 36-M	124	39	31.5	133	67	50.4	127	57	45.0	108	75	69.4	207	139	67.1	238	164	68.9
P. O. J. 213	113	0	0	127	3	2.4	164	6	3.7	239	0	0	526	31	5.9	355	26	7.3
Co. 281	99	0	0	100	0	0	127	0	0	232	0	0	441	0	0	432	0	0

variety has approximately the same ability to produce healthy plants under conditions of quick germination as under ordinary conditions where it lies in the ground during the winter before much germination takes place. In this test, however, there was only about half as much recovery in P. O. J. 234 during germination as was recorded in table 16. The percentage in P. O. J. 213 in both experiments was too small for reliable comparison.

RECOVERY SHOWN BY GREENHOUSE GERMINATIONS IN THE ABSENCE OF SECONDARY INFECTION

The complicating effect of possible secondary infections in interpreting the results of field studies reported in table 17 made it desirable to repeat the tests in an insectproof greenhouse. Accordingly, during 1932 and 1933, 20 to 100 pedigreed mosaic stools containing 1 to 8 stalks each were selected at random from the plots that furnished the data for table 17, and every stalk was cut into single-eye seed pieces and sprouted in the greenhouse. Preliminary indexing tests showed that reliable results could not be expected from very young plants, but that at least 8 to 10 weeks of growth were desirable before final judgment of their mosaic status.

The data presented in table 18 show that pedigreed mosaic seed of Co. 281 and P. O. J. 213 produced less than 3 percent healthy offspring in greenhouse trials; P. O. J. 234 varied during the 2 years from about 6 to 19 percent; while comparable seed of P. O. J. 36–M produced 56 to 62 percent healthy cane. These figures from an aphidfree greenhouse are not significantly different from those obtained in the field and indicate that some of the variations in disease expression there observed were not necessarily due to secondary infection. In the greenhouse there were also several cases among P. O. J. 36–M and P. O. J. 234 where individuals were healthy for a considerable part of the time in which they were under observation, but were tabulated as mosaic if they showed the disease distinctly at any time. Thus, the percentage of healthy plants shown in table 18 is less than the actual percentage at any stated time of observation.

TABLE 18.—*Extent of recovery manifested at germination by single-eye cuttings from pedigreed mosaic stools sprouted in an aphidfree greenhouse*

Variety	Year	Cuttings germinating	Healthy plants	
			Total	Proportion
		Number	*Number*	*Percent*
Co. 281	1932	411	8	1.9
	1933	117	0	0
P. O. J. 36 M	1932	1,261	782	61.9
	1933	101	56	55.8
P. O. J. 213	1932	518	15	2.7
	1933	100	0	0
P. O. J. 234	1932	1,947	372	19.1
	1933	105	6	5.7

The fluctuating expression of symptoms on occasional young plants in the field could not be regarded as particularly significant, because the situation was complicated by the possibility of new infections from aphids. The recurrence of infection in an aphidfree greenhouse is significant, however, in connection with an eventual explanation of the recovery process. The fluctuations here noted are not unlike the wavering host-parasite relationship in certain mycorhiza and other better known parasitic diseases. The transient appearance and disappearance of mosaic symptoms on these greenhouse plants suggested that in certain cases, at least, recovery was not necessarily caused by absence of the virus from those eyes producing healthy shoots, but that it was probably initially present and later died or failed to multiply, and was strained out during germination or early growth of the young plant. Whatever the explanation, the condition usually became stabilized in a few weeks, after which the plant remained either healthy or mosaic. This was demonstrated by growing the plants one season and reindexing the new stalks. Thus, in 1932, 126 eyes from such recovered shoots were germinated in the greenhouse and gave 100-percent healthy plants, whereas 111 eyes from initially mosaic stalks gave 47.7 percent healthy.

The sprouting of healthy plants from mosaic seed cane, as described in the foregoing sections, is, according to Stahl and Faris (*60*), apparently not to be regarded as recovery. After some preliminary indexing of P. O. J. 2714 in Cuba, in which they obtained some healthy plants from mosaic seed, they concluded: "The main point of interest is the fact that the mosaic is not distributed throughout the whole stalk, as it is in susceptible varieties." Unequal distribution, as opposed to general distribution followed by recovery during or immediately after germination, appeals to the writers, in view of the above-mentioned observations, as the less plausible explanation. When, throughout an entire growing season, the successive new leaves formed by the terminal bud in elongation of the stalk have continuously shown the symptoms, it is inconceivable that the lateral buds subtending those leaves should not also have been invaded by the virus.

DISTRIBUTION OF RECOVERY IN DIFFERENT PARTS OF THE PLANT AS SHOWN BY GREENHOUSE GERMINATION

By indexing and germinating individual eyes of pedigreed mosaic planting material, the proportionate contribution of healthy offspring by different stools and by primary and secondary stalks of the same stool, as well as of the different eyes on the same stalk, may be appraised. In May 1931, a large number of mosaic primary shoots of four commercial varieties in the experiments reported in table 17 were tagged, as well as all subsequent secondary shoots or suckers as they appeared, giving in October the following number of stools in which all stalks had shown the disease throughout their growth: 12 for Co. 281, 85 for P. O. J. 36 M, 69 for P. O. J. 213, and 145 for P. O. J. 234. During the winter of 1931-32, every stalk was divided into single-node cuttings; these were germinated in the greenhouse as in the preceding experiments, and the young plants were grown until the presence or absence of mosaic symptoms could be definitely ascertained.

The extent and distribution of healthy offspring from individual eyes representing the different varietal populations are classified in

table 19 on the basis of stool units and individual stalks. The data in the first two sections of the table compare only those stools of 3 or more stalks each. In section 1 only one-third of the stools of P. O. J. 36–M failed to show one or more stalks with exclusively healthy plants, while 3 (6.7 percent) of the 45 stools yielded entirely healthy offspring, i. e., complete germination recovery of all offspring from every stalk of the original stool. In section 2, only 11.1 percent of the stools of P. O. J. 36–M failed to give any healthy plants. Thus, there is an even greater shift toward freedom from disease when total eyes per stool, irrespective of stalks, are considered. This is, of course, explained by the large number of stalks producing both healthy and diseased plants. P. O. J. 234, in conformity with earlier tests, showed much less recovery; none of the stalks in most of the stools (87.9 percent) gave exclusively healthy plants.

TABLE 19.—*Frequency distribution of healthy offspring from indexed pedigreed mosaic seed of 4 commercial varieties of sugarcane in Louisiana*

1. DISTRIBUTION OF STOOLS ACCORDING TO THE PERCENTAGE OF WHOLE STALKS PER STOOL PRODUCING HEALTHY PLANTS

Variety	Frequency classes (percent healthy)						
	0	1–20	21–40	41–60	61–80	81–99	100
	Per-cent	*Per-cent*	*Per-cent*	*Per-cent*	*Per-cent*	*Per-cent*	*Per-cent*
P. O. J. 36–M (45 stools)	33. 3	2. 2	31. 3	13. 3	8. 8	4. 4	6. 7
P. O. J. 234 (58 stools)	88. 9	6. 9	4. 2				

2. DISTRIBUTION OF STOOLS ACCORDING TO THE PERCENTAGE OF TOTAL EYES PER STOOL PRODUCING HEALTHY PLANTS

P. O. J. 36–M (45 stools)	11. 1	6. 7	13. 6	11. 2	29. 9	20. 8	6. 7
P. O. J. 234 (58 stools)	32. 7	24. 1	27. 7	12. 1	3. 4		

3. DISTRIBUTION OF STALKS ACCORDING TO THE PERCENTAGE OF EYES PER STALK PRODUCING HEALTHY PLANTS

P. O. J. 36–M (257 stalks)	26. 1	2. 7	5. 8	8. 2	12. 1	6. 2	38. 9
P. O. J. 234 (359 stalks)	50. 7	12. 0	20. 6	8. 9	4. 7	.4	3. 3
P. O. J. 213 (166 stalks)	92. 8	4. 8	1. 8		.6		
Co. 281 59 stalks)	93. 2	5. 1	1. 7				

The classification of individual stalks according to the status of their offspring (section 3, table 19, which includes the full population of primary and secondary stalks), shows the majority of the stalks of P. O. J. 36–M concentrated at the extremes of the frequency table (fig. 22). This tendency of individual stalks (65 percent of the total) to produce either all-diseased or all-healthy shoots, which is not characteristic of entire stools (section 1), emphasizes the stalk rather

than the stool as the more important physiological unit in reaction to mosaic. This is strikingly evident only in P. O. J. 36–M, however, which most readily recovers from the disease, although examination of the detailed data on P. O. J. 234 reveals the same general tendency (fig. 22). Co. 281 and P. O. J. 213 exhibited, as in previous tests, very little germination recovery.

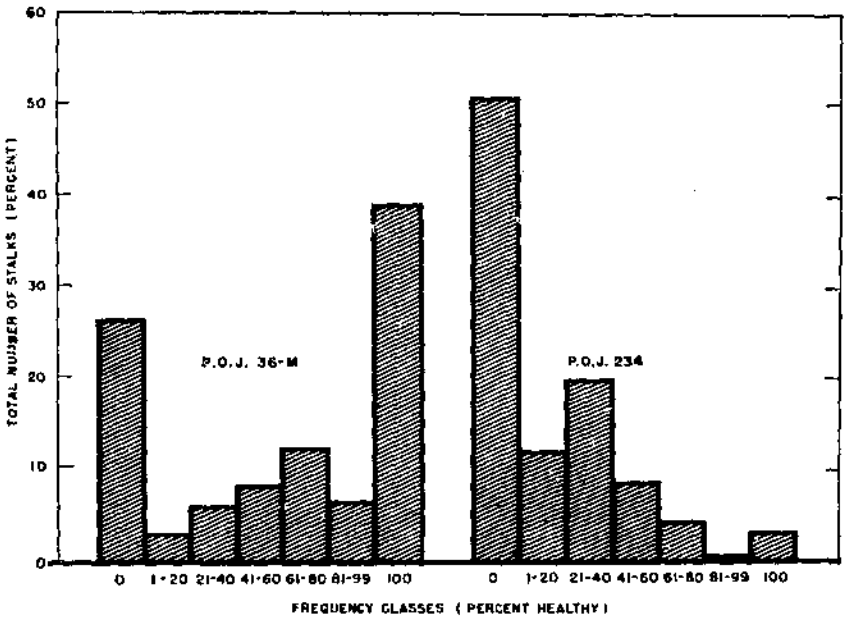

FIGURE 22.—Frequency distribution of percentage of stalks yielding different percentages of healthy shoots upon germination of pedigreed mosaic seed of P. O. J. 36–M and P. O. J. 234.

The possibility of differential rates of germination recovery was examined by classifying the same pedigreed mosaic stalks according to the total number of nodes per stalk and determining the percentage of recovery for each stalk class. The results are presented in table 20. The figures show a slight tendency toward an increase in germination recovery for the longer over the shorter stalks in P. O. J. 36–M and an even slighter but opposite tendency, i. e., toward a decrease, in P. O. J. 234, if the stalks at each extreme, where the populations are very small, are eliminated from consideration. The variations in percentage between individual classes, however, are so great that it is doubtful whether any significance can be attached to the tendencies noted.

The possibility that the percentage of recovery at the time of germination might vary for different parts of the stalk was investigated. This was accomplished by dividing each stalk into seven segments, each representing a definite percentage of the total number of buds per stalk. Segments 1 and 7 each received 12.5 percent and segments 2 to 6, inclusive, 15 percent of the buds. These values were selected because of ease of classification and the tendency for the

TABLE 20.—*Stalk classes, according to total number of nodes, with data on percentage of sprouting and germination recovery on P. O. J. 36–M and P. O. J. 234*

Stalk class	P. O. J. 36–M				P. O. J. 234			
	Stalks	Total nodes	Nodes sprouting	Healthy sprouted nodes	Stalks	Total nodes	Nodes sprouting	Healthy sprouted nodes
	Number	*Number*	*Percent*	*Percent*	*Number*	*Number*	*Percent*	*Percent*
7	8	56	27	53	3	21	19	0
8	6	48	37	89	5	40	22	22
9	10	90	29	73	6	54	19	0
10	24	240	32	58	6	60	38	35
11	23	253	23	35	18	198	32	8
12	31	372	32	54	25	300	29	23
13	33	429	42	62	32	416	31	20
14	31	434	60	35	38	532	34	28
15	37	555	41	70	47	705	36	18
16	19	304	39	74	48	768	34	22
17	21	357	32	58	42	714	36	21
18	11	198	38	59	44	792	37	17
19	4	76	34	50	21	399	33	14
20	3	60	42	56	18	360	35	22
21	2	42	43	72	11	231	35	21
22					5	110	42	4
23	1	23	43	100	3	69	49	6

top (No. 7) and bottom (No. 1) segments to include more buds than the others if the segments all represented the same percentage. Since the populations considered were large, a sufficient number of buds was included in each segment to make the results reliable.

A tendency toward lowered germination, as well as lowered recovery at both the base and top of the stalks, suggested the advisability of making a comparison between them. Accordingly, both germination and recovery percentages were computed for each of the seven segments of the same stalks of P. O. J. 36–M and P. O. J. 234 given in section 3, table 19. The results are presented in table 21 and shown graphically in figure 23. It will be noted that there is a perfect correlation between the percentages of germination and recovery in both varieties. In every case a reduction or increase in germination was accompanied by a corresponding reduction or increase in recovery, and, in most cases, there was a very close adherence in the sharpness of the two curves from one segment to the next.

The agreement of the curves for the two varieties is so close (there being a divergence only in segment No. 6) that it suggests the possibility of there being some relation between the position of a bud on the stalk and its chance for both germination and recovery from mosaic. This holds especially for those buds that fall in the two lower segments, there being a sharp rise in all curves from the first to the second. The most logical explanation for this behavior seems to be the comparatively more dormant condition of the lower (and older) buds. This dormancy apparently causes lower germination,

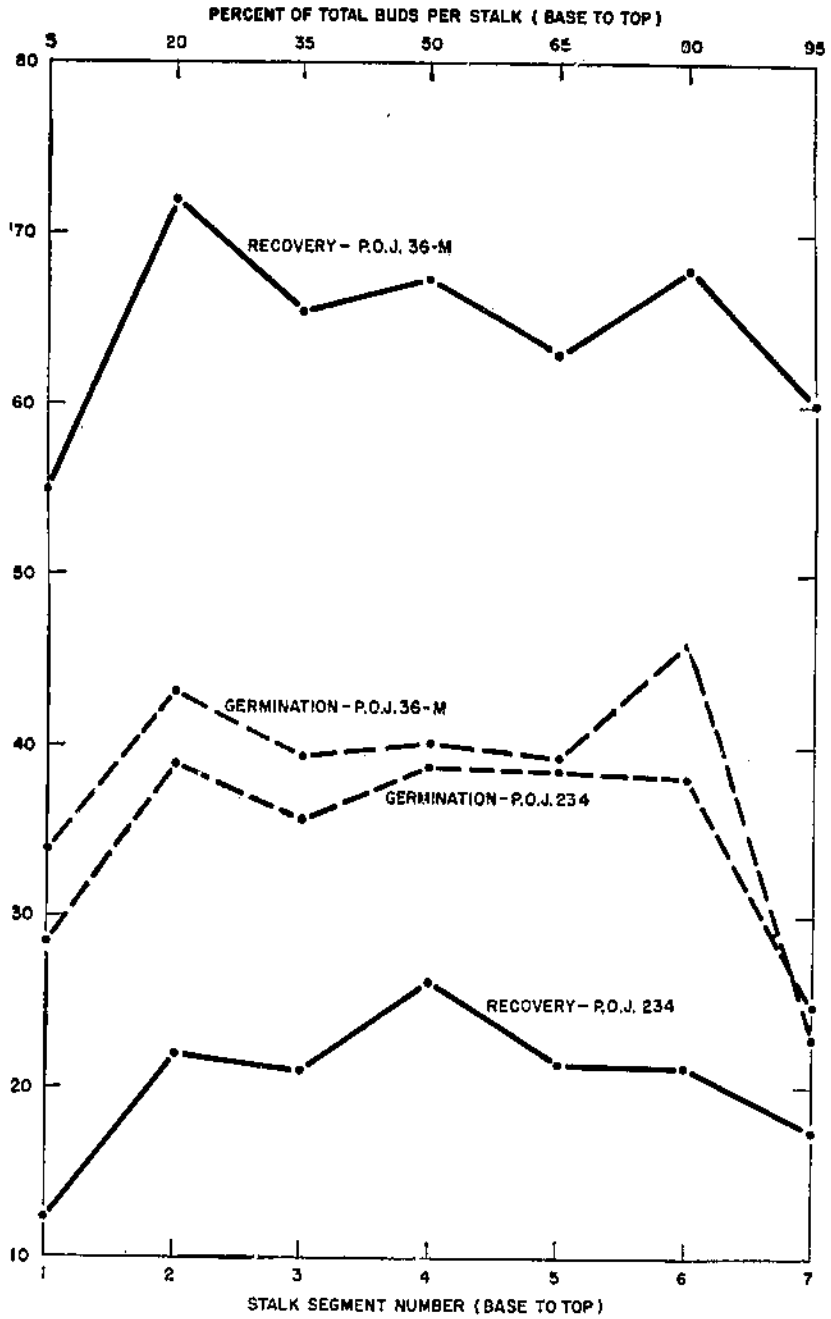

FIGURE 23.—Comparative extent of recovery in different segments of the mosaic stalk and its correlation with percentage of germination in the varieties P. O. J. 36–M and P. O. J. 234. Germination and recovery of individual buds on more than 250 stalks of each variety are averaged by segments of each stalk from base to top (Nos. 1 to 7, inclusive), according to their percentage location on the stalk.

TABLE 21.—*Relation of germination recovery in P. O. J. 36–M and P. O. J. 234 to position of buds on the individual stalk* [1]

Variety	Frequency-class subdivisions of stalk (base to top) expressed as percentages of total buds per stalk						
	5	20	35	50	65	80	95
P. O. J. 36–M (257 stalks):							
Stalk representation:							
Number	136	151	142	148	141	149	89
Percentage of total	52. 9	58. 7	55. 3	57. 6	54. 9	58. 0	36. 4
Percentage showing recovery	57. 4	73. 5	64. 8	69. 6	66. 7	71. 1	67. 4
Buds planted:							
Total number	534	471	454	495	453	494	510
Percentage germinating	33. 7	43. 1	39. 4	40. 2	39. 3	46. 2	22. 9
Percentage recovering	55. 0	71. 9	65. 4	67. 3	62. 9	68. 0	59. 8
P. O. J. 234 (364 stalks):							
Stalk representation:							
Number	188	206	217	220	214	207	134
Percentage of total	51. 6	56. 6	59. 6	60. 4	58. 8	56. 9	36. 8
Percentage showing recovery	15. 4	26. 2	23. 5	32. 7	26. 2	27. 1	23. 1
Buds planted:							
Total number	862	784	791	805	791	829	817
Percentage germinating	28. 4	39. 0	35. 7	38. 8	38. 6	38. 2	24. 7
Percentage recovering	12. 2	21. 9	20. 9	26. 3	21. 3	21. 1	17. 3

[1] Each stalk was divided into 7 segments based on the total number of buds per stalk, and the segments were compared for percentage of stalks germinating and showing recovery and for percentage of buds germinating and showing recovery.

and the resultant growth may be less active with less recovery occurring.

A similar, but less marked, tendency in the opposite direction is noted for the two upper segments of the stalks. The lower germination here is a result of immature succulent tissues that often disintegrate before the young shoots become established. The difficulty they experience in becoming established and, in many cases at least, the resultant growth retardation may explain the lowered recovery in the top segment. Thus we find a low recovery rate in the top and bottom segments correlated with poor germination, which is due in the two cases to very different causes.

The comparative extent of healthy offspring from diseased primary, or mother, shoots and from suckers, or tillers, revealed no significant difference, the figures for P. O. J. 36–M being 56.8 and 56.5 percent, respectively, and for P. O. J. 234, 20.2 and 18.2 percent.

RECOVERY IN GROWING SUGARCANE

All the data presented so far have had to do only with recovery manifested in the production of healthy offspring by the germination of cuttings from diseased stalks. It has been shown in repeated tests with P. O. J. 36–M that as high as 50 percent of the stand derived

from pedigreed mosaic seed cane may start off in spring free from the disease. Later during the growing season the total number of mosaic-free plants (barring excessive secondary spread) is further augmented by foliage recovery of many of those diseased at the time of germination. This is brought about by the production of new leaves without symptoms and the gradual dying and shedding of the earlier diseased leaves incident to the elongation of the stalk characteristic of all grasses.

An evaluation of this phenomenon as a varietal characteristic in relation to mosaic control necessarily involves the determination of (1) its comparative extent among commercial varieties, (2) variation in extent from year to year, (3) possible correlation between foliage recovery and germination recovery, (4) relative incidence among diseased plants from pedigreed mosaic and recovered lines of seed, and (5) proportion of healthy offspring shown by germination of foliage-recovered sugarcane. While additional information is desirable on most of these points, it is believed that the data presented in the following pages indicate sufficiently definite trends to warrant their publication at this time.

Systematic observations of mosaic plants for evidence of recovery by means of the individual stool- and stalk-pedigree method, already described, were carried out during the growing seasons of 1930–32, inclusive. The stools selected were always scattered through replicated plots of yield tests laid down to compare the effect on yield of planting mosaic-infected seed. Therefore, the observations on different varieties are strictly comparable, and, furthermore, the extent of disease in the regularly distributed healthy plots gave some measure of secondary infections.

In 1930 only two varieties, P. O. J. 36–M and P. O. J. 234, were compared, but in 1931 and 1932 as mosaic seed of other varieties became available, observations were extended to them. There also became available, during the latter 2 years, a considerable quantity of sugarcane produced by germinating the individual eyes of the stalks of P. O. J. 36–M and P. O. J. 234 that recovered in 1930. These were indexed by nodes and germinated in the greenhouse and the mosaic plants transferred to the field, where systematic observations were made on them in comparison with stools from pedigreed mosaic seed. A number of these individual eye-cuttings of recovered stalks produced mosaic plants in the greenhouse, and many others initially healthy later became diseased in the field, presumably owing to secondary infection. These two categories furnished an appreciable supply of diseased stalks, all of which were observed for recovery.

Unfortunately, the findings for 1931 and 1932 are complicated by a great deal of secondary spread, as shown by mosaic incidence in the healthy plots, so that the results are not so free from error as could be desired. Nevertheless, owing to the very large populations observed, they are significant from a relative standpoint and are indicative at least of results to be expected under such conditions. The figures for 1930, however, were obtained under conditions of approximate freedom from secondary spread and, therefore, should be fairly representative of the natural tendencies of the two varieties compared that year. These data are presented in table 22.

TABLE 22.—*Summary of 3 years' observance on occurrence of foliage recovery in commercial varieties, including comparisons of pedigreed mosaic and recovered lines, and the proportion of healthy offspring produced on germination of recovered stalks*

Variety and source of seed	Year	Crop	Soil type	Stools	Mosaic stalks	Recovered stalks		Recovered stalks indexed			
								Number	Eyes germinating	Eyes producing healthy shoots	
										Total	Proportion
				Number	*Number*	*Number*	*Percent*		*Number*	*Number*	*Percent*
P. O. J. 36–M:											
Pedigreed mosaic	1930	Plant cane	Sandy	10	48	7	14.6	7	43	29	67.4
	1930	do	Clay	15	95	13	13.7	13	104	60	57.7
	1931	First ratoon	do	165	537	6	1.1	6	38	38	100.0
	1932	Second ratoon	do	50	146	2	1.4	2	10	10	100.0
	1931	Plant cane	Sandy	94	448	13	2.9	13	83	82	98.8
	1932	First ratoon	do	50	299	1	.3	1	8	8	100.0
	1932	Plant cane	do	50	303	0	0	0	0	0	-------
Recovered line	1931	do	Clay	54	392	46	11.7	9	98	97	99.0
	1932	First ratoon	do	114	546	24	4.4	21	154	153	99.4
	1932	Plant cane	Sandy	9	72	4	5.6	3	33	31	93.9
P. O. J. 234:											
Pedigreed mosaic	1930	do	Clay	34	219	2	.9	1	6	2	33.3
	1931	First ratoon	do	86	368	0	0	-------	-------	-------	-------
	1932	Second ratoon	do	50	150	0	0	-------	-------	-------	-------
	1931	Plant cane	Sandy	174	870	2	.2	2	10	10	100.0
	1932	First ratoon	do	50	162	0	0	-------	-------	-------	-------
	1932	Plant cane	do	50	331	0	0	-------	-------	-------	-------
Recovered line	1931	do	Clay	3	20	0	0	-------	-------	-------	-------
	1932	First ratoon	do	7	35	2	5.7	2	20	20	100.0
	1932	Plant cane	Sandy	89	641	2	.3	0	0	0	-------

P. O. J. 213: Pedigreed mosaic.	1931	...do...	--	...do...	131	824	0	0				
	1932	First ratoon		...do...	50	385	0	0				
	1932	Plant cane		...do...	50	317	0	0				
Co. 281: Pedigreed mosaic.	1931	...do...		Sandy	215	1,125	0	0				
	1932	First ratoon		...do...	50	370	0	0				
	1932	Plant cane		...do...	50	301	0	0				
L-511: Pedigreed mosaic.	1931	...do...		Sandy	27	64	0	0				

During one year (1930) when recovery was not appreciably influenced by secondary spread of mosaic, P. O. J. 36–M, with 14.6 percent, was outstanding. Recovery for P. O. J. 234 was only 0.9 percent.

During 1931 five varieties were observed for recovery, but none occurred in P. O. J. 213, Co. 281, or L–511, and only 1.9 percent (19 cases) in pedigreed P. O. J. 36–M and 0.16 percent in P. O. J. 234. Similar results were obtained in 1932, when the percentages of the last two varieties were reduced to 0.4 and 0, respectively. L–511 was not observed in 1932. The discrepancies in the percentages for 1931 and 1932 as compared with 1930 may be explained by the great waves of secondary spread of mosaic during 1931 and 1932. This spread, caused by more or less constant introduction of new sources of infection by aphids, apparently thwarted most cases of recovery before they could be observed. In a few cases at least, the plants put out healthy new leaves but later again showed the symptoms, which persisted until harvest.

Probably the most surprising disclosure of these tests is the failure of P. O. J. 213 to show any foliage recovery. In view of its behavior during the period 1926–30, when mosaic became practically nonexistent in this variety in Louisiana after it was approximately 100 percent diseased in 1925, it was expected that it would exhibit more recovery than P. O. J. 36–M.

CORRELATION OF FOLIAGE WITH GERMINATION RECOVERY

Comparison of the percentage of foliage recovery, as shown in table 22, with those for germination recovery (tables 16 and 18) indicates sufficient agreement in trend to suggest a positive correlation between the two types of recovery. The comparative figures for the four commercial varieties—P. O. J. 36–M, P. O. J. 234, P. O. J. 213, and Co. 281—reveal the latter two showing no foliage recovery and practically no recovery at germination, while P. O. J. 234 shows a small degree of foliage recovery and considerable recovery (up to 25 percent) at germination. P. O. J. 36–M showed both the greatest recovery of foliage and the greatest from germination.

These preliminary observations suggest that should recovery at such widely separated stages of growth have the same physiological basis, the responsible factor (or factors) operating during the early stages in both varieties later apparently regains a considerable intensity only in P. O. J. 36–M. Such a hypothesis naturally presumes recovery at germination and not simply unequal distribution of the virus in the seed piece.

RECOVERY BY PEDIGREED-MOSAIC AND PREVIOUSLY RECOVERED LINES

The observations in 1931 and 1932 on foliage recovery among plants grown from pedigreed seed (table 22) included more or less comparably situated plants of P. O. J. 36–M and P. O. J. 234, which had been transplanted in spring from the greenhouse. These were the mosaic plants obtained by indexing and germinating individual stalks showing foliage recovery the preceding year (1930) and, therefore, represent a recovered line.

Owing to the possible influence of transplanting, uncertainty of

secondary infection, differences in recovery between plant cane and ratoons, and the fact that the plots were neither located nor arranged to compare these two sources of seed, definite conclusions regarding the relative tendencies toward recovery of pedigreed and recovered lines cannot be made. In all comparisons, however, regardless of year, location, or crop involved, the percentage of recovery among the previously recovered lines, despite secondary spread, was never less than 3 times, and more often 10 to 12 times, as great as any comparable percentage of the pedigreed mosaic selections.

The counts to determine mosaic spread in healthy sugarcane showed no appreciable variation within either soil area, so that it is permissible to compare the two objects on the same soil type. Therefore, in 1932, so far as secondary spread is concerned, the two ratoon crops on clay soil and the two plant-cane crops on sandy soil are comparable and show such difference in favor of the recovered line as to suggest a definitely greater tendency toward recovery.

PRODUCTION OF HEALTHY SUCKERS FROM INITIALLY MOSAIC MOTHER SHOOTS OF A RECOVERED LINE OF SEED

The preceding studies showed that mosaic plants from certain joints of recovered stalks gave an appreciably greater degree of foliage recovery when grown to maturity in the field than similar pedigreed mosaic planting material. Throughout this work it was noted that in the growth and tillering of mosaic primary shoots from such previously recovered stalks, there were occasional suckers that from the beginning appeared to be free of symptoms. In observations covering hundreds of stools over a period of 3 years no such development was ever observed from pedigreed mosaic cuttings. Invariably the suckers from the pedigreed mosaic plants came up diseased.

Data were examined to determine the frequency of production of healthy suckers from mosaic mother shoots derived from recovered seed, and out of a total of 30 stools in which the primary shoot was initially diseased, 5 stools were followed in which part or all of the suckers came out and remained healthy. A typical case on the variety P. O. J. 36–M observed in 1931 is shown in table 23. The proportion of unquestionable cases might have been higher had it not been for the masking effect of secondary mosaic spread.

PROPORTION OF HEALTHY SHOOTS FROM FOLIAGE-RECOVERED CANE

The results of germination in the greenhouse of indexed single-joint cuttings of most of the stalks that showed foliage recovery are presented in the last column of table 22. A glance at the figures shows a much higher production of healthy shoots than was obtained from mosaic stalks from the same plots, as shown in tables 16 to 21. There the average for P. O. J. 36–M was approximately 50 percent and here it varies from 57.7 to 100 percent. Therefore, the totals in table 22 must represent a summation of the effect of foliage recovery as well as any germination recovery that may be operative in the sprouting of eyes that otherwise might have manifested the disease. An evaluation of the relative influence of these two factors has not been attempted.

It may be noted that in 1930, in the absence of any noteworthy secondary spread, a high percentage of foliage recovery resulted in a

TABLE 23.—*Production of healthy suckers from a continuously diseased mother shoot derived from a recovered stalk*

Stalk recovery in field September 1930; indexed and germinated in greenhouse, November 1930		Observations [1] on primary shoot and suckers from node No. 4, transplanted to field June 20, 1931								
			July		Aug.		Sept.		Oct.	Dec.
Node No. (base to top)	Condition of plant	Plant part	25	31	13	29	11	21	28	4
11	Healthy.									
10	Do.									
9	Mosaic.									
8	Do.									
7	Failed.	Mother shoot	M	M	M	M	M	M	M	M
6	Healthy.	Suckers:								
5	Do.	1		H	H	H	H	H	H	H
4	Mosaic	2		H	H	H	H	H	H	H
3	Do.	3		H	M	M	M	M	M	M
2	Failed.	4		H	H	H	H	H	H	H
1	Healthy.	5		H	H	H	H	H	H	H
		6		H	D					

[1] M=mosaic; H=healthy; D=died.

much lower percentage of healthy progeny than was obtained in 1931 and 1932, when secondary infections were abundant and foliage recovery was low. This suggests a selective process, whereby under conditions of abundant secondary infection only the most persistent cases of recovery were shown. Healthy plants obtained both from such foliage-recovered stalks and from diseased stalks were grown from three successive vegetative generations and produced only healthy sugarcane. This is believed to be sufficient proof that they no longer contained the virus and had, in fact, completely recovered from the disease. Such plants, however, are not immune to new mosaic infection, for under field conditions they show practically the same susceptibility to secondary infection as normally healthy cane.

Thousands of additional observations both in the greenhouse and in the field, not summarized here because of similarity of the results to those already presented, were made on the incidence of both foliage and germination recovery in the three P. O. J. varieties 36–M, 213, and 234 and in Co. 281. The studies on P. O. J. 36–M and P. O. J. 234 were made with further vegetative generations of the material, mosaic infection of which presumably dated back to about 1925, whereas the studies on P. O. J. 213 and Co. 281 were made on material infected presumably in 1930 and 1931, respectively. The fallacy of such comparisons did not become apparent until some time later, because the existence of strains of the mosaic virus had not been proved and all mosaic cane was assumed to be comparable except for possible suspected differences in concentration of the virus in the host. Later germination observations on cuttings of P. O. J. 36–M and

P. O. J. 234 newly infected with known strains of the virus resulted in complete absence of recovery.

Résumé of Recovery

The recovery presented here demonstrates a widespread disappearance of symptoms in Louisiana over a period of years and actual recovery from mosaic by certain tolerant varieties. In the case of P. O. J. 36–M, in which the original infection probably dated back to about 1925, it was shown that germination recovery to the extent of about 50 percent 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.

Typical results obtained by indexing stalks of four different categories with respect to their mosaic history in the greenhouse are shown in figure 24. Pedigreed mosaic stalks of both varieties (*A*) usually produced a number of, or occasionally all, healthy plants upon germination. Stalks that showed foliage recovery (*B*) in the field usually produced mostly healthy plants, but frequently, particularly in P. O. J. 36–M, a number that were mosaic. When these healthy plants were grown to a size sufficient for making cuttings they (*C*) invariably produced only healthy plants, while the mosaic progeny of recovered stalks (*D*) behaved much like the original pedigreed mosaic material. *A* and *B*, of course, represent one vegetative generation, while *C* and *D* are the next vegetative generation of the two types of progeny in *B*. Recovery, although complete, does not confer immunity on the host, as witness the incidence of secondary spread in the field comparable with that for normally healthy cane.

Very little recovery of either type was observed in newly infected Co. 281 and P. O. J. 213 in these studies, although the latter had, during an earlier period of low secondary spread, practically eliminated the disease in sections where it had previously shown 100 percent infection. The failure of this variety to exhibit any appreciable recovery after being newly infected in 1930 and subsequently was a significant development, the import of which was not suspected at the time. The comparison of this material with that of P. O. J. 36–M, in which the mosaic infection presumably dated back to about 1925 and in which differential rates of recovery were demonstrated for two "lines"—later found to be different strains of mosaic—suggested the hypothesis that recovery in P. O. J. 36–M was explainable on the basis of attenuation of the virus. Later field germinations with cuttings from plants artificially infected with known strains of the virus, however, failed to yield any recovery in P. O. J. 36–M or P. O. J. 234, and at least two strains have been found from which Co. 281 recovers readily.

Tims, Mills, and Edgerton (*68*), of the Louisiana Experiment Station, have reported detailed studies on the recovery of the P. O. J. and other varieties that are in general agreement with the foregoing account. These authors conclude that of the several possible explanations of the peculiar behavior exhibited by P. O. J. 213 and some other varieties "the most likely one seems to rest on the existence of more than one strain of the mosaic virus."

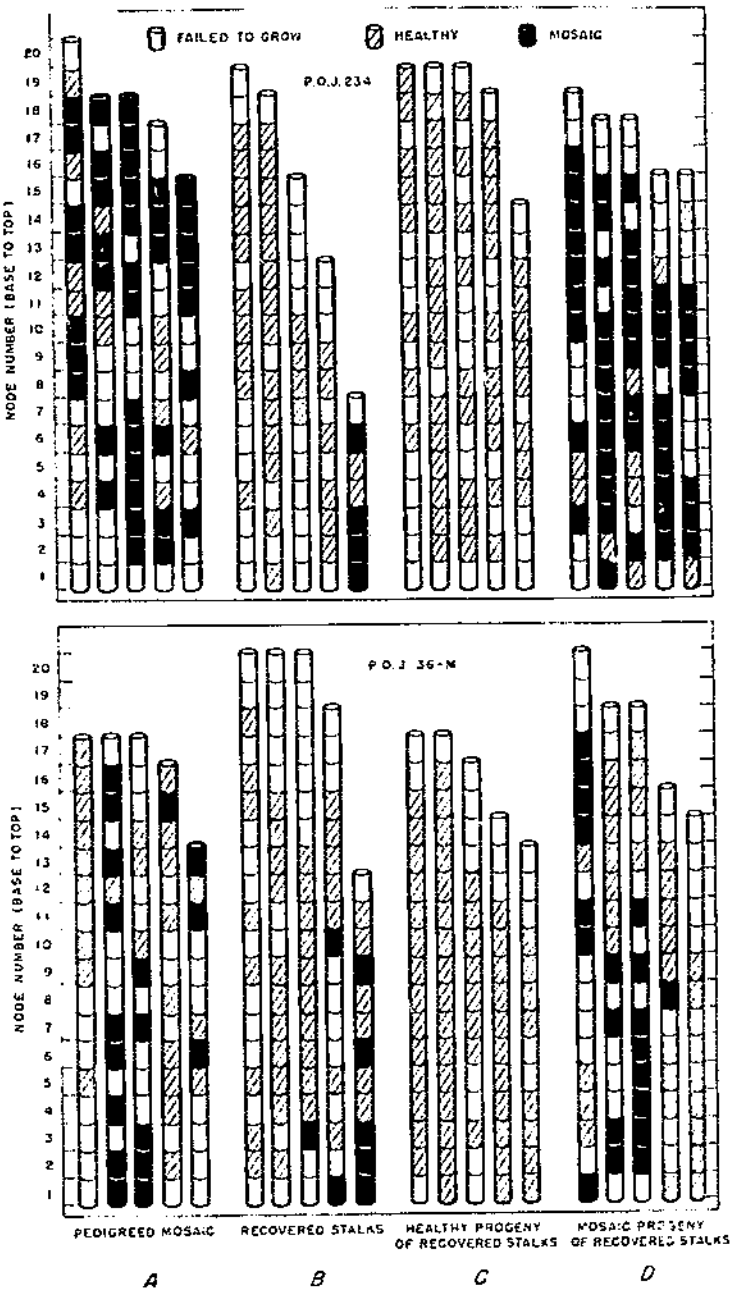

FIGURE 21.—Typical results obtained by indexing stalks of four different categories with respect to their mosaic history: *A*, Pedigreed mosaic stalks yield both diseased and healthy offspring; *B*, recovered stalks produce very few diseased offspring; *C*, healthy progeny of recovered stalks produce only healthy offspring; and *D*, mosaic progeny of recovered stalks behave much like pedigreed mosaic seed.

The discontinuation of these recovery studies was necessitated by the pressure of other work. Loss of the original material of the P. O. J. varieties makes it impossible to resume them where they were dropped. Recent developments of the mosaic problem, however, particularly from the standpoint of strains of the virus, have thrown a great deal of light upon the results previously obtained, and these would be of great value if study of the recovery problem should be resumed. At this time it seems logical to assume that occurrence of recovery is probably dependent largely upon the virus strain involved, which conceivably might be different for different varieties of cane. The conviction persists, however, that attenuation, or thinning out of the infective agent, is also a factor, but that it may be influenced to a great extent by local factors of environment and, in the case of foliage recovery, by reinfection by insect vectors.

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