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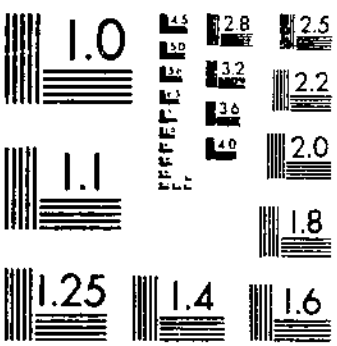

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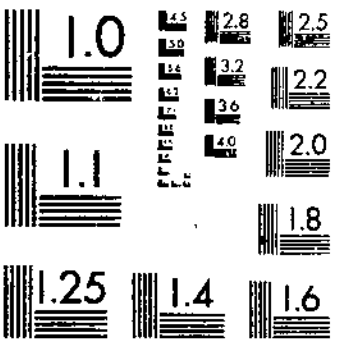

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STRAINS OF SUGARCANE MOSAIC VIRUS

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Contents

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STRAINS OF SUGARCANE MOSAIC VIRUS

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Variations in the symptom pattern of mosaic on sugarcane varieties were noted long before the existence of strains of the virus was known. Edgerton and Taggart (1924) [1] selected plants of the varieties D-74 and Louisiana Purple with mild mosaic symptoms in contrast to the more severe pattern on most plants. They interpreted the observed differences as due to variants of the sugarcane clones rather than of the pathogen, and recommended selection of mosaic "tolerant" clonal lines as a means of reducing loss from mosaic (Edgerton, Taggart, and Tims 1926). Tims and Edgerton (1931) also stated that it was possible to select plants of sugarcane clones that were decidedly resistant to mosaic. Brandes (1927) said that Edgerton's and Taggart's results led him to believe there might be more than one strain of the virus.

Tims and Edgerton (1931) suggested that differences in degree of infection with mosaic, which they observed in four varieties of sugarcane at two localities in Louisiana, indicated the presence of two strains of the virus. However, this hypothesis of strain occurrence was based on locality differences in mosaic incidence rather than differences in mosaic symptom pattern. Tims, Mills, and Edgerton (1935) later reported differences in virulence between the viruses from the areas of heavy and light mosaic incidence, and advanced the theory that the virus involved in the light infections was the earlier one in the State, whereas that from the areas of heavy infection was a more virulent strain, which eventually spread over the sugar district and became the dominant one. They concluded that "two very distinct types of mosaic, recognized by very distinct symptoms, occur in Louisiana." Storey (1927) reported two supposed strains of mosaic based on differences in regional distribution and host range in Natal, Africa.

Summers (1934, 1935) was the first to definitely differentiate strains of the virus. He described four strains or "types," differentiated principally by symptoms produced on the sugarcane variety C.P. 28-60. Later Summers (1939) described seven strains, designated A, B, C, D, E, F, and G, and three substrains of D based on symptoms produced on three host varieties, C.P. 31-294, C.P. 29-291, and Co. 281.

Summers, Brandes, and Rands (1948) discussed in detail the experiments that had led to the differentiation of the 10 strains and substrains and published a key for their identification on Summers' differential host varieties. This work was an important contribution to knowledge of the sugarcane mosaic virus (SMV) and its relationship to mosaic reaction of sugarcane varieties. It has provided the basis for explaining the occurrence of mosaic in the succession of varieties that have been grown commercially in Louisiana since 1925.

Lin (1950) described four strains of the virus in Taiwan, designating

[1] References to Literature Cited (p. 23) are herein indicated by the names of the authors followed by the year of publication

them A, B, C, and D, but since his differential hosts were different from those used by Summers, it is impossible to establish any relationship between the strains occurring in the two countries. Later Liu and Li (1953) mentioned only three strains, designated as "short stripe type, yellow stripe type, and fine stripe type."

From a study of mosaic intercepted in quarantine on sugarcane varieties imported into the United States, it is known that strains differing from those described in Louisiana occur in other countries (Matz 1938). Bennett (1941) transferred mosaic from seven sugarcane varieties in Argentina to Summers' differential hosts and identified all as strain B (type 2). Bruehl (1953, 1954) identified strains A, B, and D from several sugarcane varieties in Puerto Rico. Strain B was the most prevalent on all varieties except B. 37161, from which approximately equal numbers of collections of strains B and D were identified. He concluded that relative abundance of different strains in Puerto Rico would be affected by the relative acreage of sugarcane varieties showing marked susceptibility to certain strains.

DIFFERENTIATION OF STRAINS

Symptoms on Differential Hosts

Summers (1934, 1935) and Summers, Brandes, and Rands (1948) differentiated strains of SMV based on the following characters on selected sugarcane host varieties: (1) Nature of the chlorosis, whether mild mottling or a severe pattern of chlorotic blotches and stripes, (2) presence or absence of necrosis in the lesions, (3) leaf sheath discoloration, (4) growth retardation, (5) germination recovery, (6) relative infectiousness or "index of infection," and (7) length of incubation period.

In the investigation of sugarcane mosaic in the Southern United States since 1950, particularly with respect to the relationship of strains of the virus to the incidence of the disease, the authors have studied over 400 accessions of the virus collected from commercial fields and plantings of unreleased varieties. In connection with this work, Summers' stock cultures of five strains and four substrains he described were also studied on various differential hosts. To determine whether physical properties might supplement macroscopic symptoms in strain differentiation, thermal death point (TDP) and dilution end point (DEP) determinations were made on six of the described strains (Abbott 1953) in addition to several unknowns.

Nature of Chlorosis

The most striking macroscopic symptom of sugarcane mosaic is the pattern of chlorosis produced. This may consist of mild mottling with little or no chlorosis, or of a severe pattern of discrete, extremely chlorotic, yellowish to white streaks or blotches. Tims, Mills, and Edgerton (1935) used the terms "green" and "yellow" mosaic to describe this difference in symptoms, which they considered indicative of two "types" or kinds of mosaic. The terms are descriptive of types of symptom pattern, but are not necessarily indicative of virus strain differences, since a strain may produce "green" mosaic symptoms on one variety and "yellow" on another.

On most sugarcane varieties that have been studied, and particularly

the present and former commercial varieties in Louisiana, strains A, B, and H produce a mild mottling or "green" mosaic pattern. The symptoms produced by strain D are more chlorotic or yellow on most varieties, but they are "green" on others. Definite identification of strains cannot be made on the commercial varieties except for strain C, which produces severe chlorosis with necrosis and marked stunting on all varieties on which it has been observed. On selected differential host varieties, however, such as C.P. 31-294 and C.P. 31-588, the two kinds of symptom pattern, i.e., mild mottling or severe chlorosis, are indicative of strain differences and constitute the principal character for separating strains.

Necrosis

In this study necrosis has not been observed accompanying the mild mottling patterns (strains A, F, and H), but it is present to some degree in the severe patterns of pronounced chlorotic stripes (strains B, C, D, and E) produced on differential hosts. Presence or absence of necrosis on selected differential hosts is an important diagnostic character.

Leaf Sheath Discoloration

Summers (1939) and Summers, Brandes, and Rands (1948) based differentiation of substrain De from strain D and, in part, strain G from strain B on the presence or absence of leaf sheath discoloration. In the authors' experience this is an inconstant and unreliable character, at least in greenhouse-grown plants. Plants of Co. 281 grown from Summers' stock culture of substrain De and of C.P. 29-291 infected with his stock culture of strain B did not develop sheath discoloration. Substrain De seems of doubtful validity.

Growth Retardation

In their key for identification of strains, Summers, Brandes, and Rands (1948) used growth retardation as the principal character to differentiate strains D and E, the former causing marked stunting of growth of C.P. 31-294, and the latter none; and strains B and G, the former causing marked stunting of C.P. 29-291, and the latter little or none. However, they presented no growth-measurement data to support their conclusions.

Several accessions of virus that produce similar or identical leaf symptoms on a differential host variety may cause varying degrees of growth retardation which, if only the extremes were observed, might seem to offer a basis for separating them, but when the intermediate degrees of retardation are considered, there is no sharp point of demarcation.

Table 1 shows the variation in height of plants of C.P. 29-291 6½ weeks after inoculation with a stock culture of strain B and with four accessions of virus collected from sugarcane fields in Louisiana (M-451), Mississippi (M-468 and M-471), and Alabama (M-469). On C.P. 31-294 they produced leaf symptoms characteristic of strain B. On C.P. 29-291, plants of M-469 averaged only 9 percent less growth than the healthy control, and on that basis probably could be considered strain G. The other three accessions, however, were intermediate between strain B and M-469 in their effect on growth, and there would be no precise basis for differentiating them from either. Individual plants of each accession showed considerable difference in degree of growth retardation. It seems more logical to consider the four accessions as variants

TABLE 1.—*Effect of strain B and 4 variants on growth of C.P. 29-291, as indicated by height of plants 6½ weeks after inoculation*[1]

Plot No.	Uninoculated healthy check	Height of plants inoculated with—				
		Strain B	M-451	M-468	M-469	M-471
	Mm.	*Mm.*	*Mm.*	*Mm.*	*Mm.*	*Mm.*
1	124	65	76	156	87	91
2	165	106	157	125	138	150
3	116	116	91	81	89	397
4	108	82	63	86	181	97
5	123	110	128	97	149	80
6	173	72	118	101	171	122
7	162	57	61	93	96	64
8	142	64	111	120	89	57
9	108	61	.....	73	123	.....
10	117	.....	.....	90	124	.....
Average	137	81	101	102	125	107
Percent of control	100	59	74	74	91	78

[1] Inoculated Oct. 13 and read Nov. 28, 1961.

of strain B. This example illustrates the difficulty of using growth retardation as a critical basis for strain differentiation. The authors are therefore inclined to consider strain G as a variant of strain B.

Summers, Brandes, and Rands (1948) in their key used growth retardation as the principal basis for differentiating strains E and D. In this instance differences in symptom pattern and in susceptibility of certain sugarcane varieties to the strains offer further valid criteria for differentiating these strains.

Germination Recovery

Germination recovery in Co. 281 was the basis for separating substrain Da from strain D (Summers 1939, Summers, Brandes, and Rands 1948). In 1945 the authors inoculated healthy plants of Co. 281 with virus from Summers' stock culture of strain Da, and 100 single-bud cuttings were later planted from the stalks produced by these plants and grown in the greenhouse for observation of germination recovery. There was no recovery at germination or subsequently. Although some sugarcane varieties may recover from one strain more than from another, this phenomenon often seems to be influenced by other factors affecting growth of the cane. It is the authors' belief that this character is not reliable for strain differentiation and that substrain Da is of doubtful validity.

Index of Infection

Summers, Brandes, and Rands (1948) noted differences in relative infectiousness of strains, which they termed "index of infection," but this was not used as a major diagnostic character. They found that strains A and B, which are very different in macroscopic symptoms, have a higher index than strain D. Infectiousness is affected, of course, by relative susceptibility of different sugarcane varieties to a given strain.

Table 2 shows comparative infection and average incubation periods of four strains of the virus on C.P. 31-588 and C.P. 31-294. One of the accessions each of strains A, B, and D was Summers' stock culture; the others were field collections. The work was done in a greenhouse, where all openings were covered with 30-mesh screen. Inoculum was obtained from young plants grown from cuttings taken from infected plants in commercial fields. Juice was extracted with a food grinder and filtered through cheesecloth. The inoculations were made by pricking into the tissues with a fine needle through a drop of undiluted juice placed in the spindle. The healthy plants of the differential hosts were grown in 4-inch clay pots of steamed soil and were inoculated when about 3 weeks old. The work summarized in table 2 was done during the winter and spring over a 3-year period, 1959–61. Although the results are from experiments performed at different times, growing conditions were fairly comparable for all the experiments, since all the work was done in one greenhouse, where minimum temperatures were maintaind at about 75° F.

The results confirm those of Summers, Brandes, and Rands in showing a lower index of infection for strain D as compared with strains A and B. Strain H has a lower index of infection than the other strains—less than half that of strain D on C.P. 31-294—and a considerably longer incubation period.

Accessions identified as strain H on the basis of symptoms on C.P. 31-588 differed in their infectiousness toward C.P. 31-294. Eighty percent of the collections studied gave very low or no infection on that variety, whereas 20 percent infected it readily. These differences in infectiousness toward C.P. 31-294 could not be correlated with the source of virus in respect to variety or locality. It seems probable, however, that further study on additional differential host varieties might permit separation of viruses identified as strain H on the basis of symptoms on C.P. 31-588.

Strains of SMV differ in relative infectiousness, but the character is not critically diagnostic.

Length of Incubation Period

Incubation period, or the number of days from inoculation to appearance of symptoms, is affected by temperature, moisture, light, and other conditions affecting rate of growth of the inoculated plants, but mosaic strains will show, in general, the same relative differences in incubation period under any given set of conditions. Marked differences in length of incubation period were noted between strains A and F, the former having a short period compared with the long one of strain F (Summers, Brandes, and Rands 1948).

Table 2 shows that strain H also has a relatively long incubation period. Although a few individual plants may show symptoms within 10–12 days after inoculation with this strain, appearance of the first symptoms is usually delayed. Long incubation periods are characteristic of strains F and H, but length of incubation period is not critically diagnostic.

Physical Properties

Abbott (1953) concluded from a study of dilution end point (DEP) and thermal death point (TDP) that determination of tolerances to

TABLE 2.—*Percent infection and incubation period of 4 strains of sugarcane mosaic virus on 2 host varieties*

Strain	Accessions tested	On C.P. 31-588			On C.P. 31-294		
		Plants inoculated	Plants infected	Average days to first symptoms	Plants inoculated	Plants infected	Average days to first symptoms
	Number	*Number*	*Percent*	*Number*	*Number*	*Percent*	*Number*
A	7	74	89.2	18.1	92	65.2	17.7
B	13	106	84.0	18.1	101	70.3	17.9
D	15	181	66.9	19.1	197	45.2	22.3
H	140	902	61.0	27.5	878	20.3	37.2

dilution and heat might be of value in separating strains that show similarity in symptom patterns on differential hosts. Chona (1944) reported TDP's of 45°, 55°, and 65° C., respectively, for three collections of SMV in India, which he thought indicated strain differences. He did not describe his method of determining TDP, but his figure of 65° is considerably higher than that reported by other investigators (Abbott 1953, Adsuar 1950).

Further study of these properties was made in 1959 and 1960 on a group of six virus collections considered variants of strain D and two variants of strain H. The strain D variants produced the extremely chlorotic stripes on C.P. 31-294, characteristic of strain D, but lacked necrosis. The strain H variants differed from the type in the milder symptoms produced on C.P. 31-588, which differentiate that strain.

Aliquots of freshly expressed juice from each source were diluted 1-10, 1-100, and 1-1,000 with distilled water. For TDP determination, 3.5-cc. aliquots of undiluted juice were placed in glass tubes, 9 mm. in diameter with walls 1.2 mm. thick, which were immersed in a continuously agitated water bath for 10 minutes at 49°, 51°, 52°, and 53° C., respectively. After these treatments, inoculations were made from each source to 10 healthy plants of the sugarcane variety Louisiana Striped by pricking through a drop of inoculum in the spindle with a fine needle (Matz 1933). The results in table 3 are from two repetitions of the tests.

TABLE 3.—*Dilution end point (DEP) and thermal death point (TDP) of 3 strains and 8 variants of sugarcane mosaic virus, based on inoculations to Louisiana Striped plants* [1]

Virus strain	Dilution 1-10	Dilution 1-100	Dilution 1-1,000	49° C.	51° C.	52° C.	53° C.
A	+	+	+	+	+	+	−
D	+	+	+	+	+	+	+
Variants of D:							
M-366	+	+	−	+	−	−	−
M-367	+	−	+	+	−	+	−
M-368	+	+	+	−	−	+	−
M-369	+	+	+	+	+	−	−
M-370	+	+	+	+	+	−	−
M-372	+	+	+	+	+	−	+
H	+	+	−	+	−	−	−
Variants of H:							
M-401	+	+	−	+	−	−	−
M-402	+	+	+	+	−	−	−

[1] + = infection; − = inactivation.

All variants of strain D except M-366 were similar to this strain in DEP, but all had lower TDP's except M-372. One of the strain H variants (M-402) was similar to the type strain in TDP, but showed higher DEP.

The authors agree with Bennett (1953) that differentiation of strains on the basis of symptoms and physical properties is definitely limited. Differences in DEP and TDP of SMV were not correlated with those in symptom expression on differential hosts. Study of serological relationships was not feasible, although they might be of value in strain differentiation. Desai (1935) and Perez and Adsuar (1954) found that

SMV is antigenic, and the latter authors suggested the possibility of using the precipitin reaction in testing for relationships. Apparently, however, this line of investigation has not been pursued.

Since the TDP of SMV in vitro is lower than the temperature required for elimination of ratoon stunting disease (RSD) virus from sugarcane seedcane stalks (Schexnayder 1956), it might be assumed that the long hot-air treatment for control of RSD (8 hours with inlet temperature 58° C.) would also eliminate SMV from seedcane. However, in several experiments with the commercial long hot-air treatment of mosaic-infected sugarcane stalks, mosaic was present in the plants arising from the treated buds. SMV is not eliminated by temperatures up to those that are lethal to cane buds.

Differential Host Varieties

Summers (1939) and Summers, Brandes, and Rands (1948) based their identification of strains on the symptoms produced on the sugarcane varieties Co. 281, C.P. 29-291, and C.P. 31-294. Co. 281 differentiates strain C, which is the only strain that produces severe chlorosis and necrosis on that variety. However, strain C produces similarly severe symptoms on all other varieties on which it has been observed, and can usually be identified in the field without transfer to differential hosts. Since strain C is rare and Co. 281 does not differentiate other strains, this variety was not included as a differential host in the routine identification of field collections in the present study.

Summers (1939) used C.P. 29-291 to differentiate strains B and G, the latter including viruses with strain B leaf symptoms but which cause only slight or no growth retardation of C.P. 29-291. The authors consider strain G a variant of strain B. C.P. 29-291 does not differentiate other described strains.

C.P. 31-294 differentiates strains A, B, D, E, and F, and C.P. 31-588 differentiates strains A and H.

Description of Strains

Four strains of SMV—A, B, D, and H—were studied in some detail in the current investigation. These strains were the only ones identified from commercial fields in the surveys made in Louisiana since 1950. Strain C, which was collected rarely in the 1930's and early 1940's, has not been identified since 1944. The last identification of strain E in Louisiana (from the sirup-producing area of northern Louisiana) was made in 1943 and from the sirup-producing areas in the other Gulf States in 1948. The only identification of strain F that has been made was the type collection in 1935 (Summers, Brandes, and Rands 1948).

Strain A produces on C.P. 31-294 a coarse pattern of irregular, mild mottling, moderate stunting of growth, and no necrosis (Summers, Brandes, and Rands 1948). On C.P. 31-588 the pattern of mottling is similar, but on this variety strain A produces numerous short stripes and flecks, which are narrow, discrete, and pale chlorotic to yellowish white (fig. 1). They differentiate strain A from strain H.

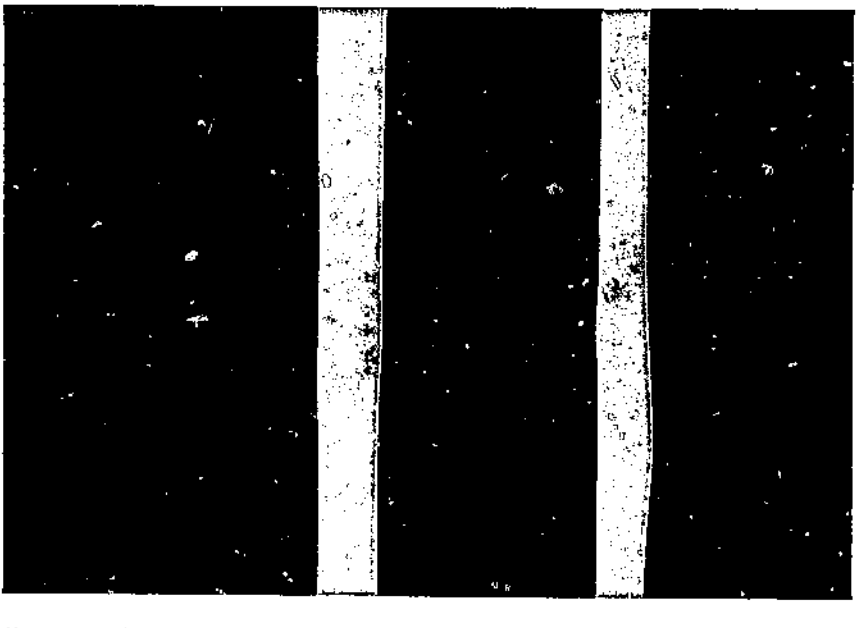

FIGURE 1.—Differential host variety C.P. 31-588 with symptoms of strain A (left) and strain H (right) as compared with healthy leaf (center).

Strain B produces on C.P. 31-294 a pattern of severely chlorotic stripes that characteristically coalesce or diffuse to give a bleached effect, particularly toward the base of the leaves, and necrosis is always present, varying from slight to severe. Most accessions identified as strain B cause marked stunting of growth of the differential host varieties C.P. 31-294, C.P. 31-586 (see table 12, U.S. Dept. Agr. Tech. Bul. 955), and C.P. 31-588, but the degree of retardation varies considerably.

Strain C produces approximately the same symptoms on all varieties on which it has been observed. They first appear as isolated, elongated, yellowish-white blotches that coalesce into long streaks, often extending the full length of the leaf. The streaks may follow the midrib and are usually accompanied by necrosis, which is 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 occurs, and excessive suckering is a common aftermath. Strain C has a low index of infection. Its symptoms are indistinguishable from those of strain D on C.P. 31-294, but strain C is differentiated from other strains by the severe chlorosis and necrosis produced on Co. 281.

Symptoms of strain D on C.P. 31-294 begin as elongate, severely chlorotic to nearly white, discrete streaks that often extend the full length of the leaf (fig. 2). Necrosis usually appears early, and although it varies in degree with different accessions, it is characteristically severe, often giving the young plants a reddened appearance. Stunting is marked and growing points of primary and secondary shoots are often killed, frequently followed by excessive tillering. The range of severity

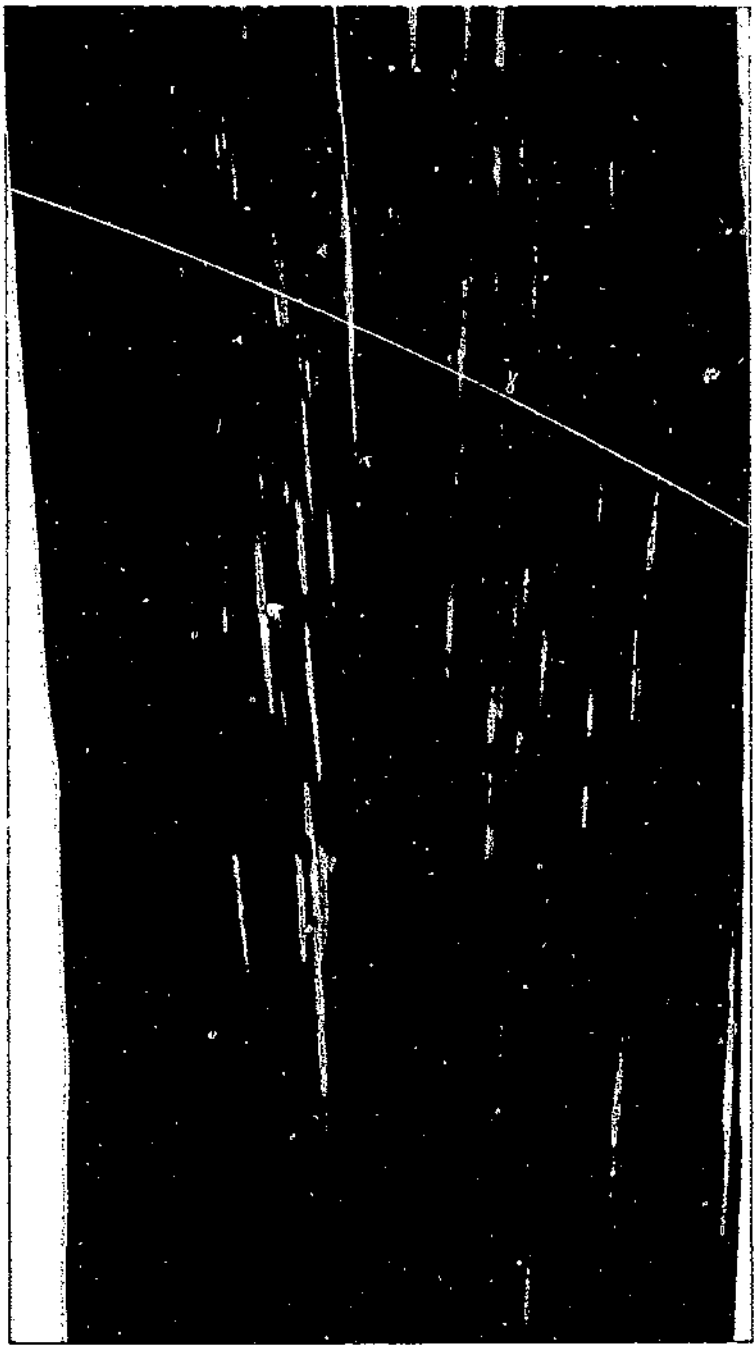

FIGURE 2.—Symptoms of strain D on differential host variety C.P. 31-294.

of symptoms produced by viruses classed as strain D is greater than for other strains studied and numerous variants are encountered. However, since the differences are of degree rather than kind, the authors prefer to consider them variants of D rather than different strains.

On commercial varieties, such as C.P. 44-101 and N. Co. 310, strain D often causes a somewhat more chlorotic mosaic pattern than other strains, which might be described as "severe." This strain has a lower index of infection than strain A or B (table 2).

Strain E, as described by Summers, Brandes, and Rands (1948), produces on C.P. 31-294 a few elongate, discrete, severely chlorotic stripes, with characteristically pronounced reddening of adjacent tissues. Strain E causes little or no growth retardation of this variety.

Strain F is differentiated from strain A on C.P. 31-294 by extremely finely divided mosaic mottling with prominent green islands (Summers, Brandes, and Rands 1948). Like strains A and H, it produces no necrosis on any variety on which it has been studied.

Strain H is differentiated from strain A on C.P. 31-588 by the diffuse mild mottling it produces in contrast to the bold or coarse mottling of strain A and by the absence of the chlorotic stripes and flecks that characterize strain A on this variety (fig. 1). It has a long incubation period. No infection of C.P. 31-294 was obtained with most of the viruses identified as strain H, but others (about 20 percent) infected it readily. This variability among viruses assigned to strain H was not correlated with their source as to variety or locality.

Viruses classed as strain H that infected C.P. 31-294 produced a faint pattern of mild mottling, which often disappeared. There was marked stunting of growth, frequent killing of growing points, and subsequent excessive tillering.

McKinney and Greeley (1960) defined the term "strain" as an "isolate, the characteristics of which remain essentially constant in series transfers." The strains described in this bulletin fall within that concept.

According to Bennett (1953), "most of the plant viruses that have been studied extensively have been found to exist in nature as complexes of related strains or variants." Bawden (1950, p. 107) said that "so many strains of some individual viruses have been identified that no attempt can be made to describe them in detail." Bennett (1960) concluded that the sugarbeet yellows virus consists of variants "so numerous that efforts to isolate and describe all of them would be impractical." SMV shows similar variation.

Numerous variants were encountered in the present study that differed from the described strains in some respect. By utilizing additional differential host varieties, it is probable that other variants could be described as strains, including some that have been grouped within the limits of strains B, D, and H. However, unless minor differences in symptoms on differential host varieties can be related to differences in virulence or to susceptibility of commercial or breeding varieties of sugarcane to mosaic, there seems to be little practical value in unlimited description of strains.

It is not to be assumed that the seven strains described represent the limits of variation of SMV. Because of the large number of infectious units involved, it is impossible to presume that any given isolate represents a pure culture of one strain or that all isolates referred to one strain are of equal virulence. Nevertheless, the evidence does show that

distinct SMV strains exist in nature and some, at least, as sufficiently constant entities as to be identifiable over a long period of time. Isolates that are easily identified as strains A, B, and D, as originally described by Summers nearly 30 years ago, have been collected repeatedly over the years and are still present in Louisiana sugarcane fields. A practical reason for differentiating strains of SMV is that those described have been shown to be associated with the occurrence of mosaic in commercial sugarcane varieties in Louisiana.

STABILITY OF STRAINS

Effect of Host Variety

The SMV strains studied in these investigations have remained stable in stock culture on the varieties P.O.J. 234 and Louisiana Purple, but on some other varieties changes from the characteristic symptoms of one strain to those of another have been noted, generally from a severe to a milder pattern. This is particularly true of strain D, which, as noted by Summers, Brandes, and Rands (1948), has many variants and "has shown a marked tendency to break up or change when continuously cultured on certain C.P. varieties." They noted that on the varieties C.P. 28-60 or C.P. 31-294 strain D often showed a gradual change of symptoms from the severe pattern of that strain to the mild mottling of strain A. They stated that "the type A symptoms appeared first as an admixture, both patterns being mingled on the same leaf, and then later the type D symptoms failed entirely. Subinoculations from such plants would usually produce the mild type of symptom." This was confirmed by Abbott (1952).

Strain D has been carried in stock culture in the greenhouse at the U.S. Sugarcane Field Station, Houma, La., for more than 20 years on the variety P.O.J. 234, which does not differentiate the symptoms of the described strains, and when inoculated to healthy C.P. 31-294, typical strain D symptoms were produced. This showed that this strain remained stable on P.O.J. 234. After 3 to 6 months on C.P. 31-294, however, a gradual change to the mild symptoms of strain A occurred in some of the plants that were observed. The discrete, chlorotic stripes characteristic of strain D became less numerous on new leaves and were replaced by the mild mottling of strain A.

In one series of 12 plants of C.P. 31-294 that were inoculated with strain D in July 1959, 5 plants still exhibited typical symptoms of strain D in September 1961, 3 had only strain A symptoms, and 3 showed mild mottling in the younger foliage with a few scattered discrete lesions of strain D on the older leaves. One plant had only mild mottling on all the leaves, but the tillers showed the marked stunting and death of growing points, which is characteristic of strain D. Inoculation to healthy C.P. 31-294 by Sein's (1930) method from plants showing either partial or complete change to strain A symptoms produced only strain A symptoms. TDP and DEP were also the same as those of strain A.

Change from severe to milder symptoms has been observed also with strain C. The principal diagnostic character of this strain is the severe pattern of chlorotic stripes with considerable necrosis produced on leaves of Co. 281. A stock culture of this strain on Co. 281 has been maintained in the greenhouse since 1934. Diminution of the severity of

the characteristic necrosis was first noted in 1950. In successive generations propagated vegetatively from the infected plants there was continued gradual loss of necrosis, although the chlorotic stripes remained.

On October 27, 1959, juice from a plant of Co. 281 showing this change was inoculated to healthy C.P. 31-294. Scattered, discrete, severely chlorotic and necrotic lesions were produced as well as stunting—typical symptoms of strain C and also of strain D on this variety. Two plants were retained for observation. One continued to show typical strain C symptoms until July 1960, when 10 of 12 shoots began to show mild strain A symptoms in the new foliage. By late October all shoots had developed strain A symptoms. A similar change occurred in the second plant, although not until a month later. Both were considerably stunted. On December 17, 1960, inoculations were made from each plant to 10 plants of the differential host variety C.P. 31-588, all of which developed strain A symptoms. In this instance strain C virus, which had become attenuated on Co. 281, apparently underwent further change to the mild symptoms of strain A on C.P. 31-294.

Strain F has been carried in stock culture in the greenhouse at Houma on Louisiana Purple by vegetative propagation since it was identified in 1935. When transferred to C.P. 31-294 from time to time, typical symptoms of strain F were produced. Strain F is characterized by very mild, finely divided mottling, very diffuse and sometimes nearly masked, in contrast to the bold or coarse mottling of strain A. Neither produces necrosis on any variety on which they have been studied. A change from one symptom pattern to the other, therefore, would be somewhat less obvious than that observed with strains C and D, which are characterized by severe chlorosis and necrosis.

In 1959 strain F was studied to determine whether it would remain stable if cultured on C.P. 31-294. Inoculations were made on April 14, 1959, and typical strain F symptoms were produced. Two plants were held for observation.

By December the symptoms on new foliage of the two plants of C.P. 31-294 inoculated with strain F showed no essential difference from those of plants infected with strain A. On December 17 inoculations were performed to C.P. 31-588 from each of the two plants and from the original stock culture of strain F on Louisiana Purple. Typical symptoms of strain F were produced by the latter source of virus, but from the two plants of C.P. 31-294, strain A symptoms were produced on C.P. 31-588.

To determine whether the observed changes in symptom expression were associated with changes in physical properties of the virus, DEP and TDP of the different virus sources were determined as described on page 7. Strain F has lower infectiousness, DEP, and TDP than strain A. Juice was extracted from strain A on C.P. 31-294 and P.O.J. 234, from one plant of strain F that had undergone a symptom change on C.P. 31-294, and one plant each of the stock cultures of strain F on P.O.J. 234 and Louisiana Purple. After the treatments, inoculations were made from each source to 10 healthy plants of Louisiana Striped.

Strain F on C.P. 31-294, which had changed to strain A symptoms, behaved similarly to the stock culture of strain A on P.O.J. 234 in high infectiousness and in reaction to dilution and heat, whereas the stock cultures of strain F on P.O.J. 234 and on Louisiana Purple showed the typical properties of that strain—low infectiousness and low tolerance

TABLE 4. *Infection of Louisiana Striped plants among 10 inoculated with 5 sources of virus after dilution and heat treatment*

Strain source	Undiluted	Plants infected after Dilution at 1–10	1–100	1–1,000	Heat treatment at 45° C.	47° C.	49° C.
	Number	*Number*	*Number*	*Number*	*Number*	*Number*	*Number*
A on C.P. 31-294	7	3	3	0	3	2	1
A on P.O.J. 234	10	5	2	0	3	1	5
F on C.P. 31-294	10	5	2	0	4	2	0
F on P.O.J. 234	1	0	0	0	0	0	0
F on Louisiana Purple	1	0	0	0	0	0	0

to dilution and heat, as indicated in table 4. This is evidence that strain F changed to strain A on C.P. 31-294, but remained stable on P.O.J. 234 and Louisiana Purple.

Several host-induced changes in plant viruses described in the literature were reviewed by Hitchborn and Thomson (1960). Carsner and Stahl (1924) found that by passing curly top virus through *Chenopodium murale* L., it was so attenuated as either to fail to produce curly top disease when inoculated to healthy beets or to produce only mild symptoms. Carsner (1925) further confirmed this and observed similar attenuation by passage of the virus through *Rumex crispus* L. and *Suaeda moquini* A. Nels. *S. torreyana* S. Wats. Lackey (1932) stated that the attenuated virus could be restored to approximately its original virulence by passage through *Stellaria media* (L.) Cyrillo. Giddings (1940) interpreted these results as due to reisolation of mild and severe strains of the virus that had occurred originally as mixtures.

Johnson (1947) found that a culture of tobacco mosaic virus (TMV) that produced severe symptoms in tobacco caused mild symptoms when inoculated to sea holly (*Eryngium aquaticum* L.). The sea holly produced this change by separating a mild strain from a severe one when the two strains existed in combination. Salaman (1939) described a change in type of symptoms produced by potato virus X from severe to mild after growing in sugarbeet or red beet.

Bawden (1956) found that a cowpea strain of TMV when purified from French bean and tobacco differed in symptoms and physicochemical properties from the original virus. He suggested that the host-induced changes could be due to (1) the presence of both forms in the inocula and selection by bean and tobacco of the respective forms, (2) the selection of randomly occurring mutants, (3) the selection of host-induced mutants, or (4) the production of phenotypic variants. The explanation of the observed changes in SMV probably lies in one of these hypotheses, but proof is lacking on the exact nature of the phenomenon. If some sugarcane varieties do induce mutation of mosaic virus strains, as is indicated by the observations on C.P. 31-294, this

would offer one possible explanation for the appearance of new strains.

In speculating on the possible origin of SMV type 3 (strain C), a strain of unusual virulence, Brandes (1950) said "it is possible that the protective agencies of a refractive host plant might change the virus in the direction of reduced virulence or conversely, passage through a specially susceptible plant might have the opposite effect." However, he found no evidence of change in the virus from passage through maize.

Effect of Temperature

Since observations on the change of symptoms from severe (strain D) to mild (strain A) were all made on plants grown in the greenhouse, where daytime temperatures may reach as high as 49° C., it seemed possible that these changes might have been induced by the high temperatures. R. D. Rands and Ernest Dopp (unpublished data) grew 10 plants of C.P. 28-60 infected with strain D in a temperature tank maintained at 46°. After 6 months no changes in symptoms were noted. Johnson (1947) induced attenuation of TMV by incubation of plants at 35° - 37° for 5-13 days and also produced entirely new strains by this treatment.

To study the effect of temperature on symptom expression and strain stability of strain D, 40 plants of healthy C.P. 31-294 were inoculated with this strain from the variety P.O.J. 234 on July 6, 1959. They were kept in the greenhouse until July 29, when mosaic symptoms developed. Notes were made on the severity of symptoms in each plant. Twenty plants were then moved to a room equipped with batteries of fluorescent Mazda[2] lights, where the temperature was maintained at 25° C. ± 1°. The remaining 20 plants were kept in the greenhouse, where daily readings were made of the maximum and minimum temperatures during the 2½ months' duration of the experiment. The ranges in temperatures are given in table 5.

TABLE 5.—*Maximum and minimum temperatures in greenhouse where 20 plants of C.P. 31-294, inoculated and infected with strain D from variety P.O.J. 234, were kept for 2½ months*

Month	Maximum		Minimum		Average
	Range	Average	Range	Average	Average
	°C.	°C.	°C.	°C.	°C.
August	34.5-46.5	42.5	21.2-29.9	25.0	33.5
September	33.5-46.0	40.5	19.5-23.5	22.2	31.0
October (1-15)	27.7-41.0	37.8	22.2-24.5	23.5	30.5

At 14-day intervals the symptom pattern on all plants was recorded. No significant changes were observed. Since it was necessary to discontinue the use of the low-temperature room, the plants were returned to

[2] Trade names are used in this publication solely for the purpose of providing specific information. Mention of a trade name does not constitute a guarantee or warranty of the product by the U.S. Department of Agriculture or an endorsement by the Department over other products not mentioned.

the greenhouse on October 15 with the other group. Subsequently several plants showed the commonly observed changes from strain D to strain A symptoms, but they occurred among those held at both low and high temperatures. In this experiment temperature had no apparent effect on symptom expression or strain stability.

CROSS PROTECTION EXPERIMENTS

According to Bennett (1960), "In general, plants infected and completely invaded by one strain of a virus are immune or highly resistant to a second strain of the same virus." However, he obtained no evidence that one strain of sugarbeet yellows virus protects against infection or injury by a second strain, and he (1955) found that strains of the curly top virus in water pimpernel did not protect against infection or injury by other strains of the virus. Giddings (1950) showed that strains of curly top virus do not protect against either infection or injury by other strains of the virus. Forbes (1938) concluded from cross protection experiments with what he termed the "green" and "yellow" strains of SMV that "the evidence is conclusive that they do not occur together in a plant." He was unable to recover either strain after inoculating it to a plant already infected with the other. However, plants of Louisiana Purple infected with a very mild mosaic readily became infected when inoculated with a severe strain occurring on that variety.

Siegel (1959) inoculated two strains of TMV to *Nicotiana sylvestris* Speg. & Comes and concluded that the two were mutually exclusive. However, Wu and Rappaport (1961), who inoculated the same two strains to *Phaseolus vulgaris* L., concluded that when both entered the same cell neither multiplied. Benda (1956) inoculated single hair cells of *N. glutinosa* L. with a mixture of the ordinary and the yellow aucuba strains of TMV and recovered both from five of the lesions produced. Bawden (1964) stated, "There seems no reason why inoculation with a mixture of two strains should not introduce both into the same cell" and observed that Benda's results "leave little doubt that, when two strains are introduced simultaneously into the same cell, both can infect."

When differential host varieties are inoculated with virus from mosaic-infected plants collected from sugarcane fields in making strain surveys, symptoms of more than one strain of the virus are sometimes obtained from the inoculum from a single plant. Summers, Brandes, and Rands (1948) reported 26 mixtures in a total of 278 collections studied. Several mixtures were found in the present investigations.

R. D. Rands and Ernest Dopp (unpublished data) performed numerous experiments in which strain mixtures were inoculated to healthy Co. 281, which differentiates strain C, and C.P. 28-60, which differentiates strains A, B, and D. They included the following single strains and strain mixtures in one series of experiments: A alone, A plus B, A plus C, A plus D, B alone, B plus C, B plus D, C alone, C plus D, and D alone.

The A plus C mixture on Co. 281 gave mixed patterns in two plants, and there was less stunting than when strain C was used alone. When juice from plants with mixed patterns was inoculated to healthy Co. 281, some plants developed strain A symptoms only and some strain C symptoms only. They observed that with mixtures involving strain A, that strain tended to predominate, either by gaining a foothold in the plant ahead of the other strains or by gradually suppressing the other

strains in an initially mixed pattern. Rands and Dopp concluded that two SMV strains can exist in the same plant.

Experiments were undertaken in 1960 as a continuation of the work initiated by Rands and Dopp to determine the extent to which more than one strain can be established in a single plant when (1) two strains are introduced together and (2) one strain is established first and another subsequently superimposed by inoculation. The following strain combinations were inoculated to healthy plants of P.O.J. 234 and Louisiana Striped: (1) Mixture of two strains: A and B, A and D, A and H, B and D, B and H, D and H; (2) strain A established as the initial strain, with strains B, D, and H later superimposed separately; (3) strain D established initially, with strains A and H later superimposed separately; (4) strain H established initially, with strains A, B, and D later superimposed separately.

Five plants of each of the host varieties P.O.J. 234 and Louisiana Striped were inoculated with each of these strain combinations. Plants of P.O.J. 234 infected with the respective strains were the source of inoculum. For the strain mixtures, equal quantities of infective juice of each strain were used. Juice was extracted by passing the leaves through a food grinder and straining the juice through cheesecloth. The initial inoculations were made on January 13, 1960. After mosaic symptoms had developed from the first inoculations, the strains to be superimposed were inoculated to the same plants, some on January 27 and others on February 3. From these plants, three different series of inoculations were made to differential host varieties to determine which strains could be recovered. C.P. 31-294 was used as the differential host for all strain combinations except those involving strain H, for which C.P. 31-588 was used. The first series of inoculations to differential hosts was made on February 15, the second on October 14, and the third on November 3, 1960. In some instances 10 and in others 5 healthy plants of the differential host varieties were inoculated from each plant furnishing the source of virus. The results are in tables 6 and 7.

TABLE 6. *Infection of sugarcane plants with 2 strains of mosaic virus by inoculation with strain mixtures*

Strains in mixture	Plants as sources of inoculum	Plants infected with indicated strains identified from inoculated differential hosts				
		Only A	Only B	Only D	Only H	Both components of mixture
	Number	*Number*	*Number*	*Number*	*Number*	*Number*
A and B	7	3	1	0	0	[1]3
A and D	6	0	0	3	0	[2]3
A and H	8	8	0	0	0	0
B and D	6	0	2	4	0	0
B and H	6	0	6	0	0	0
D and H	1	0	0	1	0	0

[1] Plants showed strain B symptoms at first, but strain A symptoms later dominated.
[2] Plants showed strain D symptoms at first, but strain A symptoms later dominated.

When two strains were introduced as a mixture, each was recovered separately in different plants, except in combinations with strain H,

TABLE 7. *Infection of sugarcane plants with 2 strains of mosaic virus by superimposing one strain on another already established*

Initial strain	Superimposed strain	Plants as sources of inoculum	Plants infected with strains identified from inoculated differential hosts		
			Initial strain only	Superimposed strain only	Both strains
		Number	*Number*	*Number*	*Number*
A	B	6	0	0	0
A	D	7	6	0	1
A	H	4	4	0	0
D	A	8	7	0	1
D	H	2	2	0	0
H	A	4	1	0	3
H	B	5	1	1	3
H	D	4	0	2	2

which was not recovered from any of the 15 plants tested. When strain A was introduced alone as the initial strain, neither strain B nor strain H was recovered from plants in which they were introduced as the superimposed strains, but strain D was recovered from one of the seven plants in which it was superimposed on strain A. Similarly, when strain D was introduced as the initial strain, strain A was recovered from one plant in which it was superimposed on strain D. When strain H was introduced as the initial strain, however, strains A, B, and D as the superimposed strains each became established and they were recovered from more of the plants tested than was the initial strain. The results show that two strains of SMV can be established in a single sugarcane plant when introduced separately, and that strains producing mild symptoms may occur in the same plant with those producing severe symptoms (A and D, H and B, H and D).

RELATIONSHIP OF STRAINS TO SUSCEPTIBILITY OF SUGARCANE VARIETIES TO MOSAIC

Association of strains of the virus with different sugarcane varieties has been a factor in the occurrence of mosaic in commercial fields in Louisiana since the disease was first recognized in the State. Summers (1939) concluded from his investigations that it was "possible to reconstruct a plausible picture of a succession of strains of sugarcane mosaic virus" in Louisiana and other Southern States. "Strain E was apparently responsible for the original mosaic that spread over Louisiana after 1919" and was so destructive to the noble canes in the early 1920's. In the later 1920's, strain D became generally distributed over the entire cane area with the distribution and general planting of the P.O.J. varieties 36, 36-M, 213, and 234. Although mosaic-free cuttings of these varieties were taken to Louisiana from Washington, D.C., in 1922 and 1923, they soon became 100-percent mosaic-infected and were so distributed throughout the sugar district.

Summers, Brandes, and Rands (1948) expressed doubt that the P.O.J. varieties would have become completely infected with strain E because

of its low infectiousness. They noted that general and fairly complete recovery from mosaic occurred in P.O.J. 213 between 1925 and 1930, but it became reinfected with strain B, which was involved in a new wave of spread, which began in 1931 after the release of Co. 281 in 1930. This variety and Co. 290, which was released in 1933, are susceptible to both strains B and D, which predominated almost to the exclusion of other strains while the Co. varieties were grown commercially (1931–50) (Abbott 1958). Strain B was the more common of the two strains on Co. 290 after Co. 281 declined in acreage.

In addition to affecting the prevalence of mosaic in commercial sugarcane varieties, Summers (1943) found that strains have a differential effect on crop losses. He determined yield reductions caused by strains A, B, D, and F on two commercial varieties in Louisiana, Co. 281 and Co. 290. He found that strain D caused much greater reduction in yield of sugar per acre of Co. 281 than the other strains, but with Co. 290, differences between strains were less marked.

The relative prevalence of strains identified in surveys made in Louisiana from 1942 through 1955, preceding the identification of strain H, and from 1956 to 1961, following its identification, is shown in table 8.

The first identification of strain A in a commercial field was made in 1943 (Abbott 1958), and it was subsequently identified from widely scattered areas on several varieties. However, with destruction of the more heavily infected fields and use of mosaic-free seedcane for new plantings, no general outbreak of mosaic resulted from the appearance of this strain.

Mosaic was of little concern to Louisiana cane growers from the late 1940's to 1955 because of the small acreage planted to varieties (principally Co. 290) that were susceptible to the strains then prevalent, but in 1956 a new strain, designated H (Abbott 1961), was identified, to which the principal commercial variety C.P. 44-101 is susceptible. Strain H has predominated in surveys conducted since 1956 (Abbott 1960) (table 8).

The rapid spread of strain H in C.P. 44-101 and in the varieties N. Co. 310 and C.P. 52-68, released in 1954 and 1958, respectively, necessitated return to roguing of seedcane for control, which had not been practiced on any large scale for many years. Two released varieties that are resistant to strain H—C.P. 36-13 and C.P. 47-193—have defects that prevent them from being acceptable as major canes. Although roguing them for mosaic control usually has not been required, they constituted only 6.3 percent of the 1963 sugarcane acreage in Louisiana (Hebert 1963).

The eventual solution to control of strain H lies in the production of generally adapted resistant varieties. However, a discouraging discovery from the standpoint of breeding for resistance was that the wild canes, used as mosaic-resistant parents in producing C.P. varieties for the Louisiana industry since 1942, are susceptible to this strain. Abbott and Todd (1962) found that the Indian and Javan forms of *Saccharum spontaneum* L. and the natural hybrid Kassoer, which were the sources of mosaic resistance in the ancestry of the C.P. canes (Brandes and Sartoris 1936, Sartoris 1943–47), are susceptible to strain H. Hitherto, these clones had been considered immune to mosaic (Brandes and Sartoris 1936, Brandes, Sartoris, and Grassl 1938, Jeswiet 1929). Abbott and Todd (1962) concluded that breeding for resistance to strains of mosaic now present in commercial sugarcane areas in the

TABLE 8.—*Prevalence of sugarcane mosaic strains identified from various sugarcane varieties in Louisiana, 1942-61*

Variety	Number of plants in 1942-55 infected with						Number of plants in 1956-61 infected with					
	Strain A	Strain B	Strain C	Strain D	Unidentified	Total	Strain A	Strain B	Strain D	Strain H	Unidentified	Total
Co. 290	1	37	0	5	2	15	0	0	2	1	0	3
C.P. 34-120	1	5	0	1	0	10	0	0	0	0	0	0
C.P. 36-105	3	2	0	1	0	6	0	0	0	3	2	5
C.P. 44-101	1	0	0	0	3	4	5	1	1	39	5	54
C.P. 52-68	0	0	0	0	0	0	0	1	0	22	0	23
N. Co. 310	0	0	0	0	0	0	1	5	3	39	11	59
Others[1]	27	18	1	5	6	57	3	6	0	21	7	40
Total	36	62	1	12	11	122	9	13	9	128	25	184
Percent of total	29	51	1	10	9		5	7	5	70	13	

[1] Minor commercial and unreleased varieties.

United States will be a more difficult problem in the future than in the past.

In the sirup-producing areas of Alabama, Florida, Georgia, and Mississippi, strain B predominated in the surveys made from 1941 to 1956, as shown in table 9. This was because of the prevalence of that strain on Co. 290, which became the leading variety during that period. Strain E, which had predominated in the earlier survey (Summers, Brandes, and Rands 1948), became less prevalent with the decline in acreage of Louisiana Purple and P.O.J. 213.

TABLE 9.—*Prevalence of sugarcane mosaic strains identified from various sugarcane varieties in sirup-producing areas of Alabama, Florida, Georgia, and Mississippi, 1941–56*

State	Number of plants infected with				Total
	Strain A	Strain B	Strain D	Strain E	
Alabama	1	10	2	1	14
Florida	1	3	1	1	6
Georgia	0	5	1	0	6
Mississippi	2	24	12	4	42
Total	1	42	16	6	68
Percent of total	5.9	61.8	23.5	8.8	

[1] Includes 3 that caused slight growth retardation of C.P. 29-291, and could be classed as Summers' strain G.

OTHER HOSTS

Summers, Brandes, and Rands (1948) listed 10 cultivated and 5 wild-grass hosts of sugarcane mosaic, but did not mention their susceptibility to individual strains of the virus. However, their listing of johnsongrass (*Sorghum halepensis* (L.) Pers.) as a host, based on Kunkel's (1924) observation, is apparently an error since Kunkel's paper contains no reference to the occurrence of mosaic on johnsongrass. Anzalone (1963, a and b) infected five varieties of rice with strain H of SMV. Abbott and Tippett (1964) infected varieties of wheat, barley, rye, and rice with several strains of SMV. Wheat, barley, and rye are seldom if ever grown in the same area as sugarcane, but rice is often grown contiguous to or in the vicinity of sugarcane. Brandes (1919) reported one instance of naturally infected rice adjacent to heavily infected sugarcane in Puerto Rico.

Abbott and Tippett (1964) infected johnsongrass with strain H, but they concluded that, although this grass may be important economically in disseminating mosaic by serving as a preferred host of the vector *Rhopalosiphum maidis* (Fitch), there is no evidence that it serves as a secondary host of SMV in nature. However, in further work the authors have found that johnsongrass plants that do not develop mosaic symptoms following inoculation with strain H may actually carry the virus, since inoculation of sugarcane plants with juice of inoculated but symptomless plants of johnsongrass produced mosaic symptoms in the inoculated sugarcane. Inoculum prepared from johnsongrass

growing in sugarcane fields heavily infected with mosaic did not produce mosaic symptoms when inoculated to susceptible varieties in the greenhouse (unpublished data).

Todd (1964) observed mosaic symptoms on St. Augustine grass (*Stenotaphrum secundatum* (Walt.) Kuntze) in Florida. When the virus was transferred to differential host varieties of sugarcane, it proved to be an undescribed strain of SMV. Abbott and Tippett (1964) infected this grass with four strains of SMV.

Lawas and Fernandez (1949) assumed that the mosaic they observed on *Rottboellia exaltata* L. in the Philippines was probably caused by SMV, but they did not report any attempt to transfer the virus to sugarcane. Abbott and Tippett (1964) inoculated this grass with three strains of SMV, but did not obtain infection.

SUMMARY

Variations in the symptom pattern of mosaic on sugarcane varieties were noted before the existence of strains of the virus was known. Differentiation of strains in Louisiana was based on the nature of the chlorosis, presence or absence of necrosis, leaf sheath discoloration, growth retardation, germination recovery, relative infectiousness or "index of infection," and length of incubation period on selected differential hosts.

The nature of the mosaic pattern on differential hosts, whether mild or severe chlorosis, is the principal diagnostic character. Presence or absence of necrosis is important and critically diagnostic. Leaf sheath discoloration is inconstant and unreliable. Growth retardation is not a highly critical character for separating strains, but may be used in conjunction with differences in symptom pattern. Germination recovery is a variable character and not reliable. Marked differences in relative infectiousness exist among strains of SMV, but this character is not critically diagnostic. Differences in length of incubation period also occur, but this character alone is not critically diagnostic.

Thermal death point and dilution end point are of limited value in differentiating SMV strains.

Symptoms of strains A, B, C, D, E, F, and H on differential hosts are described. Variability of SMV is discussed, and the conclusion is reached that attempting to describe all variants of the virus would have no practical value.

Strains C, D, and F were unstable on the differential host variety C.P. 31-294. Some plants infected with each of these strains underwent a gradual change from their typical symptoms to those of strain A.

In cross protection experiments, six mixtures of two strains each were inoculated to two sugarcane varieties to determine whether more than one strain could be established in the same plant. Each strain was recovered separately in different plants, except in combinations with strain H, which was not recovered from any of the plants tested. Some plants inoculated with combinations of strains A and B, and A and D, initially showed symptoms of both components, but strain A symptoms later dominated. When one strain was established initially and another superimposed by later inoculation, both strains were recovered from some combinations but not others. The results showed that two strains of the virus can be established in a single sugarcane plant when introduced separately.

Mosaic strains have been shown to be associated with the occurrence of mosaic in commercial varieties of sugarcane in Louisiana since the existence of strains was first recognized. The relative prevalence of strains in the Southern United States from 1942 to 1961 was studied. Since 1956 a new strain, H, has predominated in Louisiana, affecting varieties hitherto considered highly resistant to mosaic. This strain also infects breeding stocks that are highly resistant or immune to other strains and thus complicates the problem of breeding for mosaic resistance.

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