Virus Diseases of Plants and Their Insect Vectors with Special Reference to Hawaii

By D. D. JENSEN
University of Hawaii Agricultural Experiment Station

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This subject will be divided into the following three general parts:

Part I. Principles of insect transmission of plant viruses.

Part II. Insect species occurring in Hawaii known to transmit plant viruses, with a list of viruses transmitted by each.

Part III. Plant virus diseases known or reported to occur in Hawaii with special reference to their transmission by insects.

Introduction

The entomological and pathological implications of the war are far-reaching and it will probably be several years before their full significance for Hawaii can be evaluated. However, the handwriting is already on the wall. Despite the vigilance of our quarantine inspectors and the cooperation of the Army and Navy it is already apparent that undesirable insect species which were previously absent from Hawaii are being introduced. The exact manner in which recent insect arrivals reached the Territory has not been determined. Until recent years most of them were brought by boat. However, the heavy plane traffic through Hawaii during and since the war provides a means of insect entry which is a more serious hazard than that attending the slower water craft.

Several immigrant species have been discovered in Hawaii during the past two years. Because of their conspicuousness and the rapidity with which they have become abundant, some of these can be considered very recent arrivals—possibly by plane.

Quarantine inspectors in Hawaii have intercepted a number of living insects in planes which arrived from the mainland or from other Pacific areas. Due to the short period of time required by modern planes to travel thousands of miles, even delicate forms, such as aphids and leafhoppers, which cannot survive many hours away from suitable hosts, may reach Hawaii alive. Most of these insects are killed by spraying before they can escape from the planes. It is probable, however, that during the early part of the war before treatment of military planes had become a well established practice, or as a result of crash landings or other emergencies,

[1] Because of the length of the paper, the actual address as delivered was confined to Part I and representative portions of Parts II and III.

insects reached Hawaii by planes from which they escaped alive. Furthermore, the most rigid quarantine system that can be put into practical service cannot hope to intercept all insects which reach Hawaii. This service is, however, successful in reducing the introductions to a fraction of what they would be if no regulations were in effect.

This problem has a direct bearing on the subject I have chosen for discussion today, because the hazard of introducing new virus diseases by means of viruliferous insects, or of introducing more efficient vectors of viruses already established, is greatly increased by aircraft, particularly under conditions imposed by war.

This discussion will be confined primarily to the entomological aspects of virus problems. Furthermore I shall not undertake a treatment of all known plant viruses or their vectors but shall deal in particular with the viruses and vectors known to occur in Hawaii. In addition, certain viruses not known to occur in Hawaii will be considered briefly because of the bearing they have on the virus problems of the Territory.

A review of the literature on virus diseases and virus research in Hawaii reveals that although a considerable number of viruses occur here, most of them have received only passing notice. Those which have been investigated most extensively are: yellow spot of pineapple (spotted wilt of tomatoes), sugar cane mosaic, chlorotic streak of sugar cane, corn mosaic, and to a lesser extent papaya mosaic and *Commelina* mosaic. Other viruses occur on truck crops, banana, ornamental flowers and weed hosts. These received little attention in earlier years because the plants on which they occurred played a very minor role in the agricultural economy of the Territory, or the viruses were not known to cause serious damage to their host plants.

The increasing importance assumed by diversified crops in Hawaii has resulted in a greater awareness on the part of entomologists and pathologists of the importance of virus diseases on the respective crops. Furthermore, the number and importance of these problems are usually found to increase as the acreage which is devoted to the crops in question increases.

PART I. PRINCIPLES OF PLANT VIRUS TRANSMISSION BY INSECTS

Before discussing particular diseases and insect vectors which occur in Hawaii, the nature of viruses and some of the pertinent general principles and problems associated with the transmission of viruses by insects will be considered briefly.

I—*Nature of viruses.* The ultimate nature of viruses has been the subject of speculation for many years. Because they are known to multiply only in living cells, and because they exhibit other properties which are usually considered characteristic of living organ-

isms, the opinion was prevalent until recent years that viruses were living entities. The announcement by Stanley (218, 219)† in 1935 and 1936 that he had isolated what appeared to be pure tobacco mosaic virus, and that it was composed of a crystalline protein of high molecular weight, was therefore an important milestone in virus research. Stanley also demonstrated (220) that by chemical treatment, the structure of this protein, which was shown by Bawden and Pirie (10, 11) to be a nucleoprotein, could be altered with resultant loss of virus activity. Reversal of this process restored the original structure of the protein and virus activity was thereby regained. Other workers have confirmed Stanley's results and several other viruses have since been demonstrated to be nucleoproteins of chemical structure similar to but distinct from that of tobacco mosaic.

II—*Transmission of plant viruses.* All plant viruses can probably be transmitted to susceptible hosts by grafting, providing grafts can be established. A few are transmitted through the seed and many can be transmitted by mechanical inoculation. However, the most important manner in which viruses spread from plant to plant in nature is by means of their insect vectors. Although the majority of insect species which transmit plant viruses occur among the aphids and leafhoppers, several species in other groups have also been incriminated as vectors. Below is given a list of the arthropod groups which have been reported as containing vectors of plant viruses. The species names are given for the vectors in all groups except the aphids and leafhoppers which comprise an extended list.

ARTHROPOD VECTORS OF PLANT VIRUSES

(Exclusive of the major groups of vectors, i.e., Aphididae, Cicadellidae)

Note: Further confirmation is needed of the transmitting ability of species whose names are preceded by an asterisk.

Species	Virus	References
ACARINA		
Eriophyes ribis Nalepa	Black-currant reversion disease virus	4, 96
INSECTA		
Orthoptera		
Melanoplus spp.	Potato spindle tuber virus	82
COLEOPTERA		
Diabrotica vittata Fab.	Cucumber mosaic virus	51, 52
D. duodecimpunctata (Fab.)	Cucumber mosaic virus	122
D. soror Lec.	Cucumber mosaic virus	9
Leptinotarsa decimlineata Say (larvae)	Potato spindle tuber virus	122
LEPIDOPTERA		
Pieris rapae (Linn.)	Cabbage mosaic virus	121

† Figures in parentheses refer to literature cited at the end of the article.

Species	Virus	References
DIPTERA		
**Tipula paludosa* Meigen (larvae)	Potato spindle tuber virus	59
THYSANOPTERA		
Thripidae		
Thrips tabaci Lind.	Tomato spotted wilt virus	125, 187
Frankliniella nigripes (Girault)	Tomato spotted wilt virus in Australia	8, 201
F. schultzei (Trybom)	Tomato spotted wilt virus in S. Africa	148, 149
F. moultoni Hood	Tomato spotted wilt virus in N. America	76, 77, 78
F. paucispinosa Moulton	Tomato spotted wilt virus in S. America	65
HEMIPTERA		
Piesmidae		
Piesma quadrata Fieb.	Beet leaf curl (Kräuselkrankheit) virus	96
P. cinerea (Say)	Beet savoy virus	96
Miridae		
Lygus pratensis Linn.	Rape savoy virus	96
	Potato spindle tuber virus	82
	Potato mosaic virus	59
HOMOPTERA		
Coccidae		
**Pseudococcus maritimus* (Ehrhorn)	Bean mosaic virus	58
**P. citri* (Risso)	Tobacco mosaic virus	164
**Lecanium corni* Bouché	Vine mosaic virus	214
Aleyrodidae		
Bemisia gossypiperda Misra and Lamba	Cotton leaf curl virus	80
B. nigeriensis Corb.	Cassava mosaic virus	81
Psyllidae		
**Mesohomotoma tessmani* (Aulm.)	Cacao swollen shoot disease virus	189
Cercopidae		
Philaenus leucophthalmus (Linn.)	Peach yellows virus	130
Delphacidae		
Peregrinus maidis (Ashm.)	Corn mosaic virus	115
Perkinsiella saccharicida Kirk.	Sugar cane Fiji disease virus	150
P. vastatrix Breddin	Sugar cane Fiji disease virus	159

Insect Transmission of Viruses

Virus diseases fall into two general groups on the basis of the insect vector relationships involved. The factors which determine these various vector-virus relationships are of fundamental importance from the standpoint of the biological phenomena involved as well as because of their economic importance in the dissemination of virus diseases.

One type of transmission is that in which the vector is able to transmit the virus immediately or within a very short time after it first feeds on a diseased plant. Such viruses are usually not retained

by their vectors for more than a short time after the vectors feed on healthy or immune plants. Watson and Roberts (237) have called this the "non-persistent" type of virus.

The other general type, designated as "persistent" viruses, includes those which must undergo a latent or incubation period in their insect vectors before the latter are capable of infecting healthy plants. This period varies from less than an hour up to several weeks depending upon the vector and virus involved. Furthermore, the vectors of persistent viruses retain the ability to infect healthy plants during much or all of their subsequent life without the need of feeding again on a virus source.

Non-persistent viruses. Viruses of the non-persistent type have the following general characteristics:

1. They usually require no incubation period in the vector.
2. They are not long retained by the vectors after the latter leave a diseased plant.
3. They are usually transmissible by mechanical means.
4. There is usually a relatively low degree of specificity between the virus and its vectors, particularly with reference to the aphids. Most of these viruses can be transmitted by several species. There are no well authenticated cases of leafhoppers transmitting non-persistent viruses.

Most of the virus diseases known in Hawaii are of the non-persistent type. A few of this type are transmitted mechanically with such ease that much of the spread in the field may be traceable to contaminated tools, clothing or hands. Two of our most common virus diseases—tobacco or tomato mosaic and cucumber mosaic—are among those which can easily be transmitted mechanically. The former, however, is paradoxical in that it is very difficult to transmit by means of insects.

Because of this ease of transmission by mechanical means, the idea has become accepted by many people that insect transmission of non-persistent viruses is also a purely mechanical process resulting from contamination of the vector's mouthparts. Yellow dwarf of onions, which has been transmitted by more than 50 species of aphids, is usually cited as an example of mechanical transmission by sucking insects. Other viruses which have been placed in this group are those causing potato spindle tuber and common cucumber mosaic. These are included in this group because they are easily transmissible by juice inoculation and also because several chewing insects, in addition to a number of sucking species, have been listed as vectors. Mechanical transmission of viruses by insects probably occurs in some cases, but recent work indicates that transmission by most sucking insects may involve biological as well as mechanical factors. Cucumber mosaic is a case in point.

Studies by Watson and Roberts (237) in 1939 on transmission of cucumber mosaic, Hyoscyamus virus 3 and potato virus Y by means of aphids, produced some unusually noteworthy results on this subject. They tested the transmitting efficiency of *Myzus persicae* (Sulz.), *M. circumflexus* (Buck.) and *Macrosiphum solanifolii* (Ashm.) under various conditions. It was found that the efficiency of the vectors was greatly increased if they were prevented from feeding for a time just prior to being placed on the disease inoculum. The transmitting efficiency increased with increased fasting time up to 1 hour. This unusual effect was obtained, however, only if the time the aphids fed on the source of the virus was reduced to a short period immediately preceding transfer to the healthy test plants. A 2-minute feeding period on inoculum was found to be most satisfactory. The efficiency of previously starved aphids decreased as the time of feeding on the infected plants increased. If this time was extended to an hour there was no increase in efficiency over unstarved aphids. The explanation suggested by the authors for the results reported was that the viruses are inactivated by some substance, such as an enzyme, produced by the aphids during feeding. This substance, according to their hypothesis, is not produced, or at least not in effective quantities, while the aphids are fasting. Furthermore, for several minutes after feeding is resumed, the inactivating substance may not be produced in sufficient quantity to inactivate the virus being ingested. Hence, fasting and short feeding periods result in increased efficiency of virus transmission.

Watson and Roberts also found that the species of aphids varied in their efficiency as vectors despite the fact that during the short feeding periods reported, all three species fed in the same parenchyma tissue. In view of the varying efficiency of the species, while feeding in the same tissue under the same conditions, the authors concluded that aphid transmission in these instances involves a complex vector-virus relationship and that the viruses are not transmitted mechanically. *M. solanifolii* was the least efficient of the three species under consideration. This was explained on the basis of more inactivating substance being produced by this aphid. Kassanis (110, 111) obtained similar results with the same and other species of aphids in transmitting tobacco etch viruses. In these experiments, efficiency was greatest if the aphids were starved for 4 hours and then fed for 2 minutes on inoculum before being transferred to healthy test plants.

These experiments are in contrast to differential efficiency exhibited by the same species of aphids in transmitting potato leaf roll (55) where high efficiency was correlated with phloem feeding which required longer periods of feeding time on the inoculum.

As further evidence that loss of infectivity by aphids is not due to cleansing of the mouthparts during feeding, Watson and Roberts (238) demonstrated that individuals of *Myzus persicae* may infect

a succession of healthy plants if the aphids are first subjected to a fasting period followed by a 2-minute feeding on infected plants before transfer to the healthy test plants. In contrast to these results unstarved infective aphids are usually unable to infect a second healthy plant if they are allowed to feed on the first plant for several minutes. The aphids which fed discontinuously, inserted and removed their stylets from as many as 10 different healthy plants before losing infectivity. During the same time interval, unstarved infective aphids feeding continuously on a single healthy plant lost the ability to transmit the disease. Therefore, it seems highly improbable that such viruses are only mechanically transmitted and are lost by being rubbed off during insertion and withdrawal of the insect stylets through healthy plant tissue.

On the basis of these experiments Watson and Roberts concluded that the presence of the hypothecated inactivating substance is the primary difference between the so-called non-persistent viruses and those which persist in their vectors. However, this hypothesis does not provide an explanation for the incubation period of viruses of the persistent type in their insect vectors. Retention of the persistent type virus by the vector for indefinite periods might be explained by the absence of an inactivating substance, but in such a case some other fundamental process or mechanism must be responsible for the latent period of the virus in the vector before transmission is possible. Furthermore, the factors involved in this process may be more complex than those which permit indefinite retention of the virus. However, since viruses which require an incubation period in their vectors are also always retained by the vectors for indefinite periods of time, it is probable that the factors governing the two phenomena are the same or at least are intimately associated.

Viruses of the persistent type. Viruses of the persistent type usually have the following characteristics:

1. A latent or incubation period in the body of the insect vector. This is the period between the acquisition of the virus by the insect and the time when the insect becomes capable of infecting healthy plants with the virus.
2. After becoming viruliferous the insect vectors usually retain the virus for an extended period of time or for life without the need of again feeding on a diseased plant.
3. The vectors are usually phloem feeders and the viruses usually produce symptoms which are associated with phloem disturbances. However, some persistent viruses occur in both phloem and parenchyma.
4. Most persistent viruses are not sap transmissible.
5. There is a greater degree of specificity between the insect vector and the virus than occurs among the non-persistent viruses.

Incubation period in the vector. An incubation period of the virus apparently occurs in the insect vectors of all viruses of the persistent type. In Hawaii, this has been demonstrated to occur in *Peregrinus maidis* (39) in the transmission of corn mosaic and in *Thrips tabaci* (125) during transmission of yellow spot of pineapple (spotted wilt of tomato). Presumably the incubation period shown to exist in *Myzus persicae* in transmitting potato leaf roll (215) also obtains in Hawaii, although this has not been tested experimentally.

The nature of the incubation period of the virus in the vector has been a subject of considerable discussion for a number of years but as yet no unanimity of opinion has developed on the question. The principal theories suggested to explain this latent period are: (a) that the virus must multiply itself in the body of the vector in order to develop a concentration great enough to permit emission of an infective dose by the insect; (b) that the latent period is the time required for the virus to pass through the walls of the intestine, enter the blood and make its way into the salivary glands from which it is presumably injected into the plants during feeding; (c) that it is a period during which the virus is changed in some necessary manner before the vector can transmit it.

The second theory is difficult to test because of the complications encountered in trying to detect the presence of the virus in specific tissues or fluids of the insect vector. It has been accomplished with respect to some tissues in a few insects, but not in experiments which were designed to measure the rate of virus movement through the respective tissues. As yet there has been no way discovered to obtain concrete evidence on the last theory.

The theory regarding multiplication in the insect has been reviewed by Storey (222), Leach (122) and Bawden (9). In this paper it will be discussed but briefly, and some aspects which have not been considered previously will be mentioned.

There is persuasive evidence both for and against the idea that the latent period in insects is due primarily to multiplication of the virus to an infective threshold. When sufficient facts are available they may reveal that virus multiplication occurs in some species and not in others. We can be certain that the problem is not a simple one even in the case of an individual disease and its vector. Although the incubation period of a given virus in its vector is usually constant within general limits, there may be wide differences between individuals within the same species. One of the most extreme examples is that demonstrated by Carter (39) in relation to *Peregrinus maidis* and discussed more fully later in this paper. In this species the latent period varies from 4 to 29 days. If this period is based upon multiplication of the virus, it is apparent that the physiological differences occurring among individuals or genetic

lines within a species have a direct influence on the multiplication rate.

Kunkel (119) in 1937 demonstrated that if colonies of infective leafhoppers of *Macrosteles divisus* (Uhl.) were exposed to temperatures of 31 to 32° C. for 1 day or longer, they lost the ability to transmit aster yellows virus either permanently or temporarily. A 12-day exposure to this temperature resulted in permanent inactivation of the virus in the insect. However, "colonies treated from 1 to 11 days regained ability to transmit after periods varying from a few hours up to many days. The longer the colonies were heat-treated, the longer it took them to regain ability to transmit. Colonies in which virus was undergoing natural incubation were affected to a greater degree by heat treatments than colonies that were infective at time of treatment." These results were interpreted to mean that short treatments inactivated only part of the virus and transmitting ability was regained when the virus had again increased to an infective level. The 12-day treatment was assumed to have inactivated all of the virus in the insect. The more marked effect of heat treatment on colonies in which the virus was undergoing natural incubation was construed as evidence that the quantity of virus in these insects was lower at the time of treatment than in insects already infective when treated. Kunkel showed further (120) that this leafhopper can live and reproduce normally at temperatures higher than those which inactivated the virus. This fact, together with the demonstrated ability of leafhoppers to become infective a second time by feeding on a diseased plant after all the virus had once been inactivated in them by heat treatment, are cited as adequate evidence that the loss of transmitting ability due to heat treatment resulted from an effect on the virus itself and not on the insect.

Black (17) reported experiments which were presented as additional evidence that the aster yellows virus multiplies in its vector. His technique involved transmitting the virus mechanically from insect to insect, usually at 0° C. By this method he demonstrated that juice from viruliferous insects is infectious at dilutions as high as 1 : 1000 in 0.85 per cent NaCl solution. Furthermore, although the minimum incubation period in mechanically inoculated insects varied from 11 to 45 days, the insects usually remained infective until they died. Black also presented evidence indicating that the virus reached its highest concentration in the insects several days before the vectors could transmit the virus to asters. Moreover, the virus concentration in some instances appeared to decrease towards the end of the incubation period. It was therefore suggested that part of the incubation period may represent a period of multiplication of the virus while the remaining portion may be the time required for the virus to move from the centers of multiplication to a site in the insect from which it may be injected into the plant.

One of the strongest arguments cited as evidence of virus multiplication in the insect vector is based on Fukushi's work with *Nephotettix apicalis,* var. *cincticeps* Uhl., the vector of rice dwarf disease. This is the only known case of a virus being transmitted through the egg of the vector to its progeny. Fukushi (*72, 73, 74*) in 1933, 1935 and 1939 demonstrated that congenital transmission through the egg takes place only when the female parent carries the virus. Infective progeny resulted from crosses involving viruliferous females and non-viruliferous males but not when the reciprocal crosses were made. The virus was passed from insect to insect through the egg to the 7th generation without the insects in question feeding on disease inoculum.

The justification for interpreting the various experiments reported here in the manner indicated above has been questioned by some, notably Bawden (9). The work on several other insect vectors suggests that if virus multiplication occurs, the rate must vary or else it is not fast enough to maintain the original charge of the virus. Otherwise multiplication should maintain the virus at an infective level throughout the life of the insect. This does not happen in many instances, examples of which are discussed below.

Freitag (69) showed that the ability of *Eutettix tenellus* (Baker) to transmit curly top virus diminished with increased time after completion of the incubation period. During the latter part of their lives, many leafhoppers completely lost the ability to transmit the virus or transmitted more and more infrequently. However, if such leafhoppers were permitted a second feeding on inoculum, they again became viruliferous. These results suggested that loss of infectivity had been due to exhaustion of the virus supply in the insect. This fact was confirmed for *Eutettix tenellus* by Bennett and Wallace (14), for *Cicadulina mbila* (Naudé) by Storey (222) and for *Peregrinus maidis* by Carter (39). Freitag also found that the length of the original feeding period on the virus source had an effect on the duration in the insect of infective ability. Short exposure to inoculum tended to result in a shorter infective period for the insect. A feeding period of several hours or days on inoculum usually resulted in more prolonged infectivity. However, there appeared to be a limit beyond which further inoculum feeding produced no additional effect in prolonging virus retention.

Another objection to the idea of virus multiplication in the vector is, that although the length of feeding time on the diseased plant may determine to some degree how long the virus is retained by the vector, it has little effect on the duration of the incubation period itself. This may be as long in a vector which fed for several hours or days on a diseased plant as it is for the same species when fed but a fraction of this time on inoculum. If the latent period is primarily one during which the virus must multiply to an infective

level in the insect, there should be a direct relation between the initial quantity of the virus ingested by the insect and the duration of the latent period. However, such a relationship does not exist. Even Black's experiments involving high dilutions of virus, which were considered to be evidence of multiplication in the vector, show that the incubation period in leafhoppers inoculated with the juice from viruliferous insects was as long in those inoculated with undiluted juice as it was in those inoculated with dilutions of 10^{-1} and 10^{-2}.

Another aspect of the problem which has not been considered is that if the incubation period of persistent viruses is due to multiplication of the virus, the minimum infective dose of such viruses must be many times that which is effective in the case of non-persistent viruses. As discussed previously, many of the latter viruses are transmitted most readily if the vector is permitted only a short feeding period of 2 minutes on the diseased plant and is then transferred immediately to the test plant. In contrast to this, even though vectors of persistent viruses are permitted to feed for several hours or days on the virus source, during which time the amount of virus ingested must be considerable, they are nevertheless incapable of infecting a healthy plant until the usual incubation period has been run.

Furthermore, the fact that infective individual vectors of persistent viruses frequently do not infect all of the plants they feed on but fluctuate irregularly in their transmitting ability, suggests that factors other than virus multiplication must also be involved.

Storey (*222*) cited a circumstance regarding the multiplication hypothesis which is of interest. When the virus of maize streak was introduced directly into the body of *Cicadulina mbila,* this leafhopper became infective, but within 1 to 3 weeks thereafter it again became non-viruliferous. In contrast to this, leafhoppers which were permitted to feed for only 15 seconds on a diseased plant retained the virus for 9 weeks. The reason for this is not understood, but it does show that the virus is more effective if taken in by mouth than by inoculation into the body cavity. Storey also suggested that entry by the intestinal route may be a necessary condition of multiplication if such multiplication occurs.

Relation of Persistent Viruses to the Plant

Bennett (12) has pointed out that several viruses of the type referred to here as persistent, are rather closely restricted to the phloem. However, others such as maize streak and spotted wilt occur in both phloem and parenchyma. Most persistent viruses have also been found to be transmitted by insects which ordinarily feed in the phloem. Dykstra and Whitaker (55) correlated transmitting efficiency in aphid vectors with the tissues in which they fed. Those feeding habitually in the phloem were found to be efficient vectors of potato leaf roll.

Studies by Fife and Framptom (66) revealed the manner in which one insect, *Eutettix tenellus,* is guided to the phloem in which it must feed in order to acquire or transmit the virus of sugar beet curly top. The saliva of the leafhopper was found to be alkaline in reaction. The reaction of the parenchyma tissue of the sugar beet leaf petiole is acid, but there is a pH gradient toward the alkaline side as the phloem is approached. This gradient increases sharply in the cells of the bundle sheath and in the adjacent parenchyma cells. It reaches its highest point in the phloem where the pH is approximately the same as that of the leafhopper's saliva. It was inferred, therefore, that the acid reaction of the outer parenchyma is distasteful to the leafhopper and that the latter is guided to the alkaline phloem by the pH gradient described. This idea was supported by the feeding response of the leafhoppers on plants treated with a high concentration of carbon dioxide. This treatment upset the pH gradient in the petiole of the sugar beet with the result that the phloem was temporarily made more acid than some of the parenchyma. When *Eutettix* fed on plants of this type they reached the phloem in only 12 per cent of the trials, whereas in normal plants 56 per cent of the feeding trials terminated in the phloem.

The feeding habits of the sugar cane leafhopper, *Perkinsiella saccharicida,* and its role in transmitting Fiji disease of sugar cane present an unusual situation which is not yet fully understood. North and Barber (158) in 1935 reported that the nymphs of this insect usually feed in the phloem whereas the adults appear to be indifferent to the vascular bundles and feed in the mesophyll. Nymphs can easily be shown to acquire the virus but apparently adults do not. It is possible that the difference in feeding habits is responsible for the inability of the adults to obtain the virus. However, if this is true it will be a case in which the virus can be acquired by the vector only from the phloem but can be introduced into a healthy plant through the mesophyll as well as the phloem. This must follow because adult leafhoppers can transmit the virus provided they acquired it during their nymphal stage.

The fact that viruses of the persistent type are usually, but not always, restricted to the phloem or occur in the phloem in the greatest concentration, provides an explanation why such viruses are seldom found to be sap-transmissible. The insect vectors are equipped to introduce the virus into the phloem cells without causing the death of these or of the surrounding cells. However, the relatively crude methods of mechanically inoculating viruses which have been developed thus far make it exceedingly difficult to reach the phloem cells without causing severe disruption of the tissues penetrated. As a result, the virus fails to establish itself except in rare instances.

It is of interest in this connection that Storey (222) and Black (17) have been able to inoculate successfully the viruses of maize streak and aster yellows into their respective insect vectors by mechanical means, but that the same viruses cannot be transmitted from plant to plant mechanically. In the case of maize streak, the virus can be transferred mechanically to the vector from infective plant juice and also from the body juice and freshly expelled feces of viruliferous leafhoppers (222).

Some factors affecting virus transmission by insects.

One of the most intriguing problems relating to virus transmission by insects, concerns the factors which determine which insect species may serve as vectors and under what particular conditions. The problem is a complex one involving the relationships of the insect to both the virus and the plant and also the relationships of the virus to the plant. A few of the possible factors will be considered briefly.

1. Non-vector species of insects may be able to acquire a virus and retain it for extended periods of time but lack the ability to infect healthy plants. Bennett and Wallace (14) demonstrated the presence of curly top virus in non-vector species of aphids, leafhoppers, thrips and mites. When transferred to healthy plants the virus was retained by *Myzus persicae* for 14 days and by *Aceratagallia californica* (Bak.) for 21 days, yet they were unable to effect transmission. The authors concluded: "Since the virus retained its activity for several days in the bodies of the two species of insects named above and since both fed in the phloem, their inability to cause infection was not due to lack of active virus or to the tissue on which they fed, but evidently resulted from the presence of an effective barrier to virus passage in some part of the insect."

Such a barrier has been demonstrated by Storey (222) to occur in a genetic strain of *Cicadulina mbila,* the vector of maize streak virus. By selective breeding, Storey was able to produce a pure stock of leafhoppers all of the members of which could transmit the virus. Within the same species he bred another pure stock in which none of the insects could normally serve as vectors. The first stock was called the "active" race and the second the "inactive" race. When these pure races were crossed it was found that "activity" was dominant and was transmitted as a simple Mendelian character. The most significant contribution which emerged from this work was the knowledge that the barrier to activity in this species occurred in the intestinal wall of the leafhopper. Inactive individuals could be made active vectors if, after they had fed on a diseased plant, the stomach wall of the insects was punctured with a fine needle to permit some of the infective plant juice to enter the blood

in the body cavity. The same result could be obtained by injecting infective plant juice or extracts from infective insects directly into the body cavity of inactive individuals.

2. Insects may be capable of acquiring the virus during only one of the life stages. This is true of thrips and apparently is also true of *Perkinsiella saccharicida* in transmitting spotted wilt and Fiji diseases respectively. In both instances the virus apparently must be acquired before the adult stage is reached. However, infective nymphs develop into adults capable of transmitting the virus. The reason for this is not understood. It is possible that a change takes place in the permeability of the intestinal wall which prevents passage of the virus in adults. This possibility might be tested by applying the same technique Storey (222) used in making vectors of "inactive" *Cicadulina mbila.* In the case of the sugar cane leafhopper the reason may also be found in the different feeding habits of the nymphs and adults as discussed earlier.

3. The insect vector may be able to acquire the virus from one host plant but not from a related species which also carries the virus. This has been demonstrated to be the case in aphid transmission of tobacco mosaic virus from tomato but not from tobacco. The same species of aphids may however transmit other viruses to and from tobacco. Black (16) and others have proved that the juices of several insect vectors inhibit the infectivity of tobacco mosaic and several other viruses. As yet, however, it has not been demonstrated that failure of insects to transmit viruses is due to virus inactivation by the living insects, although the work of Watson and Roberts discussed earlier is very suggestive in this regard.

4. Another question which bears on the role of insects in transmitting virus diseases pertains to the manner in which an infective quantity of virus is introduced into the plant. It is common knowledge that many viruses can be transmitted at will by the use of a large number of vectors whereas tests involving individual insects often result in no transmission or in but a small percentage of positive cases.

Carsner and Lackey (33) and Severin (204) have advocated the "mass action" hypothesis. According to this idea, the increased percentage of transmission provided by groups of insects over individuals is partially due to the combined effect of several small, sub-infective doses of virus, any one of which alone would not have been sufficient to cause the disease. In contrast to this idea, Storey (222) and others hold that "a group will succeed if at least one of its members would have succeeded alone; if none would have succeeded alone, then the group will not succeed."

The same viruses and vectors were not used in arriving at these diametrically opposed hypotheses. The process by which several individually sub-infective virus injections attain infective concen-

tration in a plant has not yet been adequately explained. More work needs to be done on the subject. However, in passing, a report from Queensland is of particular interest in this regard despite the fact that experimental evidence from which the conclusions were drawn is not cited. The report of the director of the sugar experiment stations of Queensland for 1938 (194) contained a discussion of the susceptibility of certain cane varieties to Fiji disease. These varieties contain a certain amount of wild cane ancestry in their stock which provides desirable plant vigor and resistance to mosaic but confers high susceptibility to Fiji disease. The nature of the susceptibility is of unusual interest. "Whereas a stool of a resistant variety may require to be fed upon by say eight or ten infective insects [*Perkinsiella saccharicida*] before it will contract the disease, susceptible varieties may require only one insect per stool. When these insects are blown out of a diseased field by a high wind it is obvious that many stools will receive one hopper whereas very few would receive, say, five or six. Herein lies the danger of a susceptible variety in that it can become diseased when fed upon by a single infective insect."

If susceptibility and resistance of this type exists, it is strong evidence that the infective dose necessary to cause the disease varies with the variety of cane. Furthermore, the "mass action" hypothesis is necessary to account for infection by 5 to 10 leafhoppers on plants which fail to become infected when fed upon by but a single viruliferous leafhopper.

5. Two distinct viruses occurring together in a plant may be transmitted by a vector which is incapable of acquiring one of the component viruses when such occurs alone in a plant. Smith (216), during the past year, has reported that the rosette disease of tobacco is due to a complex consisting of two distinct viruses: vein-distorting virus and mottle virus. The vein-distorting virus cannot be transmitted mechanically but is transmitted by *Myzus persicae*. The mottle virus is easily transmitted mechanically but cannot be transmitted by *Myzus persicae* unless it occurs in the same plant which carries the vein-distorting virus. When this condition occurs the complex may be transmitted by the aphid at daily intervals for 20 days without again feeding on the virus source. Moreover, once the aphid has become infected with the virus complex, it may sometimes transmit only the mottle virus, at other times only the vein-distorting virus and sometimes the entire complex.

Origin of "new" virus diseases.

New virus diseases are being described each year in various parts of the world. Of interest to us here in Hawaii is the fact that less than a year ago a previously undescribed virus disease of papaya was discovered at Kailua, Oahu. This disease shows evidence of

becoming a serious threat to the papaya industry here if it continues to spread at its present rate. Whenever such a new virus appears on a commercial crop plant the question arises as to where it came from or how it originated. There are several possible answers but in the case of most new viruses the correct answer usually cannot be determined. Among the possible answers are the following:

1. The virus disease may have been introduced from some other part of the world where it had not been observed previously.
2. The virus may have been produced as a new disease for the first time in the host plant in which it is found. How this might take place has not been demonstrated. However, Johnson (104) in 1942 reported negative results from experiments he conducted to test a "viroplasm hypothesis." This postulates that extracts from healthy plants when inoculated into other healthy plants may result in some part of the protoplasm of the inoculum finding conditions compatible for growth and bring about an abnormality known as a virus disease in the host species.
3. The new virus may be a "mutant" strain of a virus already in existence, in which case the mutation may have altered the nature of the virus in relation to the host plant range, the insect vector or both. As evidence for this possibility, Jensen (102) published experimental results which he interpreted as evidence that yellow-mosaic viruses arise suddenly in plants infected with tobacco mosaic virus by some process similar to that of mutation.
4. The "new" virus may have existed previously in a different host plant, in which it may or may not have produced symptoms. It may have been confined to the original host or hosts for indefinite periods of time because, (a) a suitable vector was not previously available to transmit the virus out of the original host, or (b) though a vector was present which could transmit the virus among some hosts, no vector was previously present which was capable of disseminating the virus to susceptible hosts of economic importance.

Carter (36) has demonstrated that the operation of the situation described last is responsible for the freedom of pineapple from at least one virus to which it is definitely susceptible. This virus causes the common mosaic in *Commelina diffusa* (= *nudiflora* of Hawaiian authors[2]). He (36) showed that this virus is transmissible to young, tender pineapple plants by means of *Aphis gossypii* Glover, *Myzus persicae* and *Macrosiphum solanifolii*. Symptoms in pineapple closely resemble those produced by the yellow spot virus. Previous to Carter's work, pineapple had not been recorded as a

[2] The corrected identification of this common species of *Commelina* in Hawaii was provided by Dr. H. St. John of the University of Hawaii.

host for aphids. He found that the three species named would reproduce on very young seedlings but apparently are incapable of feeding satisfactorily on older pineapple plants. For this reason the virus is not likely to spread in the field. As pointed out by Carter, this demonstrates "that a plant may be highly susceptible to a virus but so unfavorable a host for a specific vector that natural infection is rare." It is probable that other viruses are also capable of causing serious injury to pineapple but lack vectors which can transmit them to this plant.

The only other insect-transmitted virus of pineapple is yellow spot, transmitted by *Thrips tabaci* (125). When yellow spot first appeared as a pineapple disease it was expected to be a major problem. However, as will be discussed more fully later, the disease is of minor importance because pineapple is not a preferred host of the vector.

Bennett (13) in 1944 reported a virus which needs only a suitable vector to make it a serious economic disease. This virus, named dodder latent mosaic (*Marmor secretum*), is symptomless and, until recently, was undetected in dodder, its normal host. It has now been shown by Bennett to induce a severe disease on cantaloupe, and a less severe disease on sugar beet, potato, celery, tomato and some other plants. The virus occurs naturally in dodder, *Cuscuta californica* Choisy, which grows on a desert shrub, *Eriogonum fasiculatum* Benth. in southern California. No symptoms are observable on dodder, but as this plant attaches itself to the crop plants listed, the virus is transmitted. It has been found to be juice transmissible, and, to a slight extent, is transmitted through the seed of *Cuscuta campestris,* one of three species of dodder which were shown to transmit it. No insect vector is known, but Bennett points out that with the introduction of an efficient vector, this virus might easily cause a serious disease on several cultivated plants.

PART II. INSECT SPECIES OCCURRING IN HAWAII KNOWN TO TRANSMIT PLANT VIRUSES, WITH A LIST OF VIRUSES TRANSMITTED BY EACH

Insect species occurring in Hawaii and which have been reported to transmit plant viruses in some part of the world are listed in table 1. Opposite the species names are given the number of different viruses or distinct virus strains which have been reported transmitted by the respective species. In table 2, the names of the different viruses transmitted by each species are given with references to the literature on which each entry was based. Future research will undoubtedly show some of these viruses and strains to be synonyms.

Table 1. Insect species occurring in Hawaii, known to transmit plant viruses, and the number of virus diseases transmitted by each.

Species	Virus Diseases Transmitted
1. *Aphis citricidus* (Kirk.)	1
2. *Aphis ferruginea-striata* Essig	4
3. *Aphis gossypii* Glover	16
4. *Aphis maidis* Fitch	3
5. *Aphis medicaginis* Koch	1
6. *Aphis middletonii* Thomas	4
7. *Aphis rumicis* Linn.	13
8. *Brevicoryne brassicae* (Linn.)	8
9. *Cavariella aegopodii* (Scopoli)[3]	5
10. *Macrosiphum rosae* (Linn.)	4
11. *Macrosiphum solanifolii* (Ashmead)	30
12. *Myzus circumflexus* (Buckton)	16
13. *Myzus convolvuli* (Kalt.)[4]	6
14. *Myzus persicae* (Sulzer)	53
15. *Pentalonia nigronervosa* Coq.	4
16. *Rhopalosiphum pseudobrassicae* (Davis)	6
17. *Toxoptera aurantii* (B. de Fonsc.)	2
18. *Peregrinus maidis* (Ashmead)	1
19. *Perkinsiella saccharicida* Kirk.	1
20. *Dracculacephala minerva* Ball	1
21. **Orthezia insignis* Doug.	1
22. **Pseudococcus citri* (Risso)	1
23. **Pseudococcus maritimus* (Ehrhorn)	1
24. *Thrips tabaci* Lind.	2
25. *Pieris rapae* (Linn.)	1

[3] This aphid has been listed in most literature as *Cavariella capreae* (Fab.).

[4] If *M. pseudosolani* (Theob.) is a synonym of *M. convolvuli* (see footnote **, p. 555), this species is reported to transmit 11 different virus diseases.

* Virus transmission has been recorded in the literature, however, in the light of more recent work it is doubtful if these species actually are capable of serving as virus vectors.

Table 2. Insect vectors occuring in Hawaii with a list of viruses transmitted by each species. The virus strains listed are based primarily on the classification proposed by Holmes (96).

Virus	References
Aphis citricidus (Kirk.)	
1. Pea mosaic virus	156
Aphis ferruginea-striata Essig	
1. Celery calico virus	205
2. Celery mosaic virus, western	206
3. Celery crinkle-leaf mosaic virus (strain of western celery mosaic virus)	70
4. Poison-hemlock ringspot virus	71

Virus	References
Aphis gossypii Glover	
1. Abacá mosaic virus	161
2. Bean mosaic virus	250
3. Cauliflower mosaic virus	215
4. Celery calico virus	205
5. Celery mosaic virus, western	206
6. Cowpea mosaic virus	142
7. Cucumber mosaic virus, typical strain	51, 52
8. Cucumber mosaic virus, lily mosaic strain	83, 96
9. Cucumber mosaic virus, lima bean mosaic strain	85
10. Cucumber mosaic virus, southern celery mosaic strain	53, 241
11. Lily mottle and tulip break virus group	28
(*Marmor tulipae*, amended)	
Lily coarse mottle virus	
Tulip coarse break virus	
12. Lily yellow-flat virus (= lily rosette virus)	163
13. Onion yellow dwarf virus	54
14. Pea mosaic virus	156
15. Ornithogalum mosaic virus	209
16. Poison-hemlock ringspot virus	71
Aphis maidis Fitch	
1. Cucumber mosaic virus, southern celery mosaic strain	241
2. Onion yellow-dwarf virus	54
3. Sugar cane mosaic virus	22, 84, 115
Aphis medicaginis Koch	
1. Bean mosaic virus	250
Aphis middletonii Thomas	
1. Cauliflower mosaic virus	215
2. Celery calico virus	205
3. Celery mosaic virus, western	206
4. Poison-hemlock ringspot virus	71
Aphis rumicis Linn.	
1. Bean mosaic virus	63, 250
2. Broad bean mild mosaic virus	248
3. Celery mosaic virus, western	206
Lupin sore-shin virus (= pea mosaic virus)	43
4. Narcissus mosaic virus	18, 19
5. Onion yellow-dwarf virus	54
6. Pea mosaic virus	44, 169
7. Poison-hemlock ringspot virus	71
8. Potato leaf roll virus	165
9. Soybean mosaic virus	9
10. Spinach yellows virus	49
11. Sugar beet mosaic virus	96, 215
12. Sugar beet yellows virus (= Beta virus 4)	215
13. White clover mosaic virus	215
Brevicoryne brassicae (Linn.)	
1. Bean mosaic virus	250
2. Cabbage mosaic virus	121
3. Cauliflower mosaic virus	228
4. Crucifer black ring virus	231
(Probably the same as cabbage ring necrosis virus of Bawden 9)	

Virus	References
5. Crucifer mosaic virus (= Brassica virus 4) (may be turnip mosaic virus)	215
6. Onion yellow-dwarf virus	54
7. Stock mosaic virus (mild and severe strains)	230
8. Turnip mosaic virus	229
Cavariella aegopodii (Scopoli)	
1. Carrot virus	224
2. Cauliflower mosaic virus	215
3. Celery mosaic virus, western	206
4. Pea mosaic virus	156
5. Poison-hemlock ringspot virus	71
Macrosiphum rosae (Linn.)	
1. Celery mosaic virus, western	206
2. Narcissus mosaic virus	19
3. Onion yellow-dwarf virus	54
4. Pea mosaic virus	156
Macrosiphum solanifolii (Ashmead)	
1. Bean mosaic virus (common)	63, 153, 186, 250
2. Bean mosaic virus (yellow) (= Phaseolus virus 2)	186
3. *Commelina diffusa* mosaic virus	36
4. Cowpea mosaic virus	142
5. Cucumber mosaic virus, typical strain	89, 90
6. Cucumber mosaic virus, southern celery mosaic strain	36
7. Cucumber mosaic virus, potato veinbanding strain (= potato virus "Y" = Solanum virus 2)	57, 113
8. Hyoscyamus virus III	237
9. Iris mosaic virus (probably the same as iris stripe, 9)	27
10. Lily latent virus	96
11. Lily coarse mottle virus (part of *Marmor tulipae* Holmes, amended)	28
12. Narcissus mosaic virus	18, 19
13. Ornithogalum mosaic virus	209
14. Passion fruit woodiness disease virus	154, 155
15. Pea mosaic virus	44, 156, 169
16. Pea enation mosaic virus	167, 170
17. Pepper mosaic virus of Puerto Rico	195
18. Potato mild mosaic virus (virus "A")	57
19. Potato "bigarrure" virus	233
20. Potato calico virus	188
21. Potato leaf roll virus	57, 105
22. Potato crinkle mosaic virus	57
23. Potato spindle tuber virus, typical strain	96
24. Potato spindle tuber virus, unmottled curly dwarf strain	96
Potato rugose mosaic (See No. 7 for insect-transmitted vein banding component of this virus complex)	
25. *Primula obconica* virus	210
26. Soybean mosaic virus	9
27. Sugar beet mosaic virus	92
28. Tobacco etch virus, severe strain	110
29. Tobacco mosaic virus from tomato	93
30. Tulip breaking viruses (color adding and color removing strains; part of *Marmor tulipae,* Holmes amended)	26, 28, 140, 141

Virus	References
Myzus circumflexus (Buckton)	
1. Cauliflower mosaic virus	215
2. Celery calico virus	205
3. Celery mosaic virus, western	206
4. Celery crinkle-leaf mosaic virus (strain of western celery mosaic)	70
5. Cucumber mosaic virus, typical strain	89
6. Hyoscyamus virus III	237
7. Ornithogalum mosaic virus	209
8. Poison-hemlock ringspot virus	71
9. Potato mild mosaic virus (virus "A")	57
10. Potato veinbanding virus (= potato virus "Y")	57, 237
11. Potato crinkle mosaic virus	57
12. Potato leaf roll virus	57
13. Soybean mosaic virus	9
14. Tobacco etch virus, severe strain	110
15. Tobacco mosaic virus, typical strain	91
16. Tulip breaking virus	26
Myzus convolvuli (Kaltenbach)**	
1. Celery calico virus	205
2. Celery mosaic virus, western	206
3. Celery crinkle-leaf mosaic virus (strain of western celery mosaic)	70
4. *Freesia mosaic virus	215
5. Narcissus mosaic virus	19
6. Poison-hemlock ringspot virus	71
M. pseudosolani (Theob.)**	
1. Cucumber mosaic virus, typical strain	9, 89, 91
2. Potato leaf roll virus	59, 105, 152, 210, 212, 215
3. Soybean mosaic virus	9
4. Tobacco mosaic virus	89, 93
5. Tomato narrow leaf virus	42
Myzus persicae (Sulzer)	
1. *Allium neapolitanum* mosaic virus	29
2. Bean mosaic virus	250
3. Cabbage mosaic virus	121
4. Cabbage ringspot virus (Brassica virus 1)	215
5. Cabbage ring necrosis virus	9
6. Carnation streak virus	107
7. Cauliflower mosaic virus	228
8. Celery calico virus	205
9. Celery mosaic virus, western	206
10. Celery crinkle-leaf mosaic virus (strain of western celery mosaic virus)	70
11. Cowpea mosaic virus	142
12. Crucifer black ring virus (may be a synonym of ring necrosis virus)	231

** Since according to Essig (60), *M. pseudosolani* (Theob.) is probably a synonym of *convolvuli*, the viruses transmitted by aphids under these names are listed under *convolvuli* but identified with the aphid names which appear in the literature. Essig (60) also indicates that *M. convolvuli* and *M. pseudosolani* may be synonyms of *M. solani* (Kalt.).

* Transmission doubtful or in need of confirmation.

Virus	References
13. Cucumber mosaic virus, typical strain	88, 89, 90, 96, 237
14. Cucumber mosaic virus, southern celery mosaic strain	36, 96
15. Cucumber mosaic virus, potato veinbanding strain (= potato virus "Y")	57, 96, 113
16. Cucumber mosaic virus, lima bean mosaic strain	85, 96
17. Dahlia mosaic virus	96
18. *Datura virus 1	215
19. Garlic mosaic virus	29
20. Hyoscyamus virus III	237
21. Henbane (*Hyoscyamus niger* L.) virus	193
22. Iris mosaic virus (probably iris stripe virus of Bawden, 9)	27
23. Lettuce mosaic virus	3
24. Lily mottle virus group (part of *Marmor tulipae,* amended)	28
Lily coarse mottle virus	
Lily virulent coarse mottle virus	
Lily latent mosaic virus	
25. Lupin mosaic virus	156
Lupin sore-shin virus (= pea mosaic virus)	43
26. Onion yellow-dwarf virus	54
27. Ornithogalum mosaic virus	209
27a. Papaya ringspot virus (page 577)	
28. Passion fruit woodiness disease virus	154, 155
29. Pea enation mosaic virus	9
30. Pea mosaic virus	44, 156
30a. Peach mosaic virus***	
31. Pepper mosaic virus of Puerto Rico	195
32. Poison-hemlock ringspot virus	71
33. *Potato aucuba mosaic virus [probably]	96, 215
34. Potato "bigarrure" virus	233
35. Potato mild mosaic virus (= potato virus "A")	57
36. Potato crinkle mosaic virus	57
37. Potato tuber blotch virus (= potato virus "F" = Solanum virus 8)	9, 215
Potato rugose mosaic virus (See No. 15 for insect-transmitted vein banding component)	
38. Potato leaf rolling mosaic virus	57
39. Potato leaf roll virus	57, 105, 165, 215
40. Potato spindle tuber virus, typical strain	96
41. Potato spindle tuber virus, unmottled curly-dwarf strain	9, 96
42. Rape mosaic virus (may be turnip mosaic virus)	126
43. Soybean mosaic virus	9
44. Sugar beet mosaic virus	92
45. Sugar beet yellows virus	215
46. Stock mosaic virus (mild and severe strains)	230
47. Tobacco mosaic virus, typical strain	93, 96
48. Tobacco etch virus (mild and severe strains)	110
49. Tobacco rosette virus complex	93
Vein-distorting virus	
Mottle virus (only when combined with vein-distorting virus)	
50. Tulip breaking viruses 1 and 2 (color removing and color adding viruses, part of *Marmor tulipae,* amended)	26, 28, 140, 141
51. Turnip mosaic virus	229

* Transmission doubtful or in need of confirmation.

*** Daniels, L. B. 1945. The peach mosaic disease. Science 101:87-88.

Virus	References

Pentalonia nigronervosa Coquerel

1. Abacá bunchy top virus 160, 161
2. Banana bunchy top virus 128
3. *Banana mosaic virus [probably] 129
4. *Cucumber mosaic virus, southern celery mosaic strain 96

Rhopalosiphum pseudobrassicae (Davis)

1. Bean mosaic virus 250
2. Cauliflower mosaic virus 228
3. Celery mosaic virus, western 206
4. Onion yellow-dwarf virus 54
5. Pea mosaic virus 156
6. Stock mosaic virus (mild and severe strains) 230

Toxoptera aurantii (Boyer de Fonscolombe)

1. Citrus "little leaf" virus 215
2. Lemon ribbing virus 40

Peregrinus maidis (Ashmead)

1. Corn mosaic virus 39, 84, 115
2. Corn stripe virus (may be corn mosaic virus) 7, 217, 221

Perkinsiella saccharicida Kirkaldy

1. Sugar cane Fiji disease virus 150, 158

Draeculacephala minerva Ball

1. Pierce's vine disease virus 67

Pseudococcus citri (Risso)

1. *Tobacco mosaic virus 164

Pseudococcus maritimus (Ehrhorn)

1. *Bean mosaic virus [doubtful] 58

Orthezia insignis [Listed as *insignia*] Douglas

1. **Ephiphyllum truncatum* virus [doubtful] 20

Thrips tabaci Lind.

1. Spotted wilt virus causing:
   a. Tomato spotted wilt 76, 187, 200, 213
   b. Pineapple yellow spot 125, 200
   c. Pea streak 124, 246
   d. Cineraria streak 106
   e. Tomato tip blight 96, 146, 157
2. *Tomato mosaic virus [doubtful] 47

Pieris rapae (Linn.)

1. Cabbage mosaic virus 121

* Transmission doubtful or in need of confirmation.

PART III. PLANT VIRUS DISEASES KNOWN OR REPORTED TO OCCUR IN HAWAII WITH SPECIAL REFERENCE TO THEIR TRANSMISSION BY INSECTS

A list is presented of virus diseases which have been reported in the literature to occur in Hawaii. With a few exceptions no research work has been done in Hawaii on these viruses. Therefore experimental evidence is still lacking that the majority of the viruses reported from Hawaii are identical with viruses known by the same names in other parts of the world. Most of the work reviewed in this paper, on insect transmission of the diseases listed, was carried out in other regions. However, the majority of insect species recorded as vectors of these diseases occur in Hawaii. Furthermore, most of the diseases reported from Hawaii will probably prove to be identical with or related to diseases known by the same names in other areas. Knowledge regarding the identity of the vectors and of the factors which influence insect transmission of the diseases in question is therefore of fundamental importance to future studies undertaken in Hawaii.

Plant Virus Diseases Known or Reported to Occur in Hawaii

(Only those diseases indicated by an asterisk have been transmitted experimentally in Hawaii.)

Corn Mosaic

The virus disease known as maize mosaic or corn mosaic is very destructive to corn in Hawaii. If several crops are grown in close succession, as is frequently the case in Hawaii, the disease incidence becomes very high, sometimes involving over 50 per cent of the plants. This results from the development of a high population of infective leafhoppers many of which move to the new corn plantings.

Much of the pertinent work which has been done on the relation of insects to this disease has been carried out in Hawaii. However, both the disease and the relation of the vector, *Peregrinus maidis,* to the disease need further careful investigation.

The first published description of corn mosaic was that of Weston (245) in 1917. However, Kunkel (114) in 1921 stated that the presence of the disease and its destructive nature in Hawaii were recognized by Dr. H. L. Lyon as early as 1914. During the first ten years after corn mosaic was reported in the literature, the opinion prevailed generally that, because of the similarity of the symptoms, this disease would probably prove to be due to the same virus that caused sugar cane mosaic. The fact that sugar cane mosaic is transmissible to and from corn by means of *Aphis maidis,* was a complicating factor in clarifying the identity of the disease and its vector.

Under the name of corn mosaic, Brandes (23) in 1920, discussed a virus disease which occurred in sorghum and corn in Puerto Rico and the southern United States. He described the disease and reported experiments in which *Aphis maidis* transmitted it from mosaic sorghum to healthy corn and from infected corn to healthy corn. It is now obvious that he was dealing with sugar cane mosaic virus and not that of true corn mosaic.

Kunkel (115) in 1922 reported insect transmission of sugar cane mosaic and corn mosaic under the heading "Insect transmission of yellow stripe disease." He was the first to report transmission of corn mosaic by the corn leafhopper, *Peregrinus maidis.* His experiments indicated that the virus could be transmitted from corn to corn by *Peregrinus maidis* but could not be transmitted from corn to cane. Furthermore, he reported that the corn leafhopper failed to transmit the cane disease from cane to cane. The stripe disease in cane and mosaic of corn were at that time thought to be due to the same virus. It was not until 1927 that reports were published suggesting the distinct identity of the viruses, one of which caused mosaic or stripe symptoms in sugar cane, corn and other plants (and is transmitted by *Aphis maidis*) and the other (transmitted by *Peregrinus maidis*) which caused symptoms in corn but does not infect cane.

Kunkel (118) in 1927 reported that the corn leafhopper, *Peregrinus maidis,* occurring in North Carolina was unable to transmit

the virus of sugar cane mosaic of the United States to corn. He concluded, "this suggests that the destructive mosaic of corn prevalent in Hawaii is distinct from sugar cane mosaic and from the mosaic of corn occurring in Louisiana and other Southern States."

In the same year, Stahl (217) described a stripe disease of corn in Cuba and stated it was not identical with sugar cane mosaic. It was transmitted by *Peregrinus maidis*.

In 1928 Hadden (84), working on insect transmission of sugar cane mosaic in Hawaii reported: "Two insects have transmitted the disease: the corn aphis from various grasses to cane and other grasses; and the corn leafhopper, *Peregrinus maidis,* from corn to corn and in one case from corn to Striped Tip cane." Hadden considered it probable that the single case of disease transmission from corn to cane by *Peregrinus maidis* was an accident. Stahl's conclusion that corn mosaic and sugar cane mosaic were distinct was cited by Hadden who concluded: "Corn aphis may transmit one kind of mosaic. Corn leafhoppers may transmit a different kind of mosaic. Or both of these insects may transmit more than one kind of mosaic."

The identity and relationships of the corn viruses reported from different parts of the world have not been adequately established despite the fact that they have *Peregrinus maidis* as a common vector. Holmes (96) calls them all maize mosaic virus (*Marmor zeae*). However, Bawden (9) in 1943 listed corn mosaic and maize stripe as separate diseases.

The corn mosaic or corn stripe disease reported from parts of the world other than Hawaii may be summarized as follows:

1. *Cuba.* Stahl (217) in 1927 reported transmission of corn stripe disease by means of *Peregrinus maidis* but not by *Aphis maidis.*

2. *Trinidad, B.W.I.* Briton-Jones (30) in 1933 reported a severe stripe disease of corn in Trinidad. He considered this to be distinct from corn mosaic reported from Hawaii and other parts of the world except that reported by Stahl from Cuba. Later the same year Baker (7) transmitted this disease at Trinidad from maize to great millet by means of *Peregrinus maidis.* Briton-Jones (30) stated that the width of the stripes produced on diseased plants was dependent largely on the number of *Peregrinus maidis* which fed on the affected plants. "Those heavily infested with the insect develop broader stripes than those lightly attacked." This observation has not been reported by any other writer. Furthermore, the vector experiments reported in Briton-Jones' paper were inconclusive. The fertility and condition of the soil seemed to have more effect on symptom production in the plants than did the *Peregrinus maidis* which were caged on them, although some transmission by insects apparently occurred. The observation was also made by this author

that "*Peregrinus maidis* is very common on young maize until the tassel is produced when the insect leaves the plant." In Hawaii such a migration from the plant has not been observed, and the corn leafhopper may be found in large numbers on corn plants until after the mature plants are beginning to dry up.

3. *Puerto Rico.* White stripe disease of corn in Puerto Rico was described by Cook[5] in 1936 to correspond with the symptoms reported from Cuba and Trinidad.

4. *East Africa.* Storey (221) in 1932 reported the presence of a maize disease closely resembling maize streak. However, it was found to be transmitted by *Peregrinus maidis* and not by *Cicadulina mbila,* the vector of maize streak. He suggested the possibility that it might be identical with the stripe disease of Cuba.

Although transmission of one or more corn viruses by means of *Peregrinus maidis* has been reported by Kunkel (115) and Hadden (84) in Hawaii, Stahl (217) in Cuba, Baker (7) in Trinidad, and Storey (221) in Africa, no data on the incubation period of the virus and its retention in the insect were published until Carter's paper (39) appeared in 1941. His experiments demonstrated that the incubation period of the corn mosaic virus in *Peregrinus maidis* normally varies from 11 to 29 days. However, in some instances much shorter incubation periods occurred. Some were estimated as low as four days. In all cases this short incubation period occurred in insects which were genetically closely related. Carter cited the report by Storey (222), that the ability of *Cicadulina mbila* to transmit maize streak is controlled by genetic factors and suggested that the short incubation periods in *P. maidis* in a few genetically related individuals "may also be due to genetically controlled factors affecting permeability of the insect's tissues. If such a relationship could be conclusively proved, it would surely establish incubation period as a function of the physiology of the vector rather than of the virus." This is a direction in which important fundamental work could be done on a basic problem which is still unsolved.

Carter kept some leafhoppers on a diet of 2 per cent sucrose solution for seven days during part of the incubation period of the virus in the leafhopper. In these individuals the minimum incubation period was 13 days, thus demonstrating that the normal insect diet of juice from living plants is not essential for normal incubation of the virus in the vector.

Carter's work demonstrated that *P. maidis* can acquire the virus as nymphs or adults. The percentage of individuals which acquired and transmitted the virus in several different test groups varied from 9 to 57 per cent. Most individuals, but not all, which transmitted the disease, infected an unbroken series of several plants

[5] Cook, M. T. 1936. Phloem necrosis in the stripe disease of corn. Jour. Agr. Univ. Puerto Rico 20:685-688.

when transferred at 2-day intervals. Retention of the virus by the leafhoppers varied considerably. The maximum retention, as shown by transmission to a succession of healthy plants, was 48 days after feeding on the disease inoculum.

There is need for more extensive research on the role of *Peregrinus maidis* in disseminating corn mosaic. As yet it has not been determined how long a leafhopper must feed on a diseased plant before it can acquire the virus. It should also be determined whether the leafhopper must feed on certain portions of the leaves (such as areas showing the mottle symptom) in order to acquire the virus. In the experiments reported by Carter, the leafhoppers acquired the virus from leaf tissue. It would also be of interest to know if the vector can obtain the virus from stem tissue as well.

Carter (39) pointed out that some factor exists which complicates the acquisition of the virus by the vector and this is apparently responsible for the low percentage of individuals which become vectors under some conditions.

In view of the varying symptoms which have been observed on plants affected with corn mosaic and the fact that Wellman (242) transmitted southern celery mosaic virus to corn by means of *Aphis gossypii,* the possibility exists that this virus also may be found in corn plants diagnosed as being affected with corn mosaic virus. Wellman stated that corn plants so affected were stunted and in some other respects were remarkably similar to plants infected with Cuban corn stripe.

Sugar Cane Mosaic

Mosaic has been one of the most important diseases of sugar cane in the world because of its injury to the plant and because of its cosmopolitan distribution. The almost universal occurrence of one of its insect vectors, *Aphis maidis,* has undoubtedly been responsible in large measure for the greater economic importance of this disease from a world standpoint than some of the other virus diseases of cane whose vectors are more restricted in their distribution. Although mosaic was reported first from Java, Brandes and Matz (25) consider it probable that it originated in New Guinea which is part of the general region where sugar cane itself is believed to have originated.

In Hawaii, mosaic was first noted by Lyon in 1908. It was then called yellow stripe disease. Although extensive research was conducted in Hawaii between 1910-1914, the results were not published until 1921 (127). Since that time the disease has been under investigation in Hawaii as well as in other parts of the world.

Brandes (22) in 1920 was the first to report transmission of sugar cane mosaic by means of insects. He transmitted the disease with *Aphis maidis* from mosaic sorghum to healthy cane. In the same year he (23) transmitted mosaic from sorghum and corn to

healthy corn. He reported this as mosaic disease of corn, but in the light of our present knowledge concerning the vectors of sugar cane mosaic and corn mosaic it is apparent that the virus he worked with was that of sugar cane mosaic.

Before the separate identity of corn mosaic and sugar cane mosaic had been established, Kunkel (115) in 1922 reported experiments in Hawaii on the transmission of "yellow stripe disease" in which aphids and leafhoppers were used as the test insects and corn and cane as the test plants. Apparently the series of corn plants used as inoculum contained both cane mosaic and corn mosaic because Kunkel stated "*Aphis maidis* transferred from mosaic corn plants to healthy cane plants transmitted yellow stripe disease." And, "the corn leafhopper (*Peregrinus maidis*) transmitted corn mosaic from corn to corn, but failed to transmit it to cane from corn or from cane to cane."

In the same report Kunkel stated that the sugar cane aphid, *Aphis sacchari* Zehnt., and the sugar cane leafhopper, *Perkinsiella saccharicida,* failed to transmit the virus from diseased to healthy cane.

Hadden (84) in 1928 published the results of experiments conducted in Hawaii on the transmission of sugar cane mosaic by insects. He also reported observations on the ecology of *Aphis maidis* which was found to reproduce on 34 species of grasses. Both sugar cane mosaic and corn mosaic were treated in this study but they were spoken of as the same disease in some of the discussion. However, Stahl's (217) announcement, published in 1927, that corn stripe disease in Cuba was distinct from sugar cane mosaic was cited by Hadden who therefore concluded it was possible that the mosaics transmitted in Hawaii by *Aphis maidis* and by *Peregrinus maidis* might be due to distinct viruses. During the course of this investigation *Aphis sacchari, Perkinsiella saccharicida, Stictocephala festina* (Say), *Draeculacephala mollipes* (Say)[6] and *Tetranychus exsiccator* Zehnt. failed repeatedly to transmit sugar cane mosaic.

Ingram and Summers (99, 100, 101) in 1936, 1937 and 1938, announced the discovery of two additional aphid vectors of mosaic in the southern United States. These are *Hysteroneura setariae* (Thomas) and *Toxoptera graminum* (Rond.). The former species was found to be the most common aphid on cane between 1930 and 1937 and although a much less efficient vector than *Aphis maidis,* it apparently was responsible for much of the rapid spread of mosaic because *Aphis maidis* was scarce in most areas. *Toxoptera graminum* was not found as generally as *Aphis maidis* but was common in some fields.

Tate and Vandenberg (226) in 1939 described experiments which demonstrated that the aphid *Carolinaia cyperi* Ainslie transmitted mosaic in Puerto Rico. Of 192 plants exposed to viruliferous

[6] The species in question may have been *D. minerva.* See footnote, p. 471.

C. cyperi, 31.2 per cent became diseased. In similar experiments *Aphis maidis* infected 34.5 per cent of 200 test plants. *C. cyperi* was found to breed on nut grass, *Cyperus rotundus,* in and around the cane fields. According to St. John and Hosaka (196) this grass is common in some parts of Hawaii. Exploratory experiments with *Aphis rumicis, Macrosiphum rudbeckiae* (Fitch) and *Aphis nerii* B. de Fonsc. were also reported by Tate and Vandenberg. Nine experiments with each of the first two species were all negative, but a single plant out of four exposed to *A. nerii* developed mosaic symptoms. Although this cannot be considered a demonstration of transmission by *nerii* it does mark this species as one which should be tested more extensively.

Costa Lima (50) in 1926 reported the belief that *Thrips minuta* var. *puttemansi* Costa Lima was a vector of sugar cane mosaic in Brazil. However, this has not been demonstrated experimentally.

It has been well established that there are several different strains of sugar cane mosaic. Summers (225) reported four in Louisiana in 1934 and Martin (134) in 1939 stated that 10 strains or substrains are known to exist, some of which are more virulent than others. Cuttings with mosaic were sent from Hawaii to Washington, D.C. where their virulence was compared with that of other mosaic strains. The Hawaiian strain was found relatively difficult to transmit and when infection did occur the symptoms were considered mild. This is a fortunate circumstance for Hawaii. Although mosaic was once a serious cane disease in Hawaii, it is now considered to cause only minor losses. This has come about through the development of cane varieties which are resistant to the mosaic. However, the danger of mosaic again becoming a serious problem in Hawaii is always imminent. A cane variety resistant to one strain of mosaic is not necessarily resistant to all of the other strains. Mr. Martin, of the H.S.P.A. Experiment Station, has informed the writer[7] that the POJ-36 cane variety is highly resistant to the mosaic virus strain occurring in Hawaii, but it is not resistant to the common mosaic strain of Louisiana. Both strains are transmitted by *Aphis maidis.* It would be easily possible for infective aphids to reach Hawaii alive by means of fast planes and, if allowed to escape, they could introduce the more virulent mosaic strain into Hawaiian cane fields where further dissemination could be accomplished by the *Aphis maidis* already established here. The fact that cane varieties which are resistant to some of the other mosaic strains have been developed, does not mitigate the danger. Such resistant varieties may not prove to be as satisfactory under Hawaiian conditions as are those now in use or in the process of development in Hawaii. Furthermore, converting large areas over to new varieties within a short period of time, would involve difficult problems and serious monetary losses. Such a sudden change might be necessary in the

[7] Personal communication.

event a virulent strain of the virus became established here. The rigid quarantine imposed in Hawaii on sugar cane cuttings of newly imported varieties has played a major part in preventing the introduction of new insects and diseases of this crop.

In most regions where sugar cane mosaic occurs, it has been possible to transmit the different virus strains by means of *Aphis maidis*. However, Storey[8] in 1936 announced that in Natal he found a cane variety, "Agaul," which showed typical mild mosaic symptoms, and which was assumed to harbor the prevalent mosaic virus. Attempts to transfer mosaic from "Agaul" by mechanical means or by *Aphis maidis* were unsuccessful. Furthermore, he found that there was no dissemination of the disease in the field in East Africa and concluded, "Either this mosaic pattern is a varietal character—a highly improbable conjecture—or the Agaul virus is different from the common sugar cane virus. This Agaul variety was imported to Natal from India . . ." The history of mosaic in India contains many variable results, according to Storey, including, at times, failure of transmission by *Aphis maidis*.

Sugar Cane Chlorotic Streak

This disease was first noted in Java in 1928. In 1930 Martin (131) reported its occurrence on several cane varieties in Hawaii. He gave the disease the name chlorotic streak and this name has become generally accepted throughout the world. Martin (135) reported transmission of the disease by means of hypodermic needle inoculation in one experiment. Negative results were obtained in all other trials. The disease can be killed in cane cuttings by subjecting them to hot water treatment for 20 minutes at 52° C. (132).

Martin (133) stated that the cause of the disease had not been determined, but that in many ways it resembled the virus type of disease more than the fungus or bacterial type. Holmes (96) included it in his handbook of viruses. Abbott and Sass (2) in 1945 stated that the demonstrated fact of insect transmission "gives strong indication that it is caused by a virus."

Host plants other than sugar cane have not been demonstrated, but Martin (132) reported that symptoms similar to those of chlorotic streak were found to occur on Job's tears, *Coix lachryma-jobi*, and on elephant grass, *Pennisetum purpureum*, which are often found growing in close proximity to sugar cane.

Insect transmission. Martin (132, 133) in 1936 and 1938 reported that no positive results had been secured in Hawaii from experiments conducted to determine the identity of an insect vector of chlorotic streak. The following species were used in these experiments: the cane aphid, *Aphis sacchari;* the corn leaf aphid, *Aphis maidis;* the sugar cane leafhopper, *Perkinsiella saccharicida;* the

[8] Storey, H. H. 1936. Proc. Fifth Congr. Internatl. Soc. Sugar Cane Technol. (Brisbane) 1935 : 108-116.

green sharpshooter, *Draeculacephala mollipes;* the pink sugar cane mealybug, *Trionymus sacchari* (Ckll.); the onion thrips, *Thrips tabaci;* and the sugar cane stalk mite, *Tarsonemus spinipes* Hirst.

In 1942 Abbott and Ingram (1) reported transmission of chlorotic streak by means of the leafhopper *Draeculacephala portola* Ball in the southern United States. They obtained transmission to only 25 out of 490 plants tested. These results indicate that *D. portola* is a relatively inefficient vector or else that the experimental transmission was complicated by a factor which has not yet been determined. These authors pointed out that the genus *Draeculacephala* has been reported in Hawaii, Puerto Rico, Colombia and Louisiana where chlorotic streak is reported to spread under natural conditions, but not in Java where the disease was first described.

Until recently *D. portola,* which feeds on sugar cane in the Gulf States, has been misidentified as *D. mollipes.* True *mollipes* presumably occurs primarily in the northeastern United States and is not a sugar cane pest in Florida or Louisiana (1).

Martin (133) stated that natural spread of the disease occurs in certain regions in Hawaii. In both Hawaii and Australia, the disease is most prevalent in localities where rainfall is heavy. In view of this record of field spread we can assume that a vector of the disease probably is at large in Hawaii.

The report by Abbott and Ingram on transmission of chlorotic streak by *D. portola* and the confusion which has existed regarding the identity of some species in this genus are pertinent to the situation here. Until recent years the "sharpshooter" in Hawaii, which sometimes occurs on sugar cane, was identified as *D. mollipes* (247). Recently[9] it has been announced that both *D. minerva* and *D. mollipes* occur here; *minerva* may be the more common of the two species. In view of the results obtained by Abbott and Ingram in Louisiana it is probable that insect transmission of chlorotic streak in Hawaii can be demonstrated by using the *Draeculacephala* species we have here, even though *D. portola* is not one of them. This premise is supported by the fact that phylogenetic relationships are known to exist among the vectors of several virus diseases. Frazier (67) in 1944 reported nine species of leafhopper vectors for Pierce's vine disease in California. All nine species occur in the Amblycephalinae, a subfamily in which every species (including *D. minerva*) thus far tested has proven to be a vector. At the same time, many species of leafhoppers in other subfamilies have been tested and not one has been found to transmit the grape disease. Similar vector-virus relationships occur within the genera *Cicadulina* and *Perkinsiella* which include vectors of maize streak disease and Fiji disease of sugar cane, respectively. The three known species of vectors of maize streak all occur within the genus *Cicadulina. Perkinsiella* includes two species which have

[9] See footnote, p. 471.

been shown to transmit Fiji disease. In addition, Mungomery and Bell (150) and Pemberton (185) have pointed out that a third species must be the vector in Fiji. Furthermore, others of the 21 species listed by Pemberton (185) as occurring in the genus are probably vectors in other localities.

In view of the low percentage of chlorotic streak transmission obtained by means of *D. portola* in Louisiana it is probable that long series of experiments would have to be run with the main suspect species before negative evidence could eliminate them from the suspect group.

Cucumber Mosaic

Two of the cucumber mosaic strains discussed below are known in the literature by several different names. They are:

1. Cucumber mosaic virus, typical strain, (*Marmor cucumeris* var. *vulgare* Holmes).
   Synonyms: Common cucumber mosaic virus, Cucumber virus 1; Cucumis virus 1; Spinach blight virus.
2. Cucumber mosaic virus, southern celery mosaic strain (*Marmor cucumeris* var. *commelinae* Holmes).
   Synonyms: Southern celery mosaic virus; Celery virus 1.

The properties of the cucumber mosaic virus occurring in Hawaii, and its symptoms on various hosts have not been studied. It has been assumed that we have the typical strain or common cucumber mosaic. This is probably true in view of the fact that we also have a spinach blight which agrees in general with the description of the spinach blight on the mainland known to be due to common cucumber mosaic. In addition we have in Hawaii "fern leaf" of tomato, a disease induced by common cucumber mosaic virus in tomato.

It is now apparent, however, that we have more than one variety or strain of cucumber mosaic virus in Hawaii. Although it has not been definitely proved, there is evidence that the southern celery mosaic strain of cucumber mosaic, or a related strain, occurs in Hawaii. This virus was studied by Doolittle and Wellman (53, 239, 240, 241, 242, 243, 244). It was responsible for severe losses in the celery growing areas of Florida and was found to spread to celery from *Commelina nudiflora* which grows around the fields as a weed. When first investigated, this disease was assumed to be common cucumber mosaic. However, Wellman (240) in 1934, after a comparison of the symptoms on various hosts with symptoms produced by other viruses, concluded that the virus infecting celery and *Commelina nudiflora* in Florida was distinct from the strain known as common cucumber mosaic. He gave it the name southern celery mosaic (Celery virus 1). Part of the evidence Wellman presented as a basis for his conclusions was the fact that southern celery mosaic was transmissible to watermelons which,

he stated, are immune to common cucumber mosaic virus. In addition, *Commelina nudiflora* has not been found susceptible to common cucumber mosaic whereas it is the most important weed host of southern celery mosaic. The differential susceptibility to strains of cucumber mosaic virus shown by watermelon and *Commelina* has a bearing on the identity of the virus strains occurring in Hawaii. Parris (175) in 1936 recorded mosaic on watermelon (*Citrullus vulgaris*) in Hawaii. If this mosaic was caused by one of the cucumber mosaic viruses, it is probably one more closely related to southern celery mosaic than to common cucumber mosaic in the light of Wellman's report (240) that watermelon is immune to the latter virus strain. The few records of watermelon mosaic referred to by Walker (234) in 1933 were listed as being due to cucumber mosaic without differentiation of strains. *Commelina nudiflora* has been found to be the main weed host of southern celery mosaic virus in Florida. What has been considered to be the same plant, *Commelina diffusa* Burm. (= *nudiflora* of Hawaiian authors), in Hawaii was observed by Kunkel (114) in 1921 and Hadden (84) in 1928 to carry a mosaic disease. In 1935 Carter (34) transmitted Commelina mosaic to pineapple by mechanical inoculation, and in 1937 he (36) transmitted it to pineapple by means of *Aphis gossypii, Myzus persicae,* and *Macrosiphum solanifolii.* Southern celery mosaic has been reported to be transmitted by the same species of aphids and in addition by *Aphis maidis.*

According to Price (192) the mosaic symptoms produced in the Hawaiian *Commelina diffusa* are similar to those produced by southern celery mosaic in *nudiflora* of Florida. He concluded that the viruses are probably related but that further proof is needed. In 1941 Price (192) reported the results of studies he conducted with a yellow strain and a green strain of mosaic in *Commelina diffusa* from Hawaii which he received through Dr. Carter. He found that these strains, though closely related, were not equal in their ability to infect dicotyledonous hosts when inoculated mechanically. Moreover, the symptoms produced on species of *Nicotiana, Cucumis sativus* L. and *Zinnia elegans* by the yellow and green strains of Hawaiian Commelina mosaic were similar to, but distinguishable from, those of both common cucumber mosaic and the southern celery mosaic strain of cucumber mosaic virus on the same hosts. Since the symptoms of Commelina mosaic in Hawaii were reported to vary from a brilliant yellow mottling to a mild green mottling, Price recommended referring to these strains as yellow and green variants of cucumber mosaic virus pending more comprehensive study of them under local Hawaiian conditions.

In view of the circumstances presented above, both the common cucumber mosaic virus (*Marmor cucumeris* var. *vulgare*) and the cucumber mosaic virus, southern celery-mosaic strain (*Marmor cucumeris* var. *commelinae*) will be treated in this paper.

Cucumber Mosaic Virus, Typical Strain
(*Marmor cucumeris* var. *vulgare*)

Synonyms: Cucumber virus 1; Cucumis virus 1; Common cucumber mosaic virus; Spinach blight virus; Tomato "fern leaf" virus; possibly also banana bunchy top virus.

Symptoms: This virus frequently causes yellowish green systemic mottling on cucumber. The plants become stunted, the leaves are small and may be curled or otherwise distorted. Fruits may also be mottled and misshapen (96). Parris (177) stated that the most dependable symptom is the bunching of the leaves at the tips of the vines.

Host plants. The host plant range of this virus is very wide. Holmes (96) listed 34 families of plants in which susceptible species occur. Thirty-two species are found in Cucurbitaceae alone. Among common cultivated plants listed by Holmes (96) as sometimes seriously affected are, cucumber, celery, spinach, tobacco, pepper and tomato. In spinach the cucumber mosaic virus causes "spinach blight" (see "Spinach blight") and in tomato it causes "fern leaf." Parris (177) listed the following plants in Hawaii as susceptible to cucumber mosaic virus: *Beta vulgaris* (beet), *Spinacea oleracea* (spinach), *Citrullus vulgaris* (watermelon), *Cucumis melo* (muskmelon), *C. sativus* (cucumber), *Cucurbita pepo* (pumpkin), *Nicandra physaloides* (apple of Peru), *Petunia hybrida* (petunia), *Solanum carolinense* (horsenettle), *S. nigrum* (nightshade) and *Viola* sp. (violet). On the basis of the list of susceptible hosts reported by Holmes, the group listed by Parris represents only a small portion of the number in Hawaii which should be susceptible hosts.

In addition to the diseases reported above caused by cucumber mosaic virus, Holmes (96) stated that this may also be the virus which causes bunchy top disease of bananas in Australia.

Insect transmission. Doolittle and Walker (51, 52) in 1920 and 1925 demonstrated transmission of this virus by means of *Diabrotica vittata, D. duodecimpunctata* and *Aphis gossypii.* Hoggan (88, 89, 90) in 1929 and 1930 transmitted cucumber mosaic virus by means of *Myzus persicae, Myzus pseudosolani* (*M. pseudosolani* probably = *M. convolvuli*), *Myzus circumflexus* and *Macrosiphum solanifolii.*

No studies have been reported on the transmission of common cucumber mosaic in Hawaii by means of insects. However, this is one of our most common virus diseases and it consistently takes a heavy toll of many plantings in Hawaii. Frequently the majority of cucumber plants in a field become infected before the crop has matured. Since the melon aphid, *Aphis gossypii,* is by far the most common aphid on cucurbits in Hawaii, it is probable that most of the spread in nature is due to this species. *A. gossypii* is also one

of our most common aphids on a large number of non-cucurbit host plants. It is thus able to maintain a relatively high population in nature during most of the year with the result that new plantings of cucurbits are seldom able to remain free from infestation for more than a short time.

There are varieties of cucumber which are resistant to cucumber mosaic, but their adaptability to Hawaiian conditions has not yet been adequately investigated and the varieties commonly planted here are susceptible to the disease.

Cucumber Mosaic Virus, Southern Celery Mosaic Strain (*Marmor cucumeris* var. *commelinae*)

Synonyms: Celery virus 1, celery mosaic virus.

Host plants. Holmes (96) discussed the susceptible species as follows: "*Commelina nudiflora* L.; *Apium graveolens* L., celery; *Capsicum frutescens* L., pepper; *Musa cavendishii* Lamb and *M. sapientum* L., banana; *Zea mays* L., corn; and many other vegetables, grasses, and weeds, altogether 91 species in 23 families. So far as known, the host range of this strain and of the type strain are alike." Although the host range of this strain and the type strain are alike in general, the susceptible and insusceptible species tested are not the same for both strains as was demonstrated by Wellman (240) in 1934, and discussed earlier in this paper.

Disease symptoms. In *Commelina nudiflora,* celery and peppers the leaves become mottled and in the case of the latter two plants stunting and distortion of the plant occurs (96). In banana, the new leaves are stunted and often streaked with yellowish and green regions which frequently become necrotic. The stunted plants acquire a rosette-like appearance which in some respects resembles bunchy top of banana (241).

In corn, (*Zea mays*) Wellman (242) reported "the celery virus causes a stripe disease of maize in Florida very similar to but not identical with the white stripe of maize in Cuba. After numerous experiments it was evident that the celery-virus stripe on maize in Florida is transmitted by aphids but not by *Peregrinus maidis,* which Stahl (217) found was the vector of Cuban white stripe."

Mosaic diseases in banana and corn, in many respects resembling those reported above, occur in Hawaii and are discussed more fully under "Banana Mosaic" and "Corn Mosaic."

Insect transmission. Doolittle and Wellman (53) in 1934 reported transmission of southern celery mosaic by means of *Aphis gossypii.* In the same year Wellman (241) announced transmission of this virus to 2 banana plants by means of *Aphis maidis* and to 13 of 15 Lady Finger banana plants exposed to viruliferous *Aphis gossypii.* Although both test plants exposed to *Aphis maidis* contracted the disease, it would be desirable to test this aphid on a much longer series of plants in order to confirm its ability as a

vector of this virus and eliminate the possibility that transmission may have been due to the presence of more than one aphid species in the test population. This precaution is particularly appropriate for *Aphis maidis* because this species has not been found to be as versatile a vector of plant viruses as are some species of aphids. Only two other virus diseases (sugar cane mosaic and onion yellow dwarf) have been shown to be transmissible by *Aphis maidis*.

In addition to the aphid vectors of southern celery mosaic virus listed above, Carter (36) in 1937 reported he had received a personal communication from Wellman stating that *Myzus persicae* and *Macrosiphum solanifolii* are also vectors of this virus.

Holmes (96) listed *Pentalonia nigronervosa* as a vector of southern celery mosaic. This species has not been demonstrated to transmit the virus in the United States. Its inclusion by Holmes apparently results from the fact that southern celery mosaic was shown by Wellman (241) to be transmissible to banana in which it produced symptoms similar to bunchy top of banana, a disease known to be transmitted by *Pentalonia nigronervosa* in Australia. However, that these diseases are identical has not as yet been demonstrated.

Commelina Mosaic

The virus causing common mosaic in *Commelina diffusa* in Hawaii is a strain of cucumber mosaic. Carter (36) reported transmission of this virus to pineapple by means of *Aphis gossypii, Myzus persicae* and *Macrosiphum solanifolii*. (For a more complete discussion of Commelina mosaic see "Cucumber Mosaic," p. 567.)

Lily Mosaic

The only reference encountered in the literature to lily mosaic in Hawaii is one by Parris (175), who in 1936, listed a mosaic on *Lilium longiflorum*.

The identity, host and vector relationships are not known for the lily mosaic reported from Hawaii. Until recent years it was believed that the lily virus diseases (lily mosaic and yellow flat) were distinct from similar diseases occurring in tulips and some other plants. In 1935 Wellman (244) found lily to be among the plants susceptible to southern celery mosaic virus. Price (191) in 1937 found that leaves of zinnia plants infected with a strain of lily mosaic virus were immune from infection with cucumber mosaic virus. On the basis of these tests and the similarity of cucumber mosaic symptoms in lily to those of lily mosaic in lily, he concluded that lily mosaic virus should be classified in the cucumber mosaic virus group. The fact that *Aphis gossypii* was known to be vector of both lily mosaic (83) and southern celery mosaic (240) contributed to this conclusion. Another lily disease, known as yellow

flat, was considered by Price (191) to be due to a virus distinct from that causing lily mosaic.

In accordance with the conclusions of Price on the relationships of lily mosaic, Smith (215) placed lily mosaic under Cucumis virus 1. Holmes (96) in his classification of viruses erected the name cucumber mosaic virus, lily mosaic strain (*Marmor cucumeris* var. *lilii*) to include part of the lily mosaic complex of viruses.

McWhorter (143) in 1935 stated that the virus which causes "breaking" (color-adding and color-removing) in tulips is transmissible to lilies in which it produces a disease indistinguishable from lily mosaic.

Brierley and Smith (28) in 1944 reported the results of experiments in the United States on the properties, host range and vector relations of three lily mottle viruses, (which they assigned to the tulip virus group) as well as comparative data on tulip viruses 1 (color removing) and 2 (color adding) of McWhorter (144). Unfortunately the extensive investigations reported by Brierley and Smith do not include comparison of the viruses in question with the mosaic induced in lily by cucumber mosaic virus as reported by Price (191). Most of the host studies were in *Lilium*. They concluded that the five viruses studied (coarse mottle virus, virulent coarse mottle virus, latent virus, tulip virus 1 and tulip virus 2) might justify designation as separate virus entities. However, to avoid confusion in nomenclature they placed them all in *Marmor tulipae* of Holmes which must then be amended to include color-removing as well as color-adding virus. Brierley and Smith grouped them as three subspecies: tulip color-removing subspecies, tulip color-adding subspecies and lily mottle subspecies. As thus defined, *Marmor tulipae* was found by Brierley and Smith (28) to be transmissible by sap and by *Aphis gossypii, Macrosiphum solanifolii* and *Myzus persicae*. In addition *"Aphis fabae"*[10] was found to transmit the tulip color-adding and tulip color-removing strains. The previously reported additional vectors of these latter strains were *Anuraphis tulipae* (B. de Fonsc.) (141) *Macrosiphum solanifolii* and *Myzus persicae* (139, 140).

Banana Mosaic

In 1921 Kunkel (114) mentioned a mosaic on banana in Hawaii. No further reference to a banana virus here has been recorded in the literature. On October 3, 1945 the author noted what appeared to be virus symptoms of a mosaic type on the leaves of several young Chinese banana plants (*Musa cavendishii*) at Waipahu,

[10] The identity of the species in question is uncertain. Smith (215) in 1937 listed *Aphis fabae* Scop. as a synonym of *Aphis rumicis* Linn. Jones (109) in 1942 stated that *fabae* and *rumicis* are distinct species and can be distinguished easily on morphological grounds. He also concluded that in England *fabae* is a polyphagous species but that *rumicis* colonizes only on plants belonging to the genus *Rumex*. In a letter to the writer dated January 4, 1946, Professor E. O. Essig stated that the occurrence of *fabae* in the United States has not been definitely established.

Oahu. The oldest leaves appeared to be normal but the youngest four to six leaves were streaked with light and dark green areas in a linear pattern along the secondary veins of the lamina. As the leaves became older the symptoms appeared to become more conspicuous and there was a tendency for the chlorotic streaks to become necrotic. Marginal chlorosis occurred on some leaves to a slight extent. The diseased plants were stunted and the more mature affected leaves drooped to the ground.

Considerable variation occurred in the chlorotic and light green streaks which occurred on the leaves. The streaks on some leaves were narrow while on others larger portions of the lamina were light green to yellowish in color and appeared as a mottle or blotch or as irregular, longitudinal chlorotic areas. The light green streaks were usually not continuous, but were interspersed with areas of darker green tissue. Occasionally yellowish areas appeared diamond shaped on the lamina. Sometimes such areas had a green center and in other instances the entire affected area was yellow or light green.

On October 10, 1945, in company with Dr. F. G. Holdaway and Mr. J. W. Hendrix of the University of Hawaii, the writer found, on the Campbell banana plantation at Mokuleia, Oahu, a single half grown banana plant which exhibited mosaic symptoms such as described above. Symptoms occurred only on the two youngest leaves. There is some evidence that within the last two years Mr. Campbell removed a block of several hundred plants which were similarly affected. On October 14, 1945 the writer noted several young banana plants at Laie which showed streaking and chlorosis similar to that found at Waipahu. On October 23, 1945 Mr. J. W. Hendrix and the writer examined a 2-acre planting of bananas in Manoa Valley in which approximately 10 per cent of the plants showed mosaic symptoms. The symptoms were again found to vary considerably in their pattern, ranging from relatively mild mosaic symptoms of interspered light and dark green areas, to conspicuous elongate streaks and mottles which appeared to become necrotic on some of the older leaves. These streaks or chlorotic patches also occasionally appeared diamond shaped. Enough plants, both healthy and infected, were present to see the effects the disease may have on a commercial planting. Partial strangulation of the new leaves in the center of the pseudo-stem with resultant malformation of the leaves was noted in some plants. Most of the diseased plants were found to be stunted. Those which apparently became infected while very young had made practically no growth. Young suckers being produced from diseased stools were also diseased in most instances.

The writer visited this planting again November 25, 1945. Healthy plants had produced near-mature bunches of fruits, but

most of the diseased plants were still dwarfed and making little growth. During this visit it was noted that considerable mosaic-diseased *Commelina diffusa* was growing in the vicinity of the field. Furthermore the diamond shaped mosaic symptoms on many of the leaves of *Commelina* closely resembled the symptom pattern on some of the affected banana leaves.

In view of Kunkel's report of banana mosaic in 1921 and a report by Mr. Carpenter (quoted below) that a banana mosaic has been noted sporadically in the intervening years, it is probable that this disease ordinarily does not spread with great rapidity or regularity within banana plantings. However, it is possible that at times severe damage may have been produced in localized areas without being brought to the attention of the public.

The report by Wellman (241) of transmission in Florida of the southern celery mosaic strain of cucumber mosaic from *Commelina nudiflora* to banana by means of *Aphis gossypii* and *Aphis maidis* is of particular interest. The symptoms produced in banana by this virus included stunting of the leaves and of the plant as a whole, and chlorotic streaks on the leaves. In some instances the streaks became necrotic. The plants were reported to have acquired a rosette-like appearance which resembled bunchy top of bananas in Australia. The stunting of the plants and the streaking and necrosis of the leaves resemble symptoms observed on banana in Hawaii.

In this connection a report by Mr. C. W. Carpenter of the Pathology Department of the Hawaiian Sugar Planters' Association Experiment Station is very pertinent. In a letter dated October 23, 1945 addressed to the writer Mr. Carpenter stated: "A mosaic of the Chinese banana is not uncommon on Oahu; about 10 years ago the banana mosaic was very prevalent at Mokuleia after cucumbers were interplanted. The latter were seriously affected with mosaic."

The above reports by Wellman and Carpenter, when considered with the report by Price (192) that Hawaiian *Commelina* mosaic is very closely related to both common cucumber mosaic and southern celery mosaic, suggest the possibility that Hawaiian banana mosaic may be due to one or more strains of cucumber mosaic which occur in *Commelina,* cucumber, and probably other host plants in Hawaii.

Although the banana mosaic observed in Hawaii has not been demonstrated to be a virus, the symptoms strongly suggest that such is the case. Furthermore, similar symptoms in banana in other parts of the world have been shown by experimental transmission to be due to viruses. If mosaic in Hawaii proves to be a virus its identity and relationship to other similar viruses should be determined. The symptoms observed in Hawaii resemble in some respects several of the symptoms described for other virus-induced

banana diseases. These diseases (Banana heart-rot, infectious chlorosis, and bunchy top) will be discussed briefly although they are not known to occur in Hawaii.

Banana heart-rot or infectious chlorosis (due to Musa virus 3 of Smith). This disease has not been extensively studied. However, Magee (129) in 1930 reported its occurrence in New South Wales and thought it was transmitted by the banana aphid, *Pentalonia nigronervosa.* This disease causes yellowing and mottling of the foliage throughout the year and rotting of the heart leaf and central portion of the pseudo-stem during the winter. The rotting extends down into the corms and usually kills the plants. The yellowing symptoms are variable, but usually take the form of continuous or discontinuous streaks extending from the midrib to the margin of the leaf. These streaks vary in width from very narrow to more than ½ inch. The leaf symptoms resemble, in a general way, those found on diseased plants in Hawaii. A heart-rot stage in Hawaii has not yet been observed. This disease was not reported as being widespread in New South Wales, but Magee stated that if it were to become widespread it might be a greater threat to the banana industry than banana bunchy top.

Brazilian infectious chlorosis of banana. Wardlaw (236) in 1935 discussed a type of infectious chlorosis observed on banana in the state of Sao Paulo, Brazil. This he considered closely related to the banana heart-rot disease of Australia. The leaf symptoms were described as "light chlorotic linear areas extending as irregular, broken, sometimes anastomosing, lines or stripes from the midrib to the leaf margin." In contrast to the symptoms observed in Hawaii, the leaf mottle of the Brazilian disease tends to be masked on the older leaves. The disease has been shown to be systemic and is transmitted to the daughter suckers. This disease apparently does not injure the plant and diseased stools produce normal bunches year after year, except that mosaic mottling may occur on the fruit skin. No plants have been found in which rotting of the central leaf and the core of the pseudo-stem occurred as reported for the heart-rot disease in Australia.

The leaf symptoms of the disease in Brazil vary with environmental conditions. Mottling may fade noticeably or disappear completely from plants making rapid growth as a result of hot, wet weather or fertilizing. During the cold dry season when little growth takes place the leaf mottle develops maximum intensity.

Wardlaw (236) reported experiments in which the banana aphid, *Pentalonia nigronervosa,* failed to transmit the disease.

A disease of bananas, believed by Wardlaw (236) to be the same as that reported from Brazil, has also been observed in Trinidad, West Indies, for several years. A similar disease of banana reported by Ogilvie (162) from Bermuda in 1928 is also believed by

Wardlaw (236) to be probably the same as the disease reported from Brazil. In Bermuda, however, the disease has a slight dwarfing effect on the plants.

Banana Bunchy Top

Magee (128) in 1927 reported that since its appearance in Australia in 1913 bunchy top disease has ruined the banana industry in several areas. It has also caused considerable damage in Fiji (where it is believed to have originated), and in Ceylon and Egypt (128).

The general symptoms of the disease are described by Magee (128). "The leaves of a badly infected plant are bunched together at the apex of the plant to form a rosette. Owing to the failure of the leaf stalks to elongate, the leaves stand more erect than normal. Infected plants are markedly stunted, there being little growth in height once the plant has taken the disease."

The first definite symptoms of the disease appear in the newly unfurled center leaf on which irregular, nodular, dark green streaks about 0.75 mm. wide appear along the secondary veins of the leaf lamina. Leaves produced subsequently may show whitish streaks along the secondary veins of the leaf blade before the leaf has unfurled. When these leaves unroll they bear dark green streaks along the secondary veins and dots or lines on the midrib and petiole. The leaves may also be somewhat stunted, slightly chlorotic, and the margins of the lamina may be wavy and rolled upwards slightly.

Due to strangulation or leaf congestion at the apex of the pseudostem, diseased plants often fail to throw the bunch or it may get partly out and be left standing erect or at right angles to the pseudo-stem. The fruit of such bunches is usually stunted.

Magee (128) transmitted this virus by means of the banana aphid, *Pentalonia nigronervosa.* Plants infected by means of this aphid developed primary symptoms in an average of 25 days.

Banana bunchy top virus was reported by Magee (128) to be transmissible to abacá or Manila hemp (*Musa textilis*) by means of *Pentalonia nigronervosa.* The symptoms reported by Magee in Manila hemp differ somewhat from those reported by Ocfemia for this disease in the Philippine Islands. Furthermore, the virus in the Philippines has not been found to be transmissible by *Pentalonia nigronervosa* from Manila hemp to banana. Of unusual interest is the report by Ocfemia and Buhay (160) in 1934 that in transmitting the virus from abacá to abacá in the Philippines, *Pentalonia nigronervosa* must feed on the inoculum for 12 hours in order to acquire the virus. In addition to this time the virus requires an incubation period of from 24 to 48 hours in the aphid vector before it can be transmitted to a healthy plant.

Papaya Mosaic

Parris (178, 182, 184) in 1939, 1940 and 1941 discussed a new disease of papaya which was discovered at Waialua and Lualualei, Oahu, in July 1937. In 1939 Parris (179) reported mechanical transmission of the disease to healthy plants in over 75 per cent of the tests conducted. He concluded that a virus was probably the causative agent. During the first 2 years after the discovery of the disease in Hawaii, papaya mosaic caused losses ranging from 6 to over 30 per cent in several large plantings (179). However, since 1939 diseased plants have been observed only occasionally.

Symptoms of the disease were described by Parris (179). Diseased plants are stunted and the foliage is yellowed. The petioles of diseased leaves are bent downwards at their point of attachment to the stem. "Linear, darker-green than normal, slightly raised, hydrotic-like streaks may be present on any portions of the main stem of a diseased plant, and on the petioles of yellowed leaves. . . . The streaks vary from ⅛ to 1 inch in length and from 1/32 to ⅜ inch in width. . . . Diseased leaves abscise rapidly, and 4 to 6 weeks after initial symptoms only a few badly distorted and undersized leaves remain clustered at the top. The leaves developed prior to symptom expression persist as a fringe around the base of the plant."

Parris also reported that "When a diseased papaya plant is decapitated or death of the growing point takes place naturally, new growth develops from the lower portions of the stem. It has been observed that the new shoots may all be healthy, or some be healthy and some diseased."

Holdaway and Look (95) in 1940 reported negative results from preliminary experiments conducted to determine an insect vector of papaya mosaic. The species tested included: *Aphis gossypii, A. medicaginis, A. maidis, Macrosiphum gei* (=*solanifolii*), *Myzus persicae, Empoasca solana* DeLong, *Nysius coenosulus* Stål, and *Thrips tabaci.*

Virus diseases of papaya have been reported from other parts of the world. Parris (178) considered it possible that the mosaic of Hawaii is identical with a disease in Queensland called "yellow crinkle" by Simmonds (208).

The mosaic of papaya in Hawaii is apparently distinct from the papaya mosaic reported from China by Ho and Li (87) in 1936.

Papaya Ringspot

Lindner *et al.* (123) in 1945 reported the presence in Hawaii of a previously undescribed papaya disease which appeared to be due to a virus. This disease, designated as papaya ringspot, was discovered by Dr. R. C. Lindner at Kailua, Oahu, in March 1945.

The disease is characterized by a mottle or mosaic pattern in the younger leaves resulting from irregular areas of leaf tissue

which are of a lighter green color than that of the rest of the leaf. The mosaic symptoms in the foliage are usually very conspicuous during the winter months, but mild during the summer. Diseased plants show retardation of growth and are weakened and killed more readily by adverse environmental conditions than are normal plants.

The most striking and reliable symptom of papaya ringspot disease appears in the fruits as they reach the ripening stage. Yellow rings with green centers are produced on the fruit surface. These rings vary in size from ⅛ to ¾ of an inch in diameter and may be circular or irregular in outline. The number of rings on a single fruit varies from a few to over 150.

Experiments conducted by the writer at Honolulu demonstrate that *Myzus persicae* is a vector of the ringspot virus. The disease was transmitted to 55 of 90 test plants exposed to viruliferous aphids between April 1 and July 31, 1945. After inoculation, symptoms developed in rapidly growing papayas in from 11 to 21 days.

Crucifer Virus Diseases

The relationships of some of the virus diseases which are found infecting cruciferous plants have not yet been adequately elucidated. Research work reported during recent years indicates that several viruses or virus strains are involved.

The identity, properties, host range and manner of dissemination of the virus diseases which occur in cruciferous plants in Hawaii are at present unknown. Kunkel (117) in 1924 was the first to report mosaic on Chinese cabbage, *Brassica pekinensis* (Lour.). Parris (177) in 1938 described the general types of symptoms which occur on some cruciferous plants here and cited the work done by Schultz (202) in 1921 as the source of his information on physical properties and manner of transmission. Parris (177) stated that mosaic had been seen in Hawaii on mustard, radish, Swiss chard, Chinese cabbage and turnip. In the same year he recorded (176) mosaic on daikon (*Rhaphanus* sp.) in Hawaii. Martin (138) in 1943 also reported mosaic on daikon in Hawaii. Frazier (68) reported mosaic as being among the most serious diseases of spoon cabbage and green mustard in Hawaii.

During recent years investigations have shown that several different virus diseases occur in cruciferous plants, and that most of these viruses have several plant hosts in common. The collective term, crucifer mosaic, therefore, loses meaning when the host range, symptoms, properties and methods of transmission are studied. Hoggan and Johnson (94) in 1935 described a crucifer virus transmissible by *Brevicoryne brassicae* and *Myzus persicae*. Tompkins (228) in 1937 described a mosaic disease of cauliflower which he considered distinct from the other diseases which had previously been reported from crucifers. He reported transmission by means

of the aphids *Brevicoryne brassicae, Rhopalosiphum pseudobrassicae* and *Myzus persicae*. "The host range included 51 vegetable varieties, 3 ornamentals and 5 weeds, all belonging to the family Cruciferae." In discussing Tompkins' cauliflower mosaic virus (Brassica virus 3 of Smith) Smith (215) in 1937 stated that Severin had informed him that this disease had been transmitted experimentally by the following aphids:

Species already occurring in Hawaii:

Brevicoryne brassicae—Cabbage aphid
Rhopalosiphum pseudobrassicae—False cabbage aphid
Myzus persicae—Green peach aphid
Myzus circumflexus—Lily aphid
Aphis gossypii—Melon aphid
Aphis middletonii—Erigeron root aphid
Cavariella aegopodii (= *capreae* Fab.)—Yellow willow aphid

Species not recorded from Hawaii:

Aphis graveolens Essig—Celery leaf aphid
Aphis apigraveolens Theob.—Celery aphid
Rhopalosiphum melliferum Hottes—Honeysuckle aphid

Tompkins (229) in 1938 described a mosaic of turnip and other plants which was readily transmitted by *Myzus persicae* and *Brevicoryne brassicae*. In the same year Tompkins and Thomas (232) described a mosaic disease of Chinese cabbage (*Brassica pe-tsai*) which was prevalent in central California. The host range of this virus included cauliflower, turnip, cabbage, radish, and mustard. The insect vectors were found to be the same as for turnip mosaic. In 1938 Tompkins, *et al.* (231) described a virus disease of cabbage and other crucifers which they named black ring. Again the vectors were shown to be *Myzus persicae* and *Brevicoryne brassicae*.

In 1939 Larson and Walker (121) described a destructive mosaic disease of cabbage in Wisconsin. All cruciferous plants tested were found to be susceptible to the disease. In addition, Swiss chard and spinach were among the noncruciferous hosts determined. The virus was found to be transmissible by juice inoculation. Three insects, *Myzus persicae, Brevicoryne brassicae* and the cabbage worm *Pieris rapae* were shown to be vectors of the virus. The discovery of the cabbage worm as a vector of a virus is of singular interest because it is one of the few records of a lepidopterous larva transmitting a plant virus. It was found necessary for the aphid vectors reported to feed on a diseased plant for ½ hour in order to acquire the virus. They were then capable of retaining the virus long enough to infect four different healthy plants consecutively without a second feeding on the inoculum. The feeding period on each successive test plant was 1 hour. It is of interest that this

virus can be transmitted by mechanical means and by a chewing insect and yet requires at least ½ hour of feeding by the aphid vectors in order to acquire the virus. Furthermore, once infective, the aphids retain the ability to infect four consecutive healthy plants over a period of 4 hours.

Although the relationships of the crucifer viruses reported above need further clarification, the evidence is increasing that several different but related viruses occur in these plants. Tompkins and Thomas (232) stated in 1938 that on the basis of symptoms, Chinese cabbage mosaic, cauliflower mosaic and turnip mosaic can easily be differentiated on Chinese cabbage, winter colma cabbage and purple top white globe turnip.

Walker, *et al.* (235) and Pound, *et al.* (127) in 1945 showed that a strain of turnip virus 1 of Hoggan and Johnson (94) and a strain of cauliflower virus 1 of Tompkins (228) may occur together in cabbage with the characteristic symptoms of each being apparent and varying in intensity as the air temperatures vary. Turnip virus and cabbage black ring virus symptoms were found to increase in intensity with increasing air temperature. This was in contrast to the cauliflower virus group in which symptom severity increased as the air temperature was decreased. When a strain of turnip virus or a strain of black ring virus occurred in cabbage together with a strain of the cauliflower virus "the resulting disease reaction was more severe than that produced by either virus alone."

The need for research on crucifer viruses in Hawaii is obvious. At least one of these viruses occurs here and very possibly there are more than one. Most of the known insect vectors of crucifer viruses have already become established in the Territory. This fact increases the hazard which accompanies the inevitable introduction into Hawaii of other viruses not already established here.

Spinach Blight

Parris (177) in 1938 reported blight as a virus disease of spinach in Hawaii. Symptoms were described as including mosaic, mottling, yellowing and distortion of the leaves and stunting of the plant. The disease was reported by Parris to cause severe losses in Hawaii occasionally.

Although several viruses have been reported to induce disease symptoms in spinach, blight is usually considered to be due to cucumber mosaic virus. Hoggan (92) in 1933 discussed the symptoms produced in spinach by cucumber mosaic virus, sugar beet mosaic virus and tobacco ringspot virus. Hoggan's paper also reported transmission of cucumber mosaic virus to spinach by means of *Myzus persicae* and *Macrosiphum solanifolii.*

Parris cited Hoggan's paper as a reference but listed *Aphis rumicis* as a vector in addition to the two species recorded by Hoggan. The writer has failed to find any other report in the literature

of *Aphis rumicis* as a vector of spinach blight. The report by Parris may have been based upon the fact that Hoggan had transmitted sugar beet mosaic virus to spinach. *Aphis rumicis* is a vector of sugar beet mosaic but was not used in the transmission experiments reported by Hoggan. According to Hoggan, the symptoms of sugar beet mosaic in spinach are distinct from those produced by cucumber mosaic.

Lettuce Mosaic

Parris (177) in 1938 reported that a lettuce mosaic occurred in a high percentage of plantings in Hawaii and resulted in considerable loss. Symptoms of the disease reported from Hawaii include yellowish discoloration and a mosaic pattern of the leaves, rugosity of the leaf blade, and stunting of the plants. Mosaic prevents heading in head lettuce varieties.

Holmes (96) listed the following plant species as susceptible to lettuce mosaic virus (*Marmor lactucae*): Compositae—*Senecio vulgaris* L., groundsel; *Sonchus asper* Hoffm., prickly sow-thistle. Leguminosae—*Lathyrus odoratus* L., sweet pea, *Pisum sativum* L., pea.

Transmission. Ainsworth and Ogilvie (3) in 1939 reported that experiments conducted in England resulted in lettuce mosaic transmission by *Myzus persicae* to 15 out of 33 plants and by *Macrosiphum solanifolii* to 1 out of 23 plants. The latter species was reported as being the most common lettuce aphid in southwestern England. The virus has also been shown to be juice transmissible and in addition is one of the viruses that is carried through the seed of diseased plants.

No insect transmission studies have been reported from Hawaii.

Bean Mosaic

A bean mosaic virus (*Marmor phaseoli* of Holmes) has been shown to be transmissible to several species of *Phaseolus,* to *Vicia faba* L., *V. lathyroides* L. and *Lespedeza striata* Hook. and Arn. (79), all within the family Leguminosae. It is not transmissible to garden pea or sweet pea.

The symptoms of bean mosaic virus vary considerably depending on age and variety of the host and on environmental conditions. Holmes (79) states "first leaves to be affected are crinkled, stiff, chlorotic; later leaves show chlorotic mottling; leaf margins often rolled down."

This disease seriously reduces the yield of beans in many bean-growing areas. An unidentified bean mosaic occurs in Hawaii. Parris (177) discussed it briefly and its presence in Hawaii has also been noted by Martin (136, 137) in 1941 and 1942. According to a personal communication from members of the Departments of Vegetable Crops and Pathology, University of Hawaii Agricultural

Experiment Station, bean mosaic is of moderately common occurrence in Hawaii, but usually does not cause serious crop losses.

Insect vectors. The first record of bean mosaic transmission by means of insects was reported in 1922 by Nelson (153) who found that the potato aphid, *Macrosiphum solanifolii,* served as a vector. He failed to transmit the virus by means of *Empoasca fabae* (Harris). Elmer (58) in 1925 believed the mealy bug, *Pseudococcus maritimus,* was a vector, but this has not been well established although Fajardo (63) in 1930 reported transmission of bean mosaic by means of an undetermined species of mealy bug in addition to *Aphis rumicis, Myzus persicae* and *Macrosiphum solanifolii.* In 1936 Zaumeyer and Kearns (250) reported studies conducted in Colorado where they obtained transmission with 10 of 11 species of aphids tested and failed to transmit the virus by means of 10 species of insects other than aphids. The aphids found to transmit bean mosaic were: *Aphis gossypii, Aphis medicaginis, Aphis rumicis, Brevicoryne brassicae, Rhopalosiphum pseudobrassicae, Myzus persicae, Macrosiphum solanifolii, M. pisi* (Kalt.), *Aphis spiraecola* Patch, *Hyalopterus atriplicis* (Linn.) and *Macrosiphum ambrosiae* (Thomas). The first six species listed are known to occur in Hawaii at the present time.

The aphid which failed to transmit bean mosaic was *Neothomasia populicola* (Thos.) collected from poplar. With the exception of *Aphis medicaginis,* which infected 44 per cent of the plants on which it was tested, all of the demonstrated aphid vectors transmitted the disease to over 50 per cent of the test plants. *Rhopalosiphum pseudobrassicae* infected 100 per cent of the test plants upon which it fed.

Soybean Mosaic

Martin (136, 138) in 1941 and 1943 reported the occurrence of a soybean mosaic at Kailua and Waipio, Oahu. These are the only records of this disease in Hawaii and its identity is not known. A soybean mosaic was encountered in Indiana in 1920 (75). It was described as severely stunting and distorting the plants. The petioles and internodes were shortened, the leaflets were small, misshapen and puckered with dark green puffy areas along the veins. The pods were stunted, flattened and more sharply curved than normal. Diseased plants remained green longer in the season suggesting delayed maturity. The virus was transmitted mechanically and by seed but no insects were tested as vectors. The disease was confined largely to the side of a field near a planting of garden beans which were affected with a mosaic disease. This suggests the possibility that it may have been a bean virus which spread to soybean in this instance.

Smith (215) reported that soybean is susceptible to pea enation mosaic virus (= Pisum virus 1). The symptoms were described as

"a mottled dark and light green mosaic pattern." Zaumeyer (249) in 1938 listed soybean as a susceptible host of a pea streak virus which was carried by *Macrosiphum pisi*. However, the disease symptoms on soybean were not described.

Pea Mosaic

The exact identity of the pea mosaic which occurs in Hawaii has not been established, since no transmission experiments involving the disease have been reported from Hawaii. Parris (177) listed pea mosaic in Hawaii. His description of some of the mottle symptoms produced on garden pea (*Pisum sativum*) and sweet pea (*Lathyrus odoratus*) are characteristic of common pea mosaic virus (*Marmor leguminosarum* Holmes = Pisum virus 2 of Smith). However, the reference (166) cited by Parris regarding the vector applies to a distinct virus known as pea enation mosaic virus (*Marmor pisi* Holmes).

In addition to the two pea viruses mentioned above, there have been at least three mosaic diseases of pea described as new in recent years (249), and several virus-induced pea streaks (46, 249). Furthermore, pea is a susceptible host for several other virus diseases which were described originally from other plant species (especially in the Leguminosae) in which they are more commonly encountered in nature (124, 246, 251).

Chamberlain (44) in 1936 cited literature to the vectors of pea mosaic. According to this author the following vectors have been determined: *Macrosiphum pisi* (227), *Aphis rumicis* and *Rhopalosiphum viciae* (possibly = *A. rumicis*) (21), *R. fabae* (145), *Myzus persicae* (98) and *Macrosiphum solanifolii* (168). In New Zealand Chamberlain (44) transmitted a pea mosaic to several hosts by means of *Myzus persicae, Macrosiphum solanifolii,* and *Aphis rumicis.*

Osborn (169) in 1937 reported transmission of pea virus 2 (= pea mosaic virus, *Marmor leguminosarum* Holmes) by both nymphs and adults of *Macrosiphum pisi, M. solanifolii* and *Aphis rumicis.* Transmission was obtained by means of individual aphids as well as with larger numbers. These insects were shown to be able to acquire the virus during a feeding period of 5 minutes on diseased plants and were able to transmit the virus to healthy plants during a 5 minute period immediately following removal from the inoculum. None of the colonies retained the virus for more than 1 hour when fed continuously on a healthy plant. However, Osborn states, "when held without access to food, the bean aphid was shown to retain the virus for 5 hours, the pea aphid for 8 hours, and the potato aphid in one instance for 24 hours."

Pea Enation Mosaic Virus (*Marmor pisi* Holmes)

It is not known definitely that pea enation mosaic disease occurs

in Hawaii. However, since some of the early symptoms on some varieties may be confused with those produced by common pea mosaic, it is possible that the enation disease occurs here. In view of this fact and because of the unusual insect vector-virus relationships involved, this disease will be discussed briefly. In addition to chlorotic mottling, this virus sometimes produces necrotic spots and enations on the lower surface of the leaves. Holmes (96) states that the pods become distorted and, in broad bean, striping as well as chlorotic spotting may occur on the leaves. Transmission experiments could easily yield information as to which virus occurs in Hawaii if there is only one, since pea enation mosaic virus produces symptoms on the pea varieties Perfection and Horal which are resistant to common pea mosaic, *Marmor leguminosarum* (96).

Bawden (9) lists *Myzus persicae* as a vector of pea enation mosaic virus but does not cite a reference to the publication which reported this aphid as a vector of the virus in question.

Osborn (166, 167, 168) demonstrated that pea enation mosaic virus is transmitted by the pea aphid, *Macrosiphum pisi,* and the potato aphid, *M. solanifolii,* but not by *Aphis rumicis.* Both aphid species were unable to infect healthy plants until after a period of virus incubation in the vectors. The duration of this period varied considerably, but averaged about 12 hours for both species. In two colonies of the pea aphid, held at 80° to 90° F., the incubation period of the virus in the aphids was not less than 4 nor more than 10 hours. At the other extreme, one of the colonies held on plants in a greenhouse in which the temperature varied from 64° to 70° F. showed an incubation period of not less than 72 nor more than 96 hours.

Both the pea aphid and the potato aphid were shown to retain the virus for extended periods of time after becoming infected. Infective colonies of pea aphids, when transferred daily to a succession of healthy plants, retained the virus for 29 days. Single aphids were shown to retain the virus for 23 days. Potato aphids that acquired the virus from diseased broad bean, *Vicia faba,* retained the virus for as long as 21 days while feeding continuously on insusceptible tomato plants.

Since practically all viruses transmitted by aphids require no incubation period in the vector or but a very brief one, and since such viruses are lost by their vectors soon after feeding on healthy plants, the definite and sometimes extended incubation period of pea enation virus in the pea and potato aphids, and the ability of these aphids to retain the virus for long periods of time, is of great interest. A large number of aphid species have not been tested as possible vectors of this disease. However, in view of the vector-virus relationship demonstrated, it is probable that the number of species capable of transmitting this virus will be small.

Crotalaria Mosaic

Illingworth (97) in 1931 noted in a pineapple field in Hawaii, Crotalaria plants which showed marked mosaic and yellowing symptoms. "Leaf mosaic" of Crotalaria was listed by Martin (136) in 1941 from Kohala, Hawaii. The writer has observed a conspicuous mosaic on the same plant at Kailua, Oahu during 1945.

The identity of the Crotalaria mosaic in Hawaii is not known.

Johnson and Lefebvre (103) in 1938 reported a mosaic disease which appeared in the Crotalaria nursery at Arlington, Virginia. It was described as "a disease characterized by a general stunting of the plants, by mottling, blistering, and malformation of the leaves, and by abnormally stimulated lateral branching or witch's brooming." This virus was transmitted by juice inoculation to broad bean, *Vicia faba.* No transmission by insects was reported. These authors refer to a report published in 1927 indicating that a witch's broom disease of Crotalaria was found in Java which was considered to be of a virus nature and assumed to be spread by insects.

It is possible that Crotalaria mosaic virus is a strain of one of the cucumber, bean or pea viruses which include several legumes among their susceptible host species.

Peanut Rosette

Nothing is known of peanut rosette in Hawaii except that such a disease occurs here. Martin (136, 137) listed it as occurring at Kailua, Oahu in 1941 and in several places on Oahu in 1942.

Holmes (96) classified a peanut rosette virus as *Marmor arachidis.*

Storey and Bottomley (223) in 1928 reported that rosette is a destructive disease of peanuts in South Africa and that a similar disease occurs in tropical Africa, Java and India. It was reported to be transmissible by grafting but not through the seed or by mechanical inoculation. The plant may be severely altered by the virus due to chlorosis, stunting and malformation of the leaflets. Sometimes the entire plant may appear as a tuft of small curled leaves. The first evidence of infection was described by Smith (215) as a faint mottling of the youngest leaves. Plants showing distinct mosaic symptoms usually are not severely stunted.

Three distinct graft-transmissible strains of the virus were reported by Hayes (86) to exist in the Gambia. Chlorosis rosette caused the young leaves to be flaccid and develop yellow patches which later appeared as a light green mottle. Green rosette produced no chlorosis and the leaves were darker in color than normal. Rosette type No. 3 did not affect the normal color of the leaves but caused them to be smaller and thicker than normal. The stems were much thickened and each branch curved in a clockwise direction. Smith (215) considers it possible that Hayes' chlorosis rosette and green rosette may be due to two distinct viruses since both may

be put in the same plant without exhibiting any cross immunity. Hayes' report also indicated that the presence of weeds or close spacing of the plants resulted in less spread of rosette. The reason for this effect was not determined but it was suggested that with increased vegetative ground cover there was less evaporation from the soil resulting in more dew formation which may have affected the habits of the insect vector or the physiology of the plant.

Insect transmission of rosette was accomplished by means of *Aphis laburni* Kalt. (= *A. leguminosae* Theob.) in South Africa, but 13 species of leafhoppers failed to transmit the disease (221, 223).

Since the disease is reported to be non-transmissible through the seed or by juice inoculations, and since the only demonstrated vector, *Aphis laburni,* is not known to occur in Hawaii, it is probable that additional species are capable of transmitting the virus. In Hawaii *Aphis medicaginis* and *A. rumicis* would be logical suspects.

Peanut Mosaic

Martin (136) in 1941 recorded peanut mosaic from Kailua, Oahu where peanut rosette was also noted. No peanut mosaic has been reported in the literature as a distinct disease. However, Smith (215) pointed out that the first symptoms of rosette consist of a leaf mottle. Furthermore, some plants exhibit a pronounced mosaic pattern in the leaves and only a limited amount of stunting. In view of these circumstances it is possible that the mosaic of peanut recorded by Martin was caused by the peanut rosette virus complex. Nevertheless, it may also be that a mosaic-inducing virus exists which is distinct from the rosette virus. Diseases which are characterized by great diversity of symptoms in the same host plant frequently prove to be due to a complex of related or unrelated virus strains which may be transmitted in varying proportions at different times and under different conditions.

Pepper Mosaic

Parris (146) in 1941 and Martin (136, 137, 138) in 1941, 1942 and 1943 reported mosaic on pepper in Hawaii. Although pepper mosaic in the Territory has received only occasional and brief notation in the literature, it is a very common disease. During the past two years the writer has seen several plantings on Oahu in which approximately 50 per cent of the plants were infected.

The viruses responsible for pepper mosaic in Hawaii and their insect vectors have not been determined. This disease is usually attributed to tobacco mosaic virus which, according to Holmes (96), causes "yellowish primary lesions followed by systemic chlorotic mottling." Cucumber mosaic virus, southern celery mosaic strain, also causes symptoms in pepper which are described by

Holmes (96) as "mild mottling and grayish discoloration of leaves, malformation of fruit, stunting of plant." Symptoms of both general types have been observed in Hawaii.

Eggplant Mosaic

In 1942 Martin (137) reported "common mosaic" on eggplant at Ewa, Oahu. I have been informed by members of the Vegetable Crops Department of the University of Hawaii Agricultural Experiment Station that a mosaic on eggplant has been noted on Oahu on several occasions during recent years. The identity of this disease is not known. Eggplant (*Solanum melongena*) has been used in relatively few virus studies. Harter (85) in 1938 reported it susceptible to cucumber mosaic virus, lima-bean mosaic strain (*Marmor cucumeris* var. *phaseoli* of Holmes). This virus strain is transmitted by *Aphis gossypii* and *Myzus persicae*. Black (15) in 1938 listed eggplant as a susceptible host of potato yellow-dwarf virus, which is not known to occur in Hawaii. Holmes (96) lists eggplant as susceptible to tobacco mosaic virus, tomato spotted wilt virus and tobacco ringspot virus.

Tomato Fern Leaf

The disease in tomatoes known as fern leaf is caused by the presence in tomatoes of the common cucumber mosaic virus. This disease is characterized by stunting of the plant and particularly by reduction or absence of the lamina of the leaves leaving filiform or "shoestring" leaflets.

Smith (215) stated that these symptoms may be produced also by tobacco mosaic virus (tomato mosaic virus) under conditions of low temperature and especially of low light intensity. However, Mogendorff (147) in 1930 reported the results of extensive experiments on the fern leaf problem which do not agree with those reported by Smith. Mogendorff (147) stated that "typical fern-leaf symptoms could not be produced with the ordinary tobacco-mosaic or tomato-mosaic virus (Tobacco virus 1) under any of the environmental conditions to which the infected host was submitted." He found further that fern leaf was produced only irregularly on tomato by cucumber mosaic virus (Cucumber virus 1) if the infection had been accomplished by mechanical means. However, fern leaf could be produced regularly and at will if young tomato plants were infected with cucumber mosaic virus by means of the green peach aphid, *Myzus persicae*. The production of fern leaf symptoms was also found to depend upon (a) the atmospheric temperature and (b) the age of the plants at the time they are infected. The optimum atmospheric temperature for the expression of symptoms was found to lie between 18° and 22° C., with an approximate minimum temperature of 15° C. and an approximate maximum of 25° C. Moreover the tomato plants had to be infected while they were still small if typical fern leaf symptoms were to develop.

Although several different species of aphids have been demonstrated to transmit cucumber mosaic, reports of experiments specifically designed to study fern leaf in tomatoes have not been encountered in which aphids other than *Myzus persicae* were used as the vectors. The fact that Mogendorff found fern leaf could be induced readily by aphid transmission of the virus but only irregularly when the tomato plants were artificially inoculated, is of interest.

Parris (177) reported this disease as being very common in Hawaii. Members of the Vegetable Crops Department of the University of Hawaii Agricultural Experiment Station have informed the writer that the occurrence of tomato fern leaf in Hawaii has been found to be sporadic. The greatest amount appears during the winter months as would be expected in view of Mogendorff's findings regarding temperature and light.

Tobacco and Tomato Mosaic

Synonyms: Tobacco mosaic virus, tomato mosaic virus, pepper mosaic virus.

Mosaic has been known to occur in Hawaii since 1919 when it was recorded on tobacco (*Nicotiana tabacum*) by Carpenter (32).

This virus is of importance not only because of the economic losses which have resulted from the disease it causes, but also because it has figured so prominently in the research on the nature of viruses. The first published record of experimental transmission of a virus disease was by Iwanowski in 1892 who reported that sap of tobacco plants showing mosaic symptoms infected healthy plants after the sap had passed through a bacteria-proof filter.

Working with this same virus, Stanley (218) in 1935 succeeded in isolating and crystallizing the tobacco mosaic virus in what has been considered to be pure or nearly pure form.

There are several strains of tobacco mosaic virus which produce different symptoms in the tobacco plant. The strain which has received most investigation and which is of greatest economic importance is one which we speak of as common tobacco mosaic or tomato mosaic. This is an exceedingly infectious virus and can be transmitted to healthy plants in various ways such as by leaf contact, contaminated tools, clothing or other articles, and through the soil. It withstands drying in tobacco leaves for years without losing the ability to infect healthy plants.

Despite the highly infectious nature of tobacco mosaic virus, it has proved to be one of the most difficult of all viruses to transmit by means of insects. Cleveland (47) in 1931 listed *Thrips tabaci, Epitrix cucumeris* and *Tetranychus telarius* as probable (though unimportant) vectors of tomato mosaic virus. These conclusions were based on the abundance of the species in the field and on a limited number of vector tests. In view of present knowledge

concerning the transmission of this virus, it is highly improbable that these species actually are vectors. For many years it was assumed that aphids were largely responsible for the spread of tobacco mosaic in the field. However, Hoggan (91, 93) in 1931 and 1934 demonstrated that aphid transmission of this mosaic was infrequent when compared with many other aphid-transmitted mosaics. She worked with *Myzus pseudosolani, Myzus persicae, Myzus circumflexus* and *Macrosiphum solanifolii.* According to these experiments the aphids consistently failed to transmit the virus from tobacco to other hosts. However, transmission was obtained with some regularity from tomato to tobacco and other solanaceous hosts. Transmitting efficiency of three species was determined by transferring known numbers of aphids from diseased *Lycopersicon pimpinellifolium* (93) to hybrid plants of *Nicotiana tabacum* x *N. glutinosa* and counting the number of local lesions of tobacco mosaic produced on the hybrid host. This experiment demonstrated that, "with *Myzus pseudosolani,* about 1 aphid in 129 caused infection; with *Macrosiphum solanifolii* about 1 aphid in 140; and with *Myzus persicae* about 1 aphid in 800 or more." Comparative tests in the transmission of a crucifer virus and sugar beet mosaic virus indicated that approximately 1 aphid in 4 or 5 of *Myzus persicae* served as a vector.

Hoggan's experiments were very significant in that they demonstrated the importance of a particular host species being used as the source of the virus in studies of insect transmission.

Parris (177) in 1938 stated that mosaic is the most prevalent tomato virus disease in Hawaii. Of great concern to tomato growers in Hawaii and in other parts of the world is the role of insects in spreading the tobacco mosaic virus from tomato to tomato as tomato mosaic. While Hoggan's experiments demonstrated the ability of *Macrosiphum solanifolii* to transmit mosaic from tomato to tobacco and some other hosts, no experiments have been reported in the literature on the insect transmission of this disease from tomato to tomato. It is conceivable that if aphids can acquire the virus from tomato more readily than from other host plants, they may also be able to infect tomato with greater efficiency than other plants.

Hoggan (93) concluded that it "seems unlikely that any appreciable amount of dissemination of tobacco mosaic may be brought about by aphids, except perhaps from tomato."

In 1937, Chamberlain (45) reported that attempts to transmit mosaic from tobacco, tomato and black nightshade to tobacco by means of *Myzus persicae, M. solani* and *Macrosiphum solanifolii* were unsuccessful in New Zealand. Failure to duplicate Hoggan's results may have been due to differences in technique or possibly even to inherent differences in the transmitting ability of the aphid stocks used.

In 1934 Chamberlain (41) had reported that in New Zealand healthy tomato plants growing next to infected ones did not become infected with mosaic even though the plants had carried many *Macrosiphum solanifolii* and *Myzus solani* early in the season. Absence of rapid spread in a restricted local situation such as reported by Chamberlain does not warrant the conclusion that aphids do not play an important role in the spread of tomato mosaic. The evidence seems convincing that this virus is rarely transmitted by aphids from hosts other than tomato and in all probability spread of the disease by aphids from tomato to tomato is at a relatively low rate when compared with most aphid-transmitted viruses. Nevertheless, even an inefficient vector may be responsible for serious crop losses from virus diseases when the vector occurs in large numbers as does *Macrosiphum solanifolii* in many tomato fields in Hawaii during the cooler months of the year. Such a vector may do its greatest damage by establishing new and scattered foci of infection in a field or region. Even though the number of plants infected by the aphids may be relatively low, the disease may thereby become widely scattered and result in subsequent spread to surrounding plants by more efficient means of transmission such as leaf contact, or contamination of clothing, tools and machinery.

There is a definite need for determining the role of aphids in the spread of tomato mosaic in Hawaii.

Tomato Streak

This virus disease of tomatoes was reported by Parris (177) to be present in Hawaii. He also described the disease symptoms. According to information received from the Vegetable Crops Department of the University of Hawaii Agricultural Experiment Station, this disease, though present, is not common in Hawaii. Smith (215) calls this virus Lycopersicum virus 1 and states that it is similar to, but distinct from, tobacco mosaic virus. Parris (177), on the basis of a report by Jones and Burnett (108), described it as a combination of tomato mosaic virus and potato mottle or X virus in tomato. However, Holmes (96) treats it as a single strain of tobacco mosaic. It has not been shown to be transmissible by insects. However, if ordinary tomato mosaic (tobacco mosaic) is a component part, this portion could be transmitted by the aphid vectors reported for tobacco mosaic.

Tomato Mottle Mosaic

This disease, characterized by faint interveinal mottling and some necrotic spots on the leaves, was reported by Parris (177) to occur in Hawaii but it is not common. It was reported (177) to be caused by the potato mottle virus (X virus) in tomato. No insect vector is known.

Tomato Spot Necrosis

Parris (177) listed this disease as occurring in Hawaii. However, it is not considered common in the Territory. According to Jones and Burnett (108) it is caused by the presence in tomato of rugose mosaic virus of potato. Rugose mosaic is produced by a combination of potato mottle virus (potato latent or X virus) and the veinbanding virus described by Dykstra (55). A similar disease of tomatoes produced by potato mottle virus in combination with a strain of tobacco mosaic virus is described by Holmes (96). Tomato spot necrosis has not been demonstrated to be insect transmissible although one of its components, veinbanding, is readily transmitted by *Myzus persicae* and *Macrosiphum solanifolii* (113).

Tomato Veinbanding Virus

This is the component of rugose mosaic of potato which is transmitted by *Myzus persicae* and *Macrosiphum solanifolii* (113). Parris (177) recorded its occurrence in Hawaii and stated that its detrimental effect on the plant is of minor importance.

Tomato Aucuba Mosaic

Parris (177) reported aucuba (yellow) mosaic of tomato in Hawaii but it is not common. No insect vectors are known.

Spotted Wilt

(Caused by spotted wilt virus, typical strain, *Lethum australiense* var. *typicum* Holmes)

Synonyms: Pineapple yellow spot virus, Ananas virus 1, Tomato virus 1, Lycopersicum virus 3, Kromnek disease virus of tobacco and tomato, "Vira-Cabeça" virus of tobacco and tomato, "Corcova" virus of tobacco and tomato.

Spotted wilt is an important virus disease in many parts of the world. Since its discovery in Australia (31) it has been reported from all of the main regions of the world except the Orient. Norris (157) in 1943 reported the presence in Australia of three strains of spotted wilt virus. These were designated as mild strain, ringspot strain and necrotic strain. The necrotic strain was purified and Norris stated that it appeared to be identical with the virus described from Oregon by Milbrath (146) in 1939, as tomato tip blight. Holmes (96) lists tip blight as a distinct strain of spotted wilt.

Insect transmission. Sakimura (200) in 1940 presented a review of the literature published up to 1939 on spotted wilt with particular reference to the insect vectors. Pittman (187) in 1927 reported transmission of spotted wilt virus in Australia by means of *Thrips tabaci.* Samuel *et al.* (201) and Bald *et al.* (8) in 1930 and 1931 demonstrated that the virus was transmitted in Australia by two species of thrips. One is a species of *Frankliniella,* listed in most

literature as *F. insularis* Franklin. The other is *Thrips tabaci.* The identity of the *Frankliniella* species has been the cause of much confusion. Samuel *et al.* (201) reported that the species used in their transmission test was identified by Dr. G. D. Morison as *Frankliniella insularis.* However, other material from the same source was sent to A. A. Girault in Queensland who described the thrips as a new species, *Parafrankliniella nigripes,* in 1928. Girault's description was ignored and, when the fact became apparent that the use of the name *insularis* was based upon a misidentification, Andrewartha (5) in 1937 described the species as new under the name *F. lycopersici.* This latter name was used by Sakimura (200), Leach (122) and others. However, since the species was validly described as *nigripes* by Girault in 1928, the name *lycopersici* And., applied in 1937, must be considered a synonym. The common spotted wilt vector of Australia is therefore *F. nigripes* (Girault) as listed by Essig (61) in 1942.

Samuel *et al.* (201) and Bald *et al.* (8), in addition to determining two vectors of spotted wilt virus, also reported on the unusual specific relationships which exist between the virus and the vectors. The virus must be acquired by the thrips while the latter are still in the larval stage. The virus is retained through pupation, and the emerging adults may serve as vectors. If the adult stage has been attained before the thrips are permitted to feed on virus inoculum, they are incapable of serving as vectors.

Linford (125) in 1932 reported transmission of yellow spot of pineapple in Hawaii by means of *T. tabaci.* At the time this work was done, the coidentity of yellow spot and spotted wilt was not known although Linford noted that the peculiar relationship of the virus to the vector (which he discovered independently) was the same as that existing between spotted wilt and its vectors. The virus has a relatively long incubation period in the vectors, varying from 5 to 10 days. In Hawaii this was found by Linford to be approximately 10 days in *T. tabaci.*

Bailey (6) in 1935 reported that *T. tabaci* in Hawaii was found by Dr. Carter to retain the virus for about 30 days during adult life.

Smith (211, 213) in 1931 and 1932 demonstrated that *T. tabaci* is the vector of spotted wilt in England. Gardner *et al.* (76, 77, 78) in 1934, 1935 and 1937 reported transmission of spotted wilt to a large number of plant species in California by means of *T. tabaci* and *Frankliniella moultoni* Hood. In 1936 Essig and Michelbacher (62) and in 1942 Essig (61) cited *F. occidentalis* Perg. as a vector in California. Sakimura (200) reported that he had been informed in private correspondence from Dr. S. F. Bailey, of the University of California, that the inclusion by Essig and Michelbacher of *F. occidentalis* Perg. in the place of *F. moultoni* Hood "is a matter of different view on the classification of the species."

Moore and Anderson (148, 149) in 1933 and 1939 discussed Kromnek disease of tobacco and tomato in South Africa where transmission was obtained by means of *Frankliniella schultzei* and *T. tabaci.* These authors stated (149) that the virus is probably identical with spotted wilt of Australia, Europe and America. Carter (37) in 1939 reported his observations on the Kromnek disease of South Africa. Here he found, in fields surrounded by Kromnek-diseased weeds, symptoms on pineapple, indistinguishable from yellow spot of pineapple in Hawaii. *F. schultzei,* vector of Kromnek disease, was also found in the flowers of pineapple.

Silberschmidt (207) in 1937 announced that Dr. Santos Costa had obtained transmission of "Vira-Cabeça" disease of tobacco in Brazil by means of *tabaci* and *Frankliniella* sp. Sakimura (200) quoted personal correspondence regarding the identity of the Brazil disease and its vector as follows: "Dr. A. S. Costa . . . recently informed the writer that the virus of 'Vira-Cabeça' of tobacco and tomato, widely distributed in Brazil, has been proven to be identical with S. W. (spotted wilt) virus; that the vector is a species of *Frankliniella,* possibly *F. paucispinosa* Moulton, which is also known to be the vector of 'Corcova' of tobacco and tomato in Argentina."

In 1938 and 1940 Fawcett (64, 65) reported transmission of "black pest" of tomatoes ("Corcova" disease of tobacco) in Argentina by means of *F. paucispinosa,* and stated the disease appeared to be identical with spotted wilt. The same author announced (65) that "Vira-Cabeça" of Brazil appeared to be identical with "Corcova" of Argentina.

Host range of spotted wilt virus. The host range of this virus is very large. Smith (215) listed over 100 susceptible species which occur in 23 different families. Most of them occur among the dicotyledons but some are monocotyledonous species. More host plants of the virus have been found in the Solanaceae than in any other family. In tomato, the disease is very destructive in several parts of the world.

In Hawaii, Kikuta *et al.* (112) in 1945 announced the development of a tomato variety, Pearl Harbor, which is resistant to Hawaiian spotted wilt. However, they suggest that preliminary tests with the Pearl Harbor variety in other parts of the world indicate it may not possess resistance to some strains of the spotted wilt virus.

In peas, spotted wilt virus causes streaking and necrosis of pods, stems and leaves. In 1931 Linford (124) transmitted pineapple yellow spot virus from *Emilia sonchifolia* to peas by means of *T. tabaci* and noted that the symptoms appeared to be identical with the pea streak he had observed at various localities across the United States. He suggested that the pea streak on the mainland was caused by the same or a related virus. At the time of this

report the coidentity of yellow spot and spotted wilt had not been established. In 1936 Whipple (239) transmitted spotted wilt to peas by *T. tabaci* and obtained typical pea streak symptoms.

Yellow Spot of Pineapple

(Caused by spotted wilt virus in pineapple)

This disease, first reported publicly by Illingworth (97) in 1931, was discovered in Hawaii in 1926. Its sudden appearance, rapid spread in some areas during the first few years, and the destructive effect produced on the pineapple plant appeared to mark this disease as a major pineapple problem. The steps leading to the determination of the insect vector of the disease in Hawaii constitute an interesting record since it provides an instance in which the probable identity of the vector group was established before the vector had actually been observed as a pineapple inhabiting insect. The procedure has been discussed by Illingworth (97) and Linford (125). Illingworth carried out preliminary exploratory experiments in search of the vector. The investigations which established the identity of the vector were conducted by Linford. Undetermined *Nysius,* aphids, leafhoppers, thrips, chironomid maggots, nitidulid beetles, spring tails, red spiders, tarsonemid mites as well as the pineapple mealy bug, bud-moth caterpillars and *Scolia manilae* Ashm. wasps were tested without success by Illingworth (97). Dr. R. N. Chapman (97, 125) then discovered microscopic insect punctures associated with the yellow spot symptom on the pineapple leaves. These were first assumed to be the feeding punctures of a relatively large sucking insect. However, Linford (125) discovered that the punctures represented the oviposition site of a small insect as evidenced by the finding of egg membranes in the reniform cavities beneath the surface openings. The only insects which seemed to fit the specifications for such a puncture were thrips. This was demonstrated subsequently by the findings of unhatched eggs, from which thrips emerged, in pineapple leaves taken from a disease-infected area. As these investigations were proceeding, another host plant of the virus was also being sought. The presence of a similar disease on *Emilia sonchifolia* in infected areas resulted in the investigation of this plant, its virus disease and the thrips, *Thrips tabaci,* which infested it. Proof of the vector's identity was established soon thereafter. When thrips were transferred from diseased *Emilia* to healthy pineapple, typical yellow spot disease was transmitted.

In addition to *sonchifolia, Emilia* species designated as *Emilia* No. 3 and *Emilia* No. 4 have been reported by Sakimura (198) to serve as host plants for the virus and for the thrips vector in Hawaii. Other host plants of *Thrips tabaci* in Hawaii and its life history on *Emilia sonchifolia* have been reported by Sakimura (197).

Sakimura (199) in 1940 demonstrated in a long series of tests that *Thrips nigropilosus* Uzel is not able to transmit spotted wilt virus (yellow spot virus) in Hawaii. Preliminary transmission tests with *Hercinothrips femoralis* (Reuter) were also negative (199).

The fact that the yellow spot disease did not become a limiting factor to pineapple production as had been feared, but has remained a minor problem except for occasional outbreaks, is due to the host plant preference shown by the vector as discussed by Carter (35, 38). Pineapple normally is not a host of *Thrips tabaci* which, in Hawaii, breeds preferentially on *Emilia.* Furthermore, Carter (38) reported that "Incidence of yellow spot disease in pineapple is not correlated with *Thrips tabaci* populations on *Emilia* in nearby areas. This is accounted for on the grounds that *Emilia* is a favored host from which dispersal does not normally occur." Factors which may be responsible for the occasional sporadic movement of thrips from infective *Emilia* to pineapple plants are destruction of the *Emilia* by means of cultivation or drought (38).

Although the possibility was considered for some time that tomato spotted wilt virus and pineapple yellow spot virus were the same, their coidentity was not well established until 1940 when Sakimura (200) and Parris (181) published results of their investigations in Hawaii. By means of *Thrips tabaci,* Sakimura transmitted yellow spot virus to and from spinach, broad bean, celery, potato, eggplant, bell pepper, tomato, tobacco, *Nicotiana glutinosa* L., *Datura stramonium* L., petunia, chicory, endive and lettuce, all of which are known to be susceptible to the spotted wilt virus. The symptoms produced were identical with those recorded for spotted wilt. Plants known to be immune from spotted wilt virus were also found to be immune to yellow spot virus.

Parris (181) transmitted the tomato spotted wilt virus mechanically from tomato to tomato and potato and from *Emilia sonchifolia* to *Emilia* and tomato. On the basis of host range and symptoms, he concluded that tomato spotted wilt and pineapple yellow spot were probably caused by the same virus.

The joint study of this problem by Sakimura and Parris was undertaken after an outbreak of a tomato disease, which appeared to be identical with spotted wilt (176), occurred in Hawaii in 1937. This situation provided the opportunity and need for determining the relationship of pineapple yellow spot and tomato spotted wilt.

Tomato Tip Blight

Synonyms: Tomato spotted wilt virus, tip blight strain (*Lethum australiense* var. *lethale* Holmes).

Martin (138) in 1943 reported the presence in Hawaii of a tomato disease which he considered distinct from spotted wilt and believed would prove to be tomato tip blight as reported by Milbrath (146) in 1939 in Oregon. The properties of the virus were

studied by Milbrath (146), who considered the disease to be new. Transmission was obtained by means of *Thrips tabaci* (146).

The writer is following Holmes (96) and Norris (157) in classifying this as a strain of spotted wilt virus. Norris studied three strains of tomato spotted wilt virus in Australia, one of these, designated as necrotic strain, was discussed by Norris in part as follows: "This strain appears to be identical with the virus described by Milbrath in Oregon as tomato tip blight. The symptoms produced on tomato, tobacco, and other hosts correspond closely. In Oregon this necrotic strain was apparently separated out by chance from the others, but its occurrence in close association with ordinary spotted wilt was observed. Milbrath also noted that occasionally both viruses occurred together in tomato. In such cases it would appear that other strains were present but that the necrotic strain occurred in very high concentration."

The fact that tomato tip blight is transmitted by *Thrips tabaci,* suggests strongly that the affinity of this virus and that of spotted wilt is exceedingly close, because this thrips species has not yet been incriminated as a vector of any virus proven to be outside of the spotted wilt complex.

Potato Virus Diseases in Hawaii

Virus diseases, if prevalent in potato plantings, seriously reduce the crop yields in quantity. Parris (176, 177) reported figures on yield reduction in Hawaii due to the use of virus diseased seed stock. Dr. W. A. Frazier, of the University of Hawaii has informed the writer[11] that in Hawaii potato losses due to virus diseases are not ordinarily very serious in areas where mainland-grown certified seed is used. However, the practice of some farmers of using seed grown locally often results in very serious losses due to potato viruses. This is due to the fact that potato viruses are transmitted through the tubers. If disease-free seed is used in Hawaii, most of the plants escape virus infection until late in the season when it is too late for the virus to injure the crop materially. However, before they are harvested, many normal-appearing tubers may contract virus infection. When such tubers are used as seed for subsequent crops a number of diseased plants are produced early in the season. These provide sources of infection from which insect vectors may transmit the viruses to surrounding healthy plants. Seed stock should therefore be obtained from areas which are relatively free from potato virus diseases.

Although no transmission experiments involving potato viruses have been reported in Hawaii, Parris (177) listed the following diseases as occurring in the Territory: mottle, mild mosaic, crinkle mosaic, leaf rolling mosaic, veinbanding, rugose mosaic, and leaf roll.

[11] Personal communication.

Potato Mottle Virus

Synonyms: Latent virus; "healthy potato virus"; Virus X.

Dykstra (55) in 1939 reviewed the literature on this—the most ubiquitous of all potato viruses known. It has been shown to be present in masked form in all the tubers of most, and perhaps all, of the commonly used varieties of "apparently healthy" potatoes. In Europe this virus has been found to produce a mild form of mottling in some varieties.

Dykstra (55) transmitted this virus to *Amaranthus retroflexus* and stated, "This is believed to be the only potato virus that has been successfully transmitted to plants outside the Solanaceae." Alone or in combination with other viruses it causes diseases of potato, tomato, pepper and other plants.

Holmes (96) classifies this virus into three strains, (a) potato ringspot strain, which is masked in potato but produces systemic necrosis and ring shaped lesions on tobacco and pepper; (b) yellow-mottle strain, which imparts a yellow cast to potato foliage and in tomato causes yellow-mottling mosaic and occasional necrosis of the young leaves (see tomato mottle mosaic); (c) masked-mottle strain, which produces symptomless systemic infection in potato, tobacco and *Datura stramonium,* but which produces systemic necroses in pepper.

No insect vector has been found capable of transmitting any of the strains of potato mottle virus.

Potato Mild Mosaic

According to Dykstra (55) the mild mosaic virus, in the absence of the mottle or X virus, causes only a very faint mottling in the potato foliage. However, in combination with virus X (which alone shows no symptoms in many potato varieties) mild mosaic produces a pronounced mottling and crinkling of the foliage. According to Parris (177) plants produced from tubers infected with mild mosaic may die prematurely.

Holmes (96) listed *Aphis rhamni* B. de Fonsc. (= *A. abbreviata* Patch) and *Myzus persicae* as the vectors of potato mild mosaic. However, Dykstra and Whitaker (57) in 1938 reported experiments which demonstrated that this virus can be transmitted by *Myzus persicae, M. circumflexus, M. solani,* and *Macrosiphum solanifolii.*

Reports from the Vegetable Crops Department of the University of Hawaii Agricultural Experiment Station indicate that this virus is common in some potato plantings in Hawaii.

Potato Crinkle Mosaic

This disease is similar to mild mosaic but differs in that the leaflets are more ruffled and the mottle areas are larger. Like mild mosaic and rugose mosaic it is a composite disease composed of

the mottle or X virus and at least one other component. When X virus is removed, the remaining component causes only a faint mottling. When X virus is added, the mottling becomes pronounced and the leaves become crinkled (55).

Dykstra and Whitaker (57) reported transmission of crinkle mosaic free from X virus by means of the same aphids which transmitted mild mosaic, i.e. *Myzus persicae, Myzus solani, M. circumflexus* and *Macrosiphum solanifolii.* As in the case of mild mosaic and rugose mosaic, the aphids transmitted not the disease as named, but only one component since X virus, the other necessary component, is not transmitted by insects. The percentage of positive transmission varied from 0 to 100 per cent in different experiments.

Potato Leaf Rolling Mosaic

This disease discussed by Smith (215) under the name Solanum virus 11, is characterized by a diffused mottling and an upward rolling of the leaves which are flaccid and resemble the rolling of leaves on plants affected with *Rhizoctonia* or blackleg (55, 215). It differs from leaf roll in that the plants are not dwarfed and the leaves are not tough. Schultz and Folsom (203), who originally described the disease, reported transmission by means of unspecified aphids. No report of insect transmission has been published by other workers.

Potato Veinbanding Virus

Holmes (96) classifies this virus as cucumber mosaic virus, veinbanding strain.

Smith (215) lists veinbanding virus, potato virus Y and stipple streak virus as synonyms of Solanum virus 2. Dykstra (55), however, treats veinbanding, Y and stipple streak viruses as related but distinct strains.

Veinbanding is transmitted by *Myzus persicae, M. solani, M. circumflexus,* and *Macrosiphum solanifolii* (57, 113). This virus is characterized by crinkling and downward curling of the leaves. The veins of older leaves become necrotic. Diseased plants are stunted and usually die prematurely (55). This virus in combination with the potato mottle or X virus produces rugose mosaic of potato (55).

Potato Rugose Mosaic

This disease is the result of the presence in potato of mottle or X virus and the veinbanding virus. Since the X virus component of rugose mosaic is not transmitted by insects, the records of insect transmission of rugose mosaic actually refer to transmission of the veinbanding component (see "Veinbanding Virus"). However, the almost universal occurrence of mottle or X virus in potato means that whenever veinbanding virus is transmitted to a potato plant, the resultant disease will probably be rugose mosaic.

According to Smith (215), the characteristics of this disease are: mottling of the upper leaves and dark, necrotic veins on the lower leaves, and crinkled and rugose foliage. Severely affected plants are stunted and the tubers are small. The mottle symptoms may be masked by high temperatures, but the rugosity and stunting persist.

This disease is sometimes serious in Hawaii.

Potato Leaf Roll

Potato leaf roll is considered by Smith (215) to be the principal cause of potato degeneration in several parts of Europe and the British Isles. It is also common in some parts of North America. In Hawaii, leaf roll has not been a serious problem.

The symptoms of leaf roll make it relatively easy to identify. The leaves of diseased plants are rolled, thick, rigid and leathery. This is reportedly due to an abnormally high starch content which, in turn, results from prevention of normal food translocation in the plant because of phloem necrosis in the stems. Affected plants are dwarfed and tubers are reduced in size and number. This virus has not been found to be transmissible by mechanical means.

Insect transmission. Many workers have reported insect transmission of leaf roll and the accounts are sometimes conflicting. Vectors have been reported as follows: *Aphis rumicis* L. and *Myzus persicae* by Ortwijn Botjes (165) in 1920; *Calcoris bipunctatus,* a plant bug, and *Typhlocyba ulmi* (Linn.), a leafhopper, by Murphy (151) in 1923; *Macrosiphum solanifolii* and *Myzus pseudosolani* by Murphy and McKay (152) in 1929; *Myzus persicae, M. pseudosolani, Aphis rhamni, Eupteryx auratus, Lygus* sp., *Psylliodis affinis,* and the larvae of *Tipula paludosa* by Elze (59) in 1927, the last species being listed as an underground vector; *Macrosiphum solanifolii, Myzus persicae* and *Empoasca fabae* by Cleveland (47) in 1931 with the latter two species listed as the main vectors in Indiana. Smith (210, 212, 215) in 1929, 1931 and 1934 failed to transmit the virus by means of *Calcoris bipunctatus, Lygus pabulinus, Eupteryx auratus, Chlorita viridula, Psylliodis affinis,* and *Macrosiphum gei,* (= *solanifolii*) but obtained transmission with *Myzus persicae, M. pseudosolani* and *M. circumflexus.*

This disease is of singular interest because of the unusual relationship known to exist between the virus and *Myzus persicae,* one of its vectors (215). After this aphid has fed on leaf roll inoculum, an incubation period of from 48 to 54 hours is required before *Myzus persicae* can infect a healthy plant. Furthermore, this species retains the virus in an infective condition for long periods of time while feeding on plants which are immune to the virus. A similar relationship has been reported by Osborn (168, 170) to exist between pea enation mosaic virus and its two vectors, *Macrosiphum*

pisi and *M. solanifolii,* and by Ocfemia and Buhay (160) for *Pentalonia nigronervosa* in transmitting abacá bunchy top virus in the Philippines. Except for a few cases such as these, other aphid-transmitted viruses are reported to require no incubation period in the aphid vectors. Also, such viruses are not retained by their vectors for more than a few hours at most after the last feeding on virus inoculum.

Dykstra and Whitaker (57) reported that transmission experiments using *Myzus persicae, M. circumflexus, M. pseudosolani* and *Macrosiphum solanifolii* resulted in a high percentage of potato leaf roll virus transmission by means of the *Myzus* species. *Macrosiphum solanifolii* proved to be a relatively inefficient vector in most experiments, although occasionally a high percentage of transmission was accomplished with this species. A possible explanation was suggested for the difference in transmitting ability demonstrated by the aphids. This explanation was based on the tissues in which the aphids habitually fed. The three species of *Myzus* were found to feed in the phloem in practically all cases. They exhibited approximately equally high efficiency as vectors of the disease. *Macrosiphum solanifolii* fed in the vascular tissues in only 46 per cent of the cases observed. This suggests that phloem feeding may be necessary for the aphids to acquire and/or transmit the virus.

This explanation is a logical one in view of what is now known regarding the relation of viruses to plant tissues. Bennett (12) has contributed greatly to our understanding of this problem. He has demonstrated that leaf-curl of raspberry and curly top of sugar beet are caused by viruses apparently closely limited to the phloem. He also pointed out that several other diseases, including potato leaf roll, which exhibit symptoms resulting from phloem disturbances, are not transmissible mechanically and ordinarily show the type of vector-virus relationship described for *M. persicae* and leaf roll virus.

The reported ability of many of the species listed as vectors of leaf roll needs further confirmation. In view of the incubation period of the virus and its long retention in *Myzus persicae,* and the fact that the disease is not mechanically transmissible, it seems somewhat doubtful that this virus could be transmitted by so many unrelated insect species, some of which would have to transmit the virus in a purely mechanical fashion.

Bamboo Mosaic

Kunkel (114) in 1921 reported that what appeared to be mosaic had been observed several times on *Bambusa vulgaris* Wendl. in Hawaii. No further reference has been made in the literature to the disease.

Canna Mosaic

Kunkel (114) in 1921 stated that mosaic had frequently been observed on *Canna indica* L. in Hawaii. No investigations of the virus or its manner of spread have been conducted.

Dianella odorata Bl. Mosaic

Kunkel (114) in 1921 reported that in Hawaii "A disease closely resembling the yellow stripe disease of sugar cané occurs on *Dianella odorata* Bl." This plant belongs in the lily family.

Hippeastrum Mosaic

In 1922 and 1924 (116, 117) Kunkel reported the common occurrence in Hawaii of a mosaic disease of *Hippeastrum equestre* Herb. This plant (commonly known as Barbados lily) is grown as an ornamental in Hawaii, but has also escaped from cultivation and grows wild in some of the valleys above Honolulu. Kunkel's investigation of the virus was primarily concerned with the study of amoeboid bodies in the cells of virus-diseased plants, but he was also interested in the manner in which the disease was disseminated in nature. Potted plants kept in insect-proof cages remained healthy, while most of those kept outside, a short distance from diseased plants, became infected with mosaic. These results suggested insect transmission. Kunkel made several attempts to transmit the virus by means of the corn aphid, *Aphis maidis*, but all tests were negative.

Smith (215) reported *Hippeastrum* sp. as a host of tomato spotted wilt virus in England, but the symptoms produced by this virus are distinct from those described by Kunkel for Hippeastrum mosaic in Hawaii.

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