STUDIES ON THE SEED TRANSMISSION OF PLANT VIRUS DISEASES

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Summary

Investigations were made to explain the rarity of seed transmission of plant virus diseases. Five mechanically transmitted viruses were used, none of which induced enough sterility in their hosts to account for their lack of seed transmission.

All of the viruses studied infected the seeds of their hosts, but only the seed-transmitted bean mosaic virus was found to infect the embryos.

No evidence has been found for the presence of virus inactivators in seeds or developing embryos.

Evidence is presented that the rarity of seed transmission is due to the inability of most viruses to infect mega- or microspore mother cells of infected plants together with the inability of viruses to infect the developing embryo because of the lack of plasmodesmatal connection with the endosperm.

I. Introduction

Of several hundred plant virus diseases that have now been described, only 45 are reported to be seed transmitted. These are tabulated in Appendix 1, which shows that: (1) the transmission of plant viruses through seeds is not, as has often been claimed, more common in the Leguminosae than in some other families; (2) in only four plants does the percentage transmission exceed 50; (3) the property of transmissibility is not a property of any virus, nor of any host, but is clearly an interaction of the two, virus and host. Four hypotheses have been put forward to explain the rarity of seed transmission:

Hypothesis 1.—Allard (1915) suggested that virus infection "may disturb the normal relations of stamens and pistils to such an extent as to cause sterility". In 1952 Caldwell demonstrated that in the aspermy disease of tomato "the presence of the virus in the microspore mother cell results in a complete interference with the normal stages of meiosis . . .". This hypothesis adequately explains the failure of tomato aspermy virus to pass through the seed of tomato, but cannot be extended to explain why seed transmission of other viruses is so rare when infected plants produce large quantities of seed. This hypothesis also fails to explain how embryos develop to maturity in a virus-infected endosperm without themselves becoming infected.

Hypothesis 2.—Bennett (1936) suggested that the lack of vascular connection between the embryo and its parent plant prevents the seed transmission of those virus diseases which are largely limited to the vascular tissues. This theory adequately explains the absence of seed transmission of all virus diseases of this type, but again is not capable of extension to other virus diseases.

Hypothesis 3.—Duggar (1930) suggested that the seed transmission of those highly infectious viruses, which are almost ubiquitous in their distribution within a plant, might be prevented by the inactivating action of some "specific protein or

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other specific material" in the seeds. This hypothesis was elaborated by Kausche (1940), but neither of these workers distinguished between virus inactivators and virus inhibitors in seeds. Until their "inactivators" are proved not to be inhibitors their conclusions are unjustified.

Hypothesis 4.—Bennett (1936) also suggested that the lack of plasmodesmatal connection between the embryo and the parent plant prevents the seed transmission of even the most infectious virus diseases. This suggestion rests on two assumptions, and neither can be tested experimentally: (1) that the micro- and megaspore mother cells either escape infection, or are unable to support virus multiplication; (2) that the only path of intracellular virus movement is via the plasmodesmata.

The investigations described here were made to determine (i) whether virus-induced sterility could be the reasons for the rarity of seed transmission of some of the more infectious plant viruses; (ii) what tissues of the seed are infected by these viruses; (iii) whether seed-transmitted viruses infect the embryos of their hosts before or after fertilization.

II. Experimental and Results

(*a*) *The Effect of Virus Infection on Pollen and Seed Production*

An essential requirement for investigations associated with the seed transmission of plant virus diseases is a knowledge of whether viruses are able to infect microspores or macrospores, and whether this infection interferes with the normal behaviour of these cells.

Caldwell (1952) showed that with tomato aspermy disease seed transmission is made impossible by virus-induced abortion. He suggested that this may be the usual consequence of infection of microspores or macrospores by a virus, and that when virus infection does not induce abortion, seed transmission will occur. There is evidence which seems to support Caldwell's suggestion. Tobacco ringspot virus induces no sterility in soybean in which it is seed transmitted (Desjardins, Latterell, and Mitchell 1954), but does induce sterility in both petunia and tobacco where it is not seed transmitted (Henderson 1931; Valleau 1941). Similarly, a non-seed-transmitted mosaic disease of *Dolichos biflorus* was found by Uppal (1931) to reduce fertility greatly, and a graft-transmissible disease of tobacco tested by Kostoff (1933) induced complete sterility of infected plants. However, these results do not establish whether the reduced fertility of these infected plants results from the stunting and impairment of vigor induced by the virus, or from a specific effect of the virus on microspores or macrospores.

The results of Blakerslee's (1921) investigations with the Quercina disease of *Datura stramonium* seem to be at variance with Caldwell's suggestion, for Blakerslee found that, although virus infection induced a high degree of sterility, 100 per cent. of the pollen produced on infected plants was infected. In this case, at least, virus infection does not invariably lead to the abortion of pollen mother cells. Unfortunately there is no evidence to indicate whether the sterility induced by this virus results from an interference with the meiotic divisions or from a physiological disturbance of floral development.

If Caldwell's suggestion is assumed true, the rarity of reports of virus-induced sterility is surprising, particularly with highly infective viruses such as tomato spotted wilt, cucumber mosaic, or tobacco mosaic virus. Nixon (1956) estimated that the number of particles of tobacco mosaic virus per cell of tobacco leaf tissue is of the order of 6×10^7. If their concentration in floral tissues is anything approaching this, there should be a significant reduction in fertility, unless some mechanism prevents the infection of the gametophytic tissues, or the survival of viruses in them. Allard (1915) reported a reduction in the seeds produced by mosaic-infected tobacco plants, but there are no reports of any effect of this virus on the seed production of any of its other hosts.

Few critical investigations on this aspect of the effect of plant viruses on their hosts have been done; the effects, therefore, of four viruses on the fertility of their hosts were studied in the present work. The viruses used were bean yellow mosaic, bean mosaic, tobacco mosaic, and cucumber mosaic.

(i) *Effect of Bean Mosaic and Bean Yellow Mosaic Viruses on the Fertility of Bean.*—The two viruses of bean were used because the yellow mosaic virus is not seed transmitted and the common mosaic virus is transmitted through both seed and pollen. An obvious conclusion is that if the primary cause of the lack of pollen infection by bean yellow mosaic virus is the abortion of the microspores then the virus should have a marked effect on fertility. The bean mosaic virus used was provided by Mr. J. Johnson, Queensland Department of Agriculture and Stock, Brisbane. It did not infect peas or clovers, and in beans it induced symptoms closely corresponding to those described by Reddick and Stewart (1919). The bean yellow mosaic virus used was isolated from some naturally infected bean plants at Mt. Gambier, S.A., and infected bean, pea, *Trifolium incarnatum* L. and *T. hybridum* L.: in all of these hosts the symptoms corresponded closely to those described by Pierce (1934).

Two experiments were carried out with these viruses. The first was begun early in the summer of 1955. It was repeated early in 1956, but with all plants raised in a cooled glass-house so that the symptoms of the diseases would not be masked by high temperatures. Thirty-three bean seedlings (var. Canadian Wonder) were used in the experiments, and were allocated at random to each treatment. Eleven plants were inoculated with bean mosaic virus, eleven with bean yellow mosaic virus, and eleven were left uninoculated. Throughout the growing period, records were kept of the number of flowers appearing daily on each plant, of the number of pods produced, and, after harvest, of the number of seeds per pod in each of the three treatments. The results of these experiments are given in Table 1.

The best estimate of plant fertility is the number of seeds produced per flower. On this basis a slight reduction in fertility was produced by both viruses, but it was not sufficient to explain the lack of seed transmission of bean yellow mosaic virus.

(ii) *Effect of Tobacco Mosaic and Cucumber Mosaic Viruses on the Fertility of Pungent Pepper.*—The hypothesis of virus-induced sterility could be tested more satisfactorily with viruses that reach high concentrations in their hosts. If sterility does result from the infection of the megaspores and pollen grains by viruses, then those viruses which are in highest concentration should induce most sterility. Tobacco

mosaic and cucumber mosaic viruses were chosen as suitable for this investigation. The tobacco mosaic virus was isolated from pungent pepper seed (*Capsicum frutescens* L.) obtained from Dr. N. H. McKinney, who reported 22 per cent. seed transmission to occur in this host (McKinney 1952). The cucumber mosaic virus was provided by Mr. L. L. Stubbs, Dept. of Agriculture, Burnley, Vic., and originated from naturally infected cucumbers. It infected *Cucumis sativa* L., *C. melo* L., *Cucurbita pepo* L., *Citrullis vulgaris* Shrad., *Nicotiana tabacum* L., *Lycopersicon esculentum* Mill., *Datura stramonium* L., and *Vigna sinensis* (L.) Endl. ex Hassk. and produced symptoms consistent with those described by Doolittle (1920).

TABLE 1

EFFECT OF VIRUS INFECTION ON THE FERTILITY OF BEAN PLANTS

Virus	Flowers per Plant	Pods per Plant	Seeds per Pod	Experiment 1 Seeds per Flower	Experiment 2 Seeds per Flower
None	28	10	3·8	1·4	1·1
Bean mosaic	33	10	4·1	1·3	0·4
Bean yellow mosaic	25	8	3·2	1·0	0·7

It was hoped to determine the effect of two strains of tobacco mosaic virus on the fertility of pepper, in which only one strain was seed transmitted. However, pungent peppers inoculated with the ordinary strain of tobacco mosaic virus produced only local lesions on the inoculated leaves, and systemic symptoms did not develop. It was also found that although the pepper strain of this virus is seed transmitted in pungent pepper, the embryos are not infected, but become infected by contamination from the infected testa during germination. A similar occurrence with the "tomato streak" strain of tobacco mosaic virus was described by Chamberlain (1950) in tomato.

These results thus prevented the investigation of this aspect of the problem using these two strains of a single virus on *C. frutescens*. Instead, the effect of the two viruses, tobacco mosaic and cucumber mosaic, on the fertility of pungent peppers was investigated. Experiments were carried out as described above with bean, using eight plants in each treatment.

These results (Table 2) show that tobacco mosaic virus did not affect the fertility of pungent pepper plants, whereas cucumber mosaic virus reduced their fertility by between 50 and 80 per cent. Comparisons of the pollen grains from both healthy and cucumber mosaic-infected plants did not reveal the presence of any abnormal pollen. Examinations of anthesis at an earlier stage gave no indication that either virus disturbed meiosis, and tetrads of microspores were regularly found in young anthers of infected plants. It is unlikely that a 50 per cent. reduction in the fertility of plants could be produced by interference with the normal process of meiosis without some microscopically visible effect being produced on both anthesis and pollen production. On the other hand, cucumber mosaic virus greatly upsets the normal growth of pepper plants. Infected plants are stunted, flower production is

disturbed, and they produce fewer flowers over a much longer period, and fruits are greatly distorted. It is therefore concluded that cucumber mosaic virus reduces the seed production of pungent pepper plants primarily by upsetting the normal hormonal control of plant growth with the result that fruit growth is abnormal and stunted and seed production is much reduced.

It is concluded that although virus-induced sterility may be an adequate explanation for the lack of seed transmission of tomato aspermy disease, it does not appear to be a contributing factor to the lack of seed transmission of the viruses investigated. There is no evidence that these viruses interfere with the meiotic divisions of their hosts, and, as neither is pollen transmitted, it would seem that some other mechanism must exist to prevent the infection of micro- and macrospores.

TABLE 2

EFFECT OF TOBACCO MOSAIC AND CUCUMBER MOSAIC VIRUSES ON THE FERTILITY OF PUNGENT PEPPER PLANTS

Experiment No.	Virus	Flowers per Plant	Fruits per Plant	Seeds per Fruit	Seeds per Flower
1	None	82	18·6	134	30·4
	Cucumber mosaic	82	5·1	91	5·7
	Tobacco mosaic	89	16·9	135	25·7
2	None	70	7·0	112	11·1
	Cucumber mosaic	44	2·2	43	2·2
	Tobacco mosaic	43	7·2	88	15·0

(*b*) *The Location of Viruses in the Seeds of their Hosts*

As the viruses studied did not induce abortion of the micro- or macrospores it was concluded that either (1) infection of these cells does not kill them; or (2) viruses are excluded from gametophytic tissues. Assuming the first to be true, embryos must be infected early in their development and seed transmission then prevented by the inactivation of virus in the embryo during seed maturation. If the second alternative is true then embryos are not infected initially, and seed transmission can still occur unless infection of the developing embryo is prevented.

Facts about the distribution of viruses in seeds are remarkably few. Different workers have reached conflicting conclusions; they have stated that in tomato seeds tobacco mosaic virus is (1) mainly a superficial contaminant (Chamberlain 1950); (2) present in the testa only (Ainsworth 1935); (3) capable of infecting testas and two-thirds of the embryos (Berkeley and Madden 1932). (The seeds used by Berkeley and Madden had already begun to germinate and may have been contaminated from the infected testa.) The distribution of only three other non-seed-transmitted viruses has been investigated. Bennett (1936) showed the curly top virus of sugar-beet to be present in all the tissues of the seed except the embryo. Sheffield (1941) obtained evidence from a study of virus inclusion bodies that "severe etch" virus infects the testa but not the endosperm or embryo of the seeds of *Hyoscyamus niger* L. Cheo

(1955) reported the infection of 100 per cent. of both embryos and testas of bean by "southern mosaic" virus. He suggested that seed transmission of this virus was prevented by the inactivation of the virus during maturation and storage of the seed. A similar suggestion was made by Gold *et al.* (1954) to explain the fact that although barley mosaic virus infected 100 per cent. of the developing embryos, only 50–90 per cent. seed transmission occurred.

The results of Ainsworth and Sheffield thus seem to support the hypothesis that viruses are unable to infect embryos and the results of Cheo and Gold *et al.* support the hypothesis that viruses do infect embryos but are inactivated before the seed germinates.

Investigations were made to determine whether embryos are infected, and whether there is any virus inactivation associated with the maturation of the seed. Five viruses were used: two viruses of bean, of which only one was seed transmitted, and three other viruses were chosen such that with each one host could be used in which seed transmission did occur, and a second host in which seed transmission had not been reported. The virus–host combinations were:

(1) Bean mosaic virus (43 per cent. transmission (Archibald 1921)) and bean yellow mosaic virus and bean.

(2) Tomato spotted wilt virus in cineraria (96 per cent. transmission (Jones 1944)) and tomato.

(3) Cucumber mosaic virus in wild cucumber, *Echinocytus lobata* Mich. T. and G. (22 per cent. transmission (Doolittle and Gilbert 1919)) and cucumber.

(4) Tobacco mosaic virus in pungent pepper (22 per cent. transmission (McKinney 1952)) and tomato.

(i) *Seed Dissection and Inoculation Techniques.*—No special techniques were necessary for the dissection of bean, cucumber, or wild cucumber seeds. The tissues were washed in water, ground in neutral composite buffer, and inoculated to test hosts. The presence of bean viruses was tested for by inoculation to five 10–14-day-old bean seedlings (var. Canadian Wonder) and for cucumber mosaic virus by inoculation to the cotyledons of five young cucumber seedlings (var. Long Green). The dissection of seeds of cineraria, tomato, and pepper was simply accomplished when the seeds were first soaked for 1 or 2 hr. In dissecting cineraria seeds, a small cut was made at the bottom of the fruit, through which the embryo was ejected by gently pressing with a flattened needle. Similarly, young seeds of tomato and pungent pepper can be separated into testa, endosperm, and embryo. *Nicotiana tabacum* was used in all inoculations to detect tomato spotted wilt virus because it was found to be more susceptible than *N. glutinosa.*

Tomato seeds from plants infected with tomato spotted wilt virus were obtained from naturally infected field crops. The cineraria seeds were obtained from both naturally infected field-grown plants and from glass-house plants infected with either a field strain or one of four pure strains of tomato spotted wilt virus, which were provided by Dr. R. J. Best, Waite Institute. All other viruses used have been described above. The seeds from tobacco mosaic-infected fruits were dissected in the same manner as the cineraria seeds, and the tissues soaked in 10 per cent. "Teepol" for

several hours. This inactivated any virus particles that were superficially contaminating the tissues, particularly those of young seeds, in which the endosperm is gelatinous and invariably contaminates the other tissues. "Teepol" was the only satisfactory surface-sterilizing agent found that inactivated superficial virus without significantly affecting the infectivity of virus contained in the tissues. Embryos dipped in a concentrated tobacco mosaic virus preparation could be freed from contaminating virus by rinsing in "Teepol", and it had a negligible effect on virus in tissues. *N. glutinosa* was inoculated to detect tobacco mosaic virus.

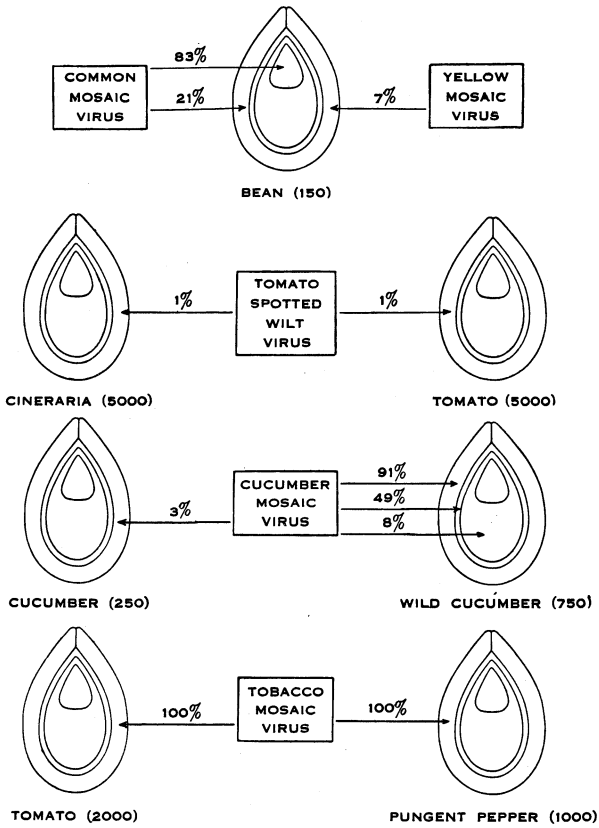

Fig. 1.—Diagrammatic representation of the distribution of viruses in the seeds of various plants. The numbers in parenthesis indicate the number of seeds dissected.

Tobacco mosaic and cubumber mosaic virus are so highly infectious that their detection by these methods presents no problem. The tissues used with the viruses of bean were large enough to ensure their detection if infected. However, tomato spotted wilt is not highly infectious and many of the tissues used were so small that the presence of the virus in the embryos of either tomato or cineraria could have escaped detection. The results of all dissection investigations are presented in Figure 1.

(ii) *Embryo-culture Experiments.*—To eliminate the possibility that the technique was not adequate to detect low concentrations of tomato spotted wilt virus, 900 tomato embryos were grown in sterile embryo-culture on solid "White's Medium" containing coconut milk (van Overbeek, Conklin, and Blakerslee 1942; White 1943). During 4–6 weeks the tomato embryos developed into small seedlings at the four- or five-leaf stage. The amount of development and active photosynthesis which had taken place by this stage would have been sufficient to allow any viruses present to multiply to easily detectable concentrations. The embryo seedlings were next ground and inoculated to tobacco. None was infected.

In all, 340 cineraria embryos were also grown in embryo-culture for 6–8 weeks and then ground and tested. Again none was infected. It was concluded that tomato spotted wilt virus does not infect the embryos of either of these hosts.

(iii) *Seed Transmission.*—It is difficult to understand how seed transmission can occur in cineraria, wild cucumber, and pungent pepper without the embryos of these seeds being infected. Several trials were carried out to determine whether seed transmission did, in fact, occur in these hosts. More than 5000 cineraria were raised from seed of plants infected with tomato spotted wilt virus. Not one infected seedling was discovered. Thus, the 96 per cent. seed transmission reported by Jones must have been obtained either with a most unusual strain of tomato spotted wilt virus, or with a different species of cineraria. A total of 400 wild cucumber seedlings were raised from the seed of plants infected with cucumber mosaic virus. Only one infected seedling was observed. Thus with this virus there seems to be no alternative to the assumption that the strain of cucumber mosaic used was not (or only very rarely) seed transmitted in the conditions of these trials. In pungent pepper, the percentage seed transmission of tobacco mosaic virus varied between 15 and 30. However, as neither embryo nor endosperm of pungent pepper is infected by this virus, seed transmission must result from the contamination of the germinating embryo from the infected testa. This was confirmed experimentally. A sample of freshly harvested pepper seeds was divided into halves: one was sown immediately and the other after the testa had been removed. 25 per cent. of the seedlings of the first lot were found to be infected when inoculated to *N. glutinosa* but not one infected seedling was detected amongst the dissected (testa removed) seed.

(iv) *Changes during Maturation.*—It has been shown above that neither tobacco mosaic nor tomato spotted wilt virus infects the embryos of their hosts at any stage, and no change in the proportion of testas infected by either of these viruses could be detected as the seeds matured. With these two viruses, therefore, the results illustrate virus distribution in seeds at all stages of development. However, the distribution of cucumber mosaic virus in the seeds of wild cucumber altered significantly during their maturation. Table 3 presents the results of one trial in which a total of 235 seeds were dissected at all stages of maturity. To calculate these results, seeds were classed as immature in which the embryo was less than half its mature size. Those in which the embryo had attained its full size and the testa had coloured were classed as mature. As the testa achieves its full size very early in seed development this arbitrary classification was easily made.

As no very young embryos and only one mature embryo was infected, a mechanism in the developing embryo must prevent its infection by viruses present in the endosperm or perisperm. There is no endosperm in mature seeds as this tissue is absorbed by the developing embryo. The decline in the percentage of testas infected could be caused by the inactivation of the virus or the production of inhibitors in the maturing testa.

The results obtained with all five viruses show that the viruses that are not seed transmitted are unable to infect the developing embryos of their hosts, and indicate that seed transmission depends on the ability of a virus to infect the embryo, even if, as with pungent pepper seeds, embryo infection occurs during germination.

TABLE 3

DISTRIBUTION OF CUCUMBER MOSAIC VIRUS IN THE SEED OF WILD CUCUMBER

	Percentage Infected:			
	Testa	Perisperm	Endosperm	Embryo
Immature	91	49	8	0
Mature	27	0	—	0·7

(c) *Effect of Environmental Factors on Seed Transmission*

All of the evidence given above strongly suggests that the lack of seed transmission of the highly infectious plant viruses is not explicable in terms of the three hypotheses that have been advanced. The viruses studied do not induce sufficient sterility to account for their lack of seed transmission: the seeds of the hosts used do not contain any virus-inactivating substances (Crowley 1955), and the developing embryos do not inactivate virus particles in the medium surrounding them (Crowley 1957). The remaining hypothesis that viruses are not seed transmitted because they are unable to infect or survive in the gametophytic cells of their hosts and are unable to infect developing embryos because of the lack of plasmodesmatal connections between embryo and endosperm cannot be investigated directly. However, it is possible to investigate a conclusion that can be drawn from this hypothesis. Those viruses which are pollen transmitted must be able to infect gametophytic cells. Most probably all viruses which are seed transmitted must also be able to infect gametes, for otherwise they must infect the developing embryo despite the lack of plasmodesmata. If the developing embryo becomes infected, the percentage of infected embryos must increase during embryo development. This has been studied with bean mosaic virus, and disproved. It was found impossible to test very young embryos and a more satisfactory method was sought.

The percentage seed transmission of bean mosaic virus varies from 20 to 60 per cent. (Harrison 1935). Environmental factors might be responsible for this variation, because of its great effect on virus concentration in plants, and through this on the

percentage of infected gametes. Temperature is probably the most important environmental factor affecting the virus concentration of bean mosaic virus in beans (Fajardo 1930), and as it is one of the most easily controlled factors, it should be possible to determine whether the percentage seed transmission of bean mosaic virus is affected by the temperature before or after fertilization. Results thus obtained should indicate when embryos become infected: before fertilization (by way of the gametes), or after fertilization (during embryo development), or both.

An experiment to obtain this information was made in two glass-houses: one was insulated by double glass walls and refrigerated to a temperature usually below 62°F, never above 75°F, and the other was equipped with thermostatically controlled heaters to keep the minimum temperature above 68°F; day temperatures here varied between 80 and 90°F.

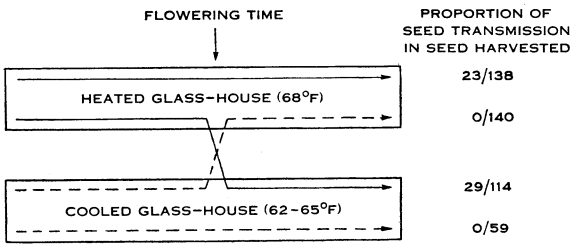

Fig. 2.—Effect of prior- and post-fertilization temperatures on the seed transmission of bean mosaic virus.

Beans (var. Canadian Wonder) were used and were inoculated with common bean mosaic virus at the primary leaf stage. A week after inoculation the plants were divided at random into four lots of 30. These groups of plants were then moved between the heated and cooled glass-houses so that an estimate could be obtained of the relative effect on seed transmission of the environment before and after fertilization. The treatments and the results are set out diagramatically in Figure 2.

In the conditions of this experiment, only the temperature before fertilization was important in determining the percentage of seed infected with bean mosaic virus, which is consistent with the explanation that even this seed-transmitted virus is unable to infect the developing embryo. With bean mosaic virus, and possibly with other seed transmitted viruses, seed transmission depends on the ability of the virus to infect the microspores, macrospores, or embryo sac before fertilization.

III. Discussion

The rarity of seed transmission of plant viruses cannot be attributed to one cause. Seed transmission is impossible for the many viruses that fall into one of the following four classes:

(1) Those which kill their hosts.
(2) Those which prevent flower formation.
(3) Those which are limited in their distribution within their host.
(4) Those which are unable to tolerate the changes that take place in the seed during its maturation and desiccation, e.g. southern bean mosaic virus.

However, these four classes include less than 10 per cent. of the known plant viruses and there must be yet other reasons for the lack of seed transmission of the remaining 90 per cent. Several theories have been put forward and each of these is now examined in relation to the evidence obtained.

Crowley (1955) demonstrated the presence of virus inhibitors in the seeds of several hosts, but failed to detect the presence of virus inactivators. Crowley (1957) also failed to detect the presence of virus inactivators in developing embryos. Duggar's (1930) theory that seed transmission is prevented by the action of virus inactivators in seeds is unsupported by evidence.

Caldwell's (1952) theory that virus-induced sterility prevents the seed transmission of plant viruses is an adequate explanation, but applies to only one or two viruses.

The only explanation for the rarity of seed transmission not at variance with the available evidence is Bennett's (1936) theory that most viruses are unable to survive in the micro- or macrospores or embryo sacs, and apparently all viruses are unable to infect developing embryos because of their lack of plasmodesmatal connections with the surrounding tissues. The results presented here show that none of the four non-seed-transmitted viruses used was able to infect the embryos of its host, and they strongly indicate that the seed-transmitted bean mosaic virus is able to infect the embryos only prior to fertilization. This theory aligns the prevention of seed transmission with the more basic problem of resistance of plants (or in this case of individual cells) to viruses, and with the genetic control of resistance. Couch (1955) found that the genotype of the host is the major factor determining the percentage seed transmission of lettuce mosaic virus, and Cation's experiments (1952) illustrate the complex nature of the interaction and the importance of the strain of the virus. He reported that in seed harvested from a single cherry tree infected with two seed-transmitted viruses, the percentage transmission of one of them (cherry ringspot) was four times as great as the percentage transmission of the other (cherry yellows). If seed transmission depends on the ability of a virus to survive in haploid gametophytic cells, it is to be expected that the genotype of both host and virus would be of major importance, and that different results would be obtained with different strains of a virus or different varieties of a host. Lindstrom (1941) reported a haploid line of tomato to be susceptible to an aucuba strain of tobacco mosaic virus, although only very slight symptoms were produced. Later, however, he reported "the haploid seems to have developed an immunity to the natural infection which was constantly affecting other tomato varieties in the same glass-house." It appears that Lindstrom's lines of haploid tomatoes did possess, if not an immunity, at least a very high degree of resistance to infection by tobacco mosaic virus, and even when infected, they did not support virus multiplication as readily as the diploid. If this resistance to virus infection is a normal characteristic of haploid cells the rarity of pollen and seed transmission is only to be expected. One test of this conclusion would be to determine whether a virus would be seed transmitted in an autotetraploid host. The gametes of such a plant would be diploid, and therefore presumably susceptible.

The low percentage of seed transmission in infected plants where it does occur is difficult to explain. The only reason that can be suggested is that viruses are not present in the nucellar and anther tissues in sufficient concentration to achieve 100 per cent. infection. The rarity of seed transmission of plant viruses has usually been regarded as a remarkable phenomenon. However, natural selection would tend to eliminate any line of plants in which the seed transmission of a severe virus disease was common. In order to be seed transmitted a virus must be able to invade its host systemically to infect and survive in the haploid gametophytic cells, and to survive in the embryo throughout its development, maturation, storage, and germination.

IV. Acknowledgments

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Appendix I

SEED-TRANSMITTED VIRUSES

Host	Virus*	Per Cent. Trans-mission	Reference
CANNABINACEAE			
Humulus lupulus	Chlorotic disease of hop	27	Salmon and Ware (1935)
CHENOPODIACEAE			
Beta vulgaris	Beet yellows	30	Clinch and Loughnane (1948)
COMPOSITEAE			
Cineraria sp.	Tomato spotted wilt†	96	Jones (1944)
Helianthus annuus	Unnamed	17–43	Traversi (1949)
Lactuca sativa	Lettuce mosaic	6	Ainsworth and Ogilvie (1939)
L. sativa	Lettuce mosaic	5	Newall (1923)
L. sativa	Lettuce mosaic	10	Ogilvie *et al.* (1934)
L. sativa	Mosaic	8	Grogan and Bardin (1950)
L. sativa	Yellow mosaic	30	Vasdeuva *et al.* (1948)
Senecio vulgaris	Lettuce mosaic	0·5	Ainsworth and Ogilvie (1939)
CONVOLVULACEAE			
Cuscuta campestris	Dodder latent mosaic	4·8	Bennett (1944)
CUCURBITACEAE			
Cucumis melo	Musk melon mosaic	28–94	Rader *et al.* (1947)
C. melo	Cucumber mosaic	2	Hendrick (1934)
C. melo	Cucumber mosaic	16	Mahoney (1935)
C. pepo	Musk melon mosaic	—	Rader *et al.* (1947)
C. sativa	Cucumber mosaic	1·4	McClintock (1916)
Cucurbita pepo	Squash mosaic	0·96	Middleton (1944)
Cucurbita pepo	Cucumber mosaic	1·5	Chamberlain (1939)
Cucumis moschata	Musk melon mosaic	28–94	Rader *et al.* (1947)
Echinocystus lobata	Cucumber mosaic†	22	Doolittle and Gilbert (1919)
GRAMINEAE			
Hordeum vulgare	False stripe‡	50–100	Gold *et al.* (1954)
H. vulgare	False stripe	86	Hagborg (1954)
Triticum vulgare	False stripe	71	Hagborg (1954)
H. vulgare	False stripe	58	McKinney (1951)
LEGUMINOSEAE			
Dolichos biflorus	*D. biflorus* mosaic	25–40	Uppal (1931)
Glycine soja	Soybean mosaic	10–25	Kendrick and Gardner (1921)
G. soja	Tobacco ring spot	54–78	Desjardins *et al.* (1954)
G. soja	Tomato ring spot	76	Kahn (1956)
Lathyrus odoratus	Pea mosaic	—	Dickson (1922)
Phaseolus limensis	Lima bean mosaic	25	McClintock (1917)
P. vulgaris	Bean mosaic‡	—	Reddick and Stewart (1919)
P. vulgaris	Bean mosaic	43	Archibald (1921)
P. vulgaris	Bean mosaic	20–59	Harrison (1935)
P. vulgaris	Bean mosaic	10–30	Fajardo (1930)
P. vulgaris	Red node	27	Thomas and Graham (1951)
Pisum sativum	Pea mosaic	0·5	Dickson (1922)
Trifolium hybridum	Pea mosaic	0·5	Dickson and McRostie (1922)
T. pratense	Pea mosaic	47	Dickson and McRostie (1922)
Vicia faba	Mosaic	1	Quantz (1953)
Vigna sesquipedalis	Asparagus bean mosaic	37	Snyder (1942)

APPENDIX I (*Continued*)

Host	Virus*	Per Cent. Trans-mission	Reference
LEGUMINOSEAE (*continued*)			
V. sineusis	Cowpea mosaic	14	Gardner (1927)
V. sineusis	Cowpea mosaic	7	McLean (1941)
V. sineusis	Cowpea mosaic	11	Yu (1946)
RUTACEAE			
Citrus aurautifolia	Xyloporosis	66	Childs (1956)
MALVACEAE			
Abutilon sp.	*Abutilon* mosaic	<1	Keur (1933)
ROSACEAE			
Prunus avium (var. Mazzard)	Cherry ringspot	5	Cochran (1946)
P. avium (var. Mazzard)	Cherry ringspot	56	Cation (1952)
P. cerasus	Cherry ringspot	30	Cation (1949)
P. mahaleb	Cherry yellows	7·8	Cation (1949)
P. mahaleb	Cherry yellows	41	Cation (1952)
P. mahaleb	Cherry ringspot	10	Cation (1952)
SOLANACEAE			
Capsicum frutescens	Tobacco mosaic†	22	McKinney (1952)
C. annum	Mosaic‡	—	Ikeno (1930)
Datura stramonium	Q disease‡	100	Blakerslee (1921)
Lycopersicon esculentum	Tomato streak	66	Berkeley and Madden (1932)
L. esculentum	Tobacco mosaic	—	Berkeley and Madden (1932)
L. esculentum	Tobacco mosaic	2	Doolittle and Beecher (1937)
L. esculentum	Cucumber mosaic	0·2	van Koot (1949)
Nicotiana tabacum	Tobacco ringspot	17	Valleau (1941)
Petunia sp.	Tobacco ringspot	20	Henderson (1931)
Physalis peruviana	Tomato bunchy top	29	McClean (1948)
Solanum tuberosum	Virus Y	16	Sprav (1951)
S. tuberosum	Virus Y	14	Reddick (1936)
S. uncanum	Tomato bunchy top	53	McClean (1948)
URTICACEAE			
Ulmus campestris	Elm mosaic	3	Bretz (1950)

*Names of viruses used throughout are taken from the "Virus Index" in *Rev. Appl. Mycol.* **24** (13): 515–56, 1945.

†Results not confirmed in the present paper.

‡Also recorded to be pollen transmitted.