Inoculum Thresholds of Seedborne Pathogens

Viruses

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During recent years, an increasing number of plant viruses have been shown to be seedborne. In 1969, Bennett (4) reported 53 viruses that were seedborne; by 1974, that number had reached 85 (23), and the latest review (17) lists 119. However, when synonyms are taken into account, the actual number is in the order of 100, infecting 162 plant species. Viruses that have been recognized for several years and thought not to be transmitted through the seed may in fact be seedborne, albeit in a very low percentage of seed in some of their hosts. Furthermore, new viruses are continually being discovered, and a portion of these are seedborne. The phenomenon of seed transmission of plant viruses is anything but rare. It would not surprise us if, with further research, close to one-third of the recognized plant viruses were shown to be seedborne in at least one of their hosts.

The term "inoculum threshold" is not used to any extent by plant pathologists. "Inoculum" is a widely used and well understood word, not only with respect to viral pathogens but also for fungal and bacterial pathogens. "Threshold" is in a different category. It is rarely, if ever, used in the plant virus literature, used to some extent in fungal and bacterial literature, and used to a greater extent in entomological literature. When the word "threshold" is used, it is normally combined with a second word to form such phrases as "damage threshold," "action threshold," "control threshold," "warning threshold," "threshold concept," "threshold theory," and "detection threshold"—as in virus concentration necessary for detection by enzyme-linked immunosorbent assay (ELISA), etc. (32). These terms are used in relation to assessments of crop loss, which are difficult to define. As noted by Cook (7), "A grower can lose trees because of nematodes or diseases, or a commodity during storage, but cannot lose what was never produced in the first place, nor can a yield be reduced below some yield that was never achieved." Although we recognize that the terminology used by plant pathologists is changing and the meaning of specific terms may be confusing, in this review we shall combine the terms "inoculum potential" with "damage threshold" and use "inoculum threshold" to convey the concept of the maximum amount of inoculum that can be tolerated without an appreciable constraint to yield and its concomitant limitation of economic return.

As noted by Zadoks (32), the threshold concept, cornerstone of the integrated pest management system, was a great innovation but, unfortunately, it contains an element of exaggeration, because it suggests a sense of reliability that does not really exist (i.e., the damage threshold depends on many variables: the price of the product and pesticide, the distance to the market, and other factors). So many factors affect the damage threshold that the validity of the concept must be questioned. The same criticism can be leveled at our attempt to relate the inoculum threshold concept to seedborne viruses. Hard data are woefully lacking and, even though there is an enormous mass of observations bearing on the ecology of plant viruses, specific information with respect to threshold levels of the various seedborne viruses is available for only a few crop-virus interactions. Despite this limitation, some general conclusions can be gleaned from the literature.

A distinction has been made in the literature (2,4) between those viruses that are retained on the seed surface or in the endosperm or perisperm and those that infect the embryo. This is an important distinction, because embryo infection virtually assures that the ensuing seedling is infected, whereas viruses occurring in other parts of the seed do not result in seedling infection unless they are resistant to inactivation and are highly infectious by mechanical transmission. When the virus survives as a contaminant in or on the seed, it may be eliminated or significantly reduced by heat therapy or chemical treatments. Viruses that infect the embryo cannot be eradicated without loss of seed viability. For this reason, the viruses that are transmitted via the embryo are particularly difficult to control and consequently are of considerable epidemiological significance. Those viruses that do not infect the embryo but do infect or infest other parts of the seed are usually of minor epidemiological significance although they may be of considerable economic importance (e.g., tobacco mosaic virus in pepper and tomato).

In this review, we have no intention of writing yet another article on the general subject of virus transmission through seed. Indeed, the subject has been thoroughly covered in the review articles cited above, and all that is lacking is a compilation of those viruses that have been added to the seedborne category since about 1980, when the literature for the last review (17) was completed. Our intention is to critically examine the ecological factors influencing viral epidemiology and where possible to assess what level of damage can be reasonably tolerated.

FACTORS CONTRIBUTING TO INOCULUM THRESHOLD

The first report on the potential importance of seedborne viruses is attributed to Doolittle and Gilbert (9), who noted that wild cucumber is susceptible to cucumber mosaic and that the disease appears in the wild host 3–4 wk before it is found in cultivated cucumber in the field. When they demonstrated that a proportion of the seed from infected wild cucumber plants produced diseased plants the following year, they concluded that this seedborne inoculum was of considerable importance in the overwintering of the disease and as a primary inoculum source. Despite this early observation, relatively little importance was attached to the economic implications of virus transmission by seeds for the next several decades, perhaps because of the belief that few viruses were seedborne and that those recognized to be seedborne were associated with local spread of diseases in annual crops. This myth was further perpetuated by imprecision of early virus epidemiological investigations. The disease-producing potential of seedborne viruses was neither understood nor appreciated until the various factors contributing to disease epidemiology were defined and investigated.

Substantial reduction in yield potential may be overlooked before the cause of a disease is identified, particularly when a virus is only one component of a disease complex. Even after viral etiology has been established, disease incidence is difficult to measure by visual inspection, the technique that was most widely used in early epidemiological studies. Visual inspection is a particularly poor technique for assessing the incidence of seedborne viruses in seedling progeny because the symptoms are sometimes so subtle that even a trained observer has difficulty distinguishing infected seedlings from healthy ones. In general, infected seedlings are somewhat stunted and, in this respect, symptoms resemble those of chronically infected plants. Such infected plants could readily escape notice if they are growing in a field planting where other factors may contribute to a scattering of unthrifty plants.

Virus epidemiological studies tend to be site-specific, and results from studies on a virus in one region may not be applicable to the same virus in another region because of differences in cultural practices, weather, soil type, vector populations, alternative sources of virus, and reliability of detection techniques. Some or all of these factors have a bearing on virus epidemiology, and seedborne inoculum may be important in one disease occurrence but insignificant in another. The false premise that seedborne inoculum is but another source of virus may indicate a failure to appreciate the biological implications of this unique inoculum source.

Doolittle and Gilbert (9) identified two significant aspects associated with a seedborne virus that may act as constraints to potential yield: 1) seed transmission provides a strategy whereby the virus can survive under conditions in which other sources of inoculum have been eliminated, and 2) the seedborne inoculum creates centers of infection from which the virus can be vector-transmitted to nearby cultivated plants. It is interesting to note that this early report involved seed transmission in a weed rather than a crop host. Over the years, the importance of other seedborne viruses in weed hosts has been recognized. Moreover, other aspects that contribute to economic damage have been identified. In this review, we shall attempt to identify these aspects and provide a few examples of each. Furthermore, we shall relate these, insofar as is possible, to recognized plant virus groups.

Survival of inoculum. With a few exceptions, plant viruses are extremely fragile and are thus incapable of surviving for more than a few hours outside living host tissue. This characteristic does not affect the survival of those viruses that are able to invade perennial hosts, but it is a serious disadvantage for those viruses that infect annual plants, particularly in areas of the world where the annual hosts are killed by cold or drought, resulting in a period during the year in which perpetuation from inoculum sources is limiting. During this critical period, the virus could be eliminated unless a survival mechanism had been evolved to ensure perennation. An effective strategy would be to somehow link survival of the virus, which is an obligate parasite, to the survival of its host plant. In essence, this linkage is established when the virus invades developing embryos of the host-plant seeds. With most virus-host combinations involving seedborne viruses, the virus survives in the embryo for as long as the seed remains viable. The nature of the survival form of the virus is not known. There is no reason to assume, as a requisite for seed transmission, that the survival form is a virion which, during seed germination, would be required to reinitiate an infection process (i.e., rearrangement and disassembly of the protein coat, followed by release of the genomic nucleic acid and its subsequent translation into gene products that are required to support the infective process). As Hamilton (12) speculated, recent advances in the molecular biology of viruses suggest other bases for the transmission of plant viruses via infected embryos (e.g., incorporation of viral RNA in the plant genome or transmission by viral replicative RNA or a specialized form of genomic RNA).

There are many well documented cases where seed infection is a dominant factor in virus survival. In many of these cases, it is not seed from a crop host but rather seed from a weed host that presents the opportunity for virus survival. Viruses are seedborne in their weed hosts at approximately the same frequency as they are in their crop hosts, but in many instances a much higher proportion of weed seed remains in the soil. Tomlinson and Walker (27) found that cucumber mosaic virus persisted in the seeds of *Stellaria media* (L.) Cyrill. in soil from one year to the next, providing a means of perennation of the virus and ensuring its availability for aphid transmission to vegetable crops throughout affected fields. Similarly, Murant and Lister (21) found that infected weed seeds allowed some nepoviruses to survive conditions under which nematode vectors ceased to feed.

Some viruses appear to be solely dependent on seed transmission for their survival. An example here is barley stripe mosaic virus (BSMV), which is seedborne to a high degree but has no known vector. BSMV is known to be present in most barley-producing areas of the world and, because no weed or wild grass is a significant reservoir of the virus, it depends on seed-transmission in a single plant species, *Hordeum vulgare* L., for survival from year to year. Survival is relatively independent of environmental or other external factors and more dependent on the virus strain-host cultivar combination (26). Those combinations found in nature usually result in mild or moderate reactions (i.e., virus-host compatibility), which favor continuous survival of both the host and virus. Those cultivar-strain combinations that maintain a high rate of seed transmission would cause the host plants to succumb to the disease if the viruses were severe in their reaction. Alternatively, combinations in which a barley cultivar is highly resistant would favor survival of the cultivar and destruction of the virus strain. A compatible strain-cultivar combination is therefore a critical factor in the successful perpetuation of BSMV.

Dispersal of virus. An obvious consequence of the survival of virus in seed is the opportunity provided for virus dispersal via movement of seed. Aside from seed commerce, virus dispersal would be confined to relatively short distances, depending on the efficiency of transport by wind and water. Long-distance spread of seed (e.g., by birds) and its associated seedborne viruses could be accomplished by the cumulative effect of a series of movements over a long period of time. However, with human assistance, seedborne viruses can be introduced in seed lots to countries where the particular virus is not known to occur. When the seeds are planted, optimum conditions for an epidemic may occur if a suitable climate and vector are present. Even if a suitable vector is not available where the seed is planted, there is a possibility that the virus will be maintained in self-propagated or perennial material until a strain that is compatible with a local vector develops (25).

Dissemination of virus in seeds rather than by means of a vector appears to be a reversal of the usual order of events in plant virus ecology. An example of seed dispersal overshadowing vector dispersal is with the viruses belonging to the nepovirus group. Dissemination of these viruses in infected seed, particularly weed seed, explains why they are widespread, despite the relative immobility and space confinement of their nematode vectors. Alternative means of dissemination (i.e., other than the vector) are therefore decisive in the epidemiology of the nepoviruses. Although it is difficult to prove that virus spread actually occurs via infected seed in nature, this can be inferred by the natural occurrence of infected seed in soils (21).

Primary inoculum source. Seed infection is epidemiologically important because it ensures that the virus will be associated with the planted crop, because infected seeds are randomly dispersed in the field, and because the infected seedlings serve as sources of inoculum from which secondary spread can be initiated. When the infected seedlings are virtually the only source of inoculum, seed transmission plays a critical role in virus epidemiology. There are many instances in plant virus epidemiology when seedborne viruses appear to be the sole or the primary source of inoculum and, in considering the inoculum threshold of seedborne viruses (i.e., the maximum amount of inoculum that can be tolerated), this aspect is by far the most significant.

An example of a virus that is spread primarily by mechanical contact is tomato mosaic virus (ToMV). About one-half of the seeds from infected tomato fruit carry the virus, the proportion differing with tomato cultivar, time of infection, truss position, and method of cleaning. The virus is usually carried on the seed surface, but it may be carried within the testa or the endosperm. No evidence of embryo infection has ever been obtained (5). ToMV persists in the testae for several months or years after harvest. Seedlings are not infected when left undisturbed, but infection occurs when virus carried by the testae contaminates the seedlings by entering through mechanical abrasions made during transplanting. Because of the usual frequent handling of plants during their culture, only a few seedlings need to be infected in a tomato crop for the virus to spread rapidly.

Two beetle-transmitted viruses, broad bean stain and broad bean true mosaic, are also seedborne. Observations suggest that infected seed is the main source of these viruses in spring-sown field bean crops in the United Kingdom (6). Although weevils may occasionally bring these viruses into crops, the most effective means of controlling the spread of the viruses and of decreasing the damage they cause is to ensure that crops are grown from seed that is as free as possible from infection. For example, 5–14% infection was observed in bean crops shortly after flowering, following planting of seed with 0.1% infection.

Seedborne viruses that are also transmitted by aphids are of particular concern when infected seed serves as the primary source of inoculum. Some of the most economically important plant virus diseases are in this category. This appears to be the case for bean common mosaic virus (BCMV), blackeye cowpea mosaic virus, lettuce mosaic virus (LMV), cucumber mosaic virus, peanut mottle virus, peanut stripe virus, and soybean mosaic virus (SMV). Critical experiments to determine the inoculum threshold of the aphid-transmitted, seedborne viruses are difficult to undertake, particularly in the vicinity of the crop plant. Adams and Kuhn (1) found that a 0.1% level of seed transmission is significant in the epidemiology of peanut mottle disease.

The most reliable data relate to controlling LMV disease in the California lettuce crop (11). This virus is a good example of one that is seedborne at a low level and yet can induce an epidemic from only a few infected seedlings. Trials conducted in California attempted to answer the question of how much LMV-infected seed could be permitted while still providing acceptable control of the disease. A seed transmission rate of greater than 0.1% is usually unsatisfactory. Although this low level provided adequate control in the earliest planting, inoculum accumulated on overlapping plantings during the long growing season. This tolerance level was inadequate, and the disease was still prevalent, over a 10-yr test period, causing severe losses over much of the lettuce acreage. A revised standard of 0 seedborne LMV in 30,000 seedlings was adopted to ensure that the level of seed transmission was nil or very low. This standard, coupled with 1) eliminating potential weed hosts in and near borders of lettuce fields, 2) avoiding the planting of new lettuce fields adjacent to old fields, and 3) disking lettuce fields immediately after harvest has provided good control in California since 1963. Although seed lots that index 0 in 30,000 may not be completely virus-free, this stringent standard ensures that all approved seed stocks have a very low level of seedborne LMV.

LMV is a typical potyvirus in that it is naturally spread in a nonpersistent manner by aphids and experimentally by mechanical inoculation of sap, is transmitted to some extent through seed, and has a restricted natural host range (15). It differs from most other potyviruses in infecting a crop of high economic value, causing sufficient damage to warrant expensive control programs, and having a low level of seedborne virus as essentially the sole source of inoculum. This combination of factors has stimulated efforts to control the virus by using virus-free seed for planting the crop, coupled with an enforced fallow period during which all other potential inoculum sources are eradicated. The current commercial trend in Great Britain is directed towards the use of cultivars containing LMV-resistance genes, with the possibility that resistant cultivars will replace the present control strategy of using virus-free seed (30). Should this happen, the tolerable inoculum level of LMV could be dramatically reduced.

Contamination of germ plasm lines. Plant breeders have a broad concern for the maintenance and preservation of germ plasm, which involves collection of plant species from various parts of the world. It is difficult to compare the evolution of plants with that of viruses, but the evidence suggests that viruses probably coevolved with their hosts. By extrapolation, one could generalize that valuable germ plasm collections are also valuable virus collections. This generalization is true whether viruses are seedborne or not, but in the context of this paper, we shall confine our remarks to germ plasm collections involving seedborne viruses.

Seedborne viruses may be of considerable importance as contaminants in germ plasm lines, particularly when the virus is generally absent in commercial fields and may become a problem only when it escapes from plant breeding plots or when advanced breeding lines are contaminated. A notable example is SMV, a virus that has been distributed worldwide through soybean seed, probably since the international movement of soybean germ plasm began some two centuries ago (10). Another example is pea seedborne mosaic virus (PSbMV), which was found to occur independently in several commercial and institutional breeding programs in the United States. Of the 420 *Pisum* breeding lines that were found to be infected, 144 were introduced into the United States in 1970 and 1971, principally from India (13). In both instances, promising control has been achieved by identifying germ plasm sources that show a high degree of immunity to the virus, coupled with a low incidence of seed transmission.

It is probable that virtually all crop breeding programs are plagued to a certain extent by seedborne viruses in germ plasm lines. In an examination of the germ plasm accessions associated with 17 major crops in the United States, seedborne viruses were either known to occur or expected to occur in every crop (14). The germ plasm lines have frequently coevolved with their seedborne viruses, so symptoms are mild or completely masked, with the result that the viruses are widely distributed before they are even detected. Many seedborne viruses are often detected in breeding plots for the first time, thus emphasizing the importance of germ plasm as a reservoir of viruses and the role that germ plasm exchange plays in dissemination of seedborne viruses. Recognition of this problem coupled with increasing emphasis on virus detection techniques should facilitate control of seedborne viruses in germ plasm lines, despite the inevitable expansion of international germ plasm exchange.

Contamination of virus-free planting material. There are no practical treatments to cure virus-infected plants once they are set out in the field, so the production and propagation of virus-free planting material is the only practical method of controlling virus diseases in many crops. It is the initial phase of propagation in which virus freedom is the most critical, because this is the material that is used to establish foundation blocks for production of certified planting stock. The material certified for virus freedom includes tubers, seedlings, seed, runner plants, cuttings, scion wood, and clonal rootstock. There are many references in the virus literature of planting material in certification schemes becoming contaminated with seedborne viruses. A few representative cases will be reviewed here.

Certification schemes for the production of stone fruit scion wood prescribe standards of scion wood selection and a degree of isolation from possible virus sources. Such certification schemes are of limited value if seedling rootstocks are carrying seedborne viruses. Two ilarviruses, prunus necrotic ringspot virus (PNRSV) and prune dwarf virus (PDV), are seed-transmitted in many *Prunus* species. Most stone fruit certification programs specify that *Prunus* seedling lots must be tested for these viruses, and to meet certification standards, they must contain fewer than 5% virus-infected plants.

Because the demand for certified seed is greater than the supply available in most years, nurseries often purchase large amounts of *Prunus* seed of unknown virus content, some of which exceeds the 5% tolerance (20). Actually, even a 5% tolerance level results in the production and distribution of contaminated budwood. The development of the ELISA technique for the routine detection of PNRSV and PDV has provided guidelines for a more stringent tree-fruit certification scheme in Great Britain (28) and for a *Prunus* seed and seedling monitoring program in North America (20). There is every reason to believe that a zero tolerance level for these two viruses in *Prunus* seedlings can be achieved and will be demanded in the future.

Avocado, another plantation crop that is propagated on seedling rootstocks, is susceptible to sunblotch disease, caused by avocado sunblotch viroid (ASV), which is seed-transmitted to a high percentage of seedlings. Seedlings from a diseased, but symptomless, parent tree may be infected and yet appear symptomless. If these seedlings are used as rootstock for budding of scion wood, the viroid infects the scion. A serious problem may arise, where the scion is also symptomless so that the viroid remains undetected. Some seed sources regularly produce symptomless seedlings, and the use of such sources for rootstock has been responsible at times for a high level of infection in commercial nurseries (31).

ASV is an example of a serious viroid disease that is perpetuated mainly by the practice of grafting budwood onto infected seedlings. The main reason the disease became a problem is that the plants arising from infected seeds were symptomless and there was no suitable technique for detecting the viroid. The detection problem has been solved by using the polyacrylamide gel electrophoresis (PAGE) technique to detect the viroid RNA in infected leaf tissue (29) or flower buds (8). The PAGE index method can be completed within 6 hr and is virtually 100% reliable. A more sophisticated method uses complementary DNA in a dot blot hybridization procedure on partially purified nucleic acid extracts to detect ASV (3). Because ASV does not appear to spread naturally by means other than seed or pollen, there is every likelihood that it will cease to be a problem now that effective detection techniques are being coupled with certification schemes.

Direct injury to crop. Of the various types of potential constraint to yield associated with seedborne viruses, direct injury to the crop was probably a significant factor in the past in instances where symptomless or very mild infections were not recognized and seed transmission often occurred at a high level. Currently, at least in those parts of the world where intensive agriculture is practiced, certified seed sources are available and, provided that adequate cultural procedures are followed in producing the seed and relatively low tolerance levels are set for seedborne viruses, direct injury to the crop is insignificant. However, if certified seed is not used and the virus incidence is high in those plants that are used as a source of seed for future crops, direct injury could be appreciable.

Reports of significant direct injury are rare. Chronic infection of beans by the seedborne BCMV in Morocco is one such report (16). The most widely cultivated bean cultivar is susceptible to locally occurring strains of BCMV, and those plants that are infected via the seed are severely stunted and produce few pods. Seed obtained from local seed companies produced no infected plants, whereas seeds purchased from urban or rural markets were infected to varying degrees. When seed had a high incidence of BCMV, potential yield was reduced by about 50%, with the indication that potential yield loss resulted primarily from the seedborne virus rather than from secondary spread. In these cases, the role of the insect vector would be to increase the percentage of contaminated seed used in the following sowing. The problem can be readily controlled by implementing a seed certification program, by persuading growers to purchase fresh planting stock each year from reputable suppliers, or by using varieties that are immune to infection with BCMV.

In contrast to the severe symptoms induced by BCMV, one of the major characteristic features associated with most seedborne viruses is the ability of infected embryos to survive in the seed and produce seedlings that are almost symptomless. However, the degree to which infected plants show symptoms varies considerably with different viruses and different host plants. The so-called "shock reaction" that is commonly observed in plants infected at the seedling stage or later is absent in seedlings arising from embryo-infected seeds, perhaps because the shock that is associated with the initial infection has already occurred in the dormant embryo before germination. A typical example is tomato ringspot virus infection in strawberry (*Fragaria vesca* L.). Plants that are infected as a consequence of grafting show shock symptoms consisting of necrosis, chlorosis, and epinasty. These plants eventually recover and exhibit chronic symptoms (i.e., leaves slightly smaller than normal, leaflets slightly rounded, and petioles somewhat red and swollen). More than 50% of the seed from chronically infected plants is infected (19), but the infected seedlings show none of the shock symptoms observed in graft-inoculated plants, although most seedlings do show symptoms that resemble the chronic symptoms exhibited by the grafted plants.

Another seedborne virus that may result in considerable direct reduction in yield potential is BSMV. Highly susceptible cultivars are distinguished by the production of little or no seed, but such reactions are rare in nature because they result in a destruction of both the host plant and the virus. More common are the virus strain-host cultivar combinations in which there is a high level of seed transmission but only a mild reduction in potential yield. The initial level of infection, combined with the rate of secondary infection by leaf contact, determines the potential reduction in yield. Yield in barley is a product of heads per unit area, number of seeds per head, and seed weight. Infection with seedborne BSMV causes a reduction in each of these yield factors. If little spread occurs in the field, the reduction in grain yield, number of heads, and seed weight appears to be linear in response to the level of seed infection (22).

SEED TRANSMISSION AND TAXONOMIC GROUPS

Bennett (4) noted that seed-transmitted viruses have certain general characteristics in common. Most are readily sap-transmissible, indicating an ability to invade parenchymatous tissue. Symptoms consist chiefly of mottling, local chlorotic or necrotic lesions, etch, and other types of abnormalities having their origin in parenchymatous tissue. Several are characterized by a "shock" reaction in the host followed by recovery. Viruses transmitted by certain types of vectors are more often seedborne than those transmitted by other types of vectors. For example, viruses transmitted by leafhoppers and those transmitted by aphids in a persistent manner are not seedborne, whereas those transmitted by nematodes, beetles and, in a nonpersistent manner, by aphids may be seedborne.

Bennett (4) was unable to relate his generalized conclusions to the taxonomic grouping of viruses, because meaningful schemes of plant virus classification were just beginning in 1969 and similar viruses were not yet classified into orderly groups. He therefore tabulated the list of known seedborne viruses in an alphabetic order, a practice that has been retained in more recent tabulations (17,24). These tabulations are valuable, but there is merit in examining the phenomenon of seed transmission and its relative significance in those viruses that are included as recognized or possible members of established virus groups. This does not provide a complete picture, because some viruses that are known to be seedborne (e.g., raspberry bushy dwarf virus) are yet to be placed in a virus group. Table 1 lists the virus groups approved by the International Committee on Taxonomy of Viruses, the number of members and possible members in each group, the number of members where seed transmission has been demonstrated, and the type of injury most commonly associated with seed transmission.

Several generalized conclusions may be drawn from the data summarized in Table 1. First, the phenomenon of seed transmission occurs in approximately 18% of those viruses to be accorded recognition as members or possible members of established plant virus groups. Second, although seed transmission has been recorded for one or more viruses belonging to 21 of the 28 groups, it is considered to be of economic significance in only 10 of the groups (i.e., those groups in Table 1 where one or more types of potential injury are indicated). Third, injury attributed to virus survival, virus dispersal, and primary inoculum source (A, B, and C in Table 1) are commonly associated with and are the dominant types of potential injury caused by seedborne viruses. Fourth, of the 11 virus groups that contain seedborne viruses of economic significance, only the potyvirus group contains viruses that are associated with all types of potential injury.

TABLE 1. Relative importance of seed transmission of viruses that are considered to be members or possible members of the recognized virus groups

	Number of members		Type of potential injury[b]					
Virus group[a]	in group	seedborne	A	B	C	D	E	F
Alfalfa mosaic	1	1	+	+	+			
Bromovirus	5	1	+	+	+			
Carlavirus	47	2						
Caulimovirus	7	0						
Closterovirus	15	1						
Comovirus	15	6	+	+	+			
Cucumovirus	4	4	+	+	+			
Dianthovirus[c]	3	0						
Geminivirus	16	1						
Hordeivirus	3	2	+	+	+			+
Ilarvirus	13	8					+	
Luteovirus	29	0						
Maize chlorotic dwarf	2	0						
Maize rayado fino[c]	4	0						
Necrovirus[c]	3	1						
Nepovirus	30	22	+	+	+			
Pea enation mosaic	1	1						
Plant reovirus	9	0						
Potexvirus	38	4						
Potyvirus	117	16	+	+	+	+	+	+
Rhabdovirus	74	1						
Rice stripe[c]	6	0						
Sobemovirus	10	2						
Tobamovirus	18	7	+	+	+			
Tobravirus	3	3	+	+	+			
Tombusvirus	11	1						
Tomato spotted wilt	1	1						
Tymovirus	18	3						
Viroids	15	5				+	+	

[a] Based on Fourth Report of the International Committee on Taxonomy of Viruses (18). Viroids are included for comparative purposes.
[b] A, survival of inoculum; B, dispersal of inoculum; C, primary inoculum source; D, contamination of germ plasm lines; E, contamination of virus-free planting material; and F, direct injury to crop (see text for details).
[c] Group names approved at meeting of International Committee on Taxonomy of Viruses, Sendai, Japan, Sept. 1–7, 1984 (unpublished).

CONCLUSIONS

In our review of the literature on inoculum thresholds of seedborne viruses, we searched for experimental data that would provide an indication of the maximum amount of seedborne virus inoculum that could be tolerated without an appreciable constraint to yield. It is a regrettable fact that this aspect of seed transmission has been critically studied with only a few host-virus combinations. These few cases have several characteristics in common: 1) The level of seed transmission does not have to be high; in fact, it can be exceedingly low and still be of critical importance when the few infected seedlings arising from contaminated seed constitute the sole source of inoculum and when the virus is readily acquired and actively vectored in a crop. 2) The crop must be annual, because infected seed usually would not be the sole inoculum source in perennial crops. 3) The vector is an aphid and the virus is transmitted in a nonpersistent manner. 4) The virus is confined to a narrow natural host range. 5) When the virus is sufficiently characterized to justify inclusion in a virus group, it would belong to either the potyvirus or cucumovirus group. Viruses meeting these criteria would have an inoculum threshold of zero or close to zero.

Those seedborne viruses that are not as actively vectored but may constitute the sole source of inoculum are epidemiologically important, and although tolerable levels are not generally stated, it is obvious that the levels must be low. For those seedborne viruses that have a broad natural host range, including both annual and perennial crops, seed transmission may play an insignificant role in virus epidemiology, and consequently, a high inoculum level could be tolerated.

If a new or little characterized seedborne virus disease is being studied, assignment of the causal virus to an established virus group may have predictive value in estimating an inoculum threshold.

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