PRINCIPAL VIRUS DISEASES OF SWEET POTATOES, THEIR CONTROL AND ERADICATION

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INTRODUCTION

Sweet potatoes (Ipomoea batatas (Lam.) L.) harboring graft transmissible agents which cause diseases can be found in nearly every commercial sweet potato planting not produced from disease-indexed planting material. The reason for this phenomenon is the necessity to propagate sweet potatoes with vegetative organs to preserve varietal purity. Vegetative propagation provides an excellent vehicle to perpetuate these agents from one planting to the next. It is generally assumed that viruses or viroids are the cause of these diseases. Indeed, at least five distinct viruses and a mycoplasma-like organism have been associated with these diseases (Table 1). However, the etiology of many of these sweet potato diseases remains to be determined (Table 1).

Early on in the development of information on sweet potato viruses each new disease thought to be caused by a virus was frequently given a new name. The name was usually indicative of the symptoms expressed in the genotype in which the disease was described. It has seldom been possible to compare biological or biochemical characteristics of the causal agent(s). This practice has lead to the problem of synonymy similar to that which occurred in the early studies of virus diseases of other crops (4, 21). Our knowledge of the viruses which cause these diseases has been slow in coming because the sweet potato-virus pathosystems have not been adequately developed. The viruses are difficult to mechanically transmit to and from sweet potato, virus-indexed sweet potato is not always available for completion of Koch's Postulates, high quality antiserum for virus comparison and indexing purposes is not readily available because of the difficulty in purifying these viruses and there has been no internationally agreed upon criteria for sweet potato virus identification.

The viruses that infect sweet potato interfere both directly and indirectly with the farmers ability to realize the full potential of sweet potatoes. Viruses have been implicated in acute and chronic sweet potato diseases. The acute diseases such as the sweet potato virus disease complex found in Africa (25) and russet crack (3, 4) and internal cork (22) found in the United States have a dramatic impact on production of edible roots. Chronic diseases such as those caused by various strains of sweet potato feathery

mottle virus (SPFMV) (20) have a less severe effect on individual plants but still may significantly reduce yield when considered on a regional basis. This reduction in grower efficiency may go unnoticed when all the plants in the area are infected with the same virus(es).

Grower efficiency is indirectly reduced by the justified impediment viruses cause to the international exchange of germplasm and, thus, slow the introduction of desirable traits into national breeding programs to resist stresses and generally improve yield potential. This should not be interpreted to imply that quarantine regulations should be relaxed. In general, quarantine standards are based on the available biological information. Our lack of understanding of sweet potato viruses has greatly contributed to the conservative establishment of standards to reduce the probability of introducing new pathogens. Improved understanding of this group of pathogens will significantly increase the confidence with which these standards are upheld as well as providing more efficient technologies that can be used to document the status of plant health.

The status of sweet potato viruses is, however, not a hopeless one. The resurgence of interest in sweet potatoes has already begun to promote additional research into sweet potato viruses. Only a few viruses have been identified and there are many disease syndromes with an unclear etiology (Table 1). Future research should be directed at obtaining a complete understanding of the biological and biochemical properties of these viruses. Only then can we devise the most efficient and expeditious strategies to control, and hopefully eradicate, these viruses from our germplasm stocks. These same procedures may well be extended with minimal modification to evaluate germplasm and breeding material for resistance to these viruses.

The solution to the problem of viruses in sweet potato germplasm will include research in several areas. Little can be accomplished until we more fully understand the etiology of these diseases. Only then can effective and reliable virus-indexing or detection procedures be developed. Then a source of "healthy" genotypes can be made available for distribution to facilitate biological comparison of viruses and to serve as indicator hosts, especially where biochemical assays may not be feasible.

Virus Identification

There are many sweet potato diseases suspected of having a viral etiology that have been reported from all over the world (Table 1).

The named viruses identified from sweet potatoes, are sweet potato feathery mottle virus (SPFMV) (3, 4, 13, 19, 20), sweet potato mild mottle virus (SPMMV) (12), sweet potato vein mosaic virus (SPVMV) (23, 24), sweet potato latent virus (SPLV) (6, 15) and cucumber mosaic virus (CMV) (J. C. Thouvenel, Ivory Coast). SPVMV is a long flexuous rod but has not been purified and its relationship to other viruses is unknown (24). SPFMV, SPLV, and SPMMV are all long flexuous rods (750 - 900 nm), but are not serologically related (A. A. Brunt, R. J. Chiu, personal communication and J. Moyer, unpublished data). In general, though, there remains much confusion over the identity of sweet potato viruses (4, 21).

SPFMV is the predominant virus infecting sweet potatoes in the United States (4, 13, 20). Research in progress has also shown that strains of SPFMV occur in virtually every country where sweet potatoes are grown (17). This virus is a member of the potyvirus group, but has an unusually large capsid protein and long virion with a correspondingly large RNA genome (19). The virus virion and RNA are 10 - 15% larger than in most potyviruses. However, other biological properties and the presence of pinwheel-type cytoplasmic inclusions are consistent with characteristics of the potyvirus group (4, 14, 20). The other viruses isolated from sweet potato have only been partially characterized and high quality antiserum for comparative purposes and for indexing are not yet available for all of those viruses.

Two new virus-like agents, which are distinct from SPFMV, are also currently under investigation in my laboratory. One virus was isolated from sweet potato plants exhibiting distinct chlorotic mottling. The intensity of the symptoms, the chlorotic pattern and failure of the symptoms to go into remission were all characteristics of a disease syndrome distinctly different from that caused by SPFMV. In addition, sap from infected plants did not react serologically with SPFMV antiserum. Research on the second virus-like agent has recently been initiated to identify the cause of a disease with symptoms similar to those described for Georgia Mosaic or Yellow Dwarf (8, 10).

There are many other sweet potato diseases which have one or more characteristics that suggest a viral or viroid etiology (Table 1). The vectors are known for some of the agents associated with these diseases such as the sweet potato virus disease and sweet potato leaf curl (6, 25). Unfortunately, there are many other disease syndromes whose causal agents have been graft transmitted, but little additional information is available. Indexing sweet potatoes for these disease agents is cumbersome at best and then the evaluation of large number of genotypes is not practical.

Indexing

Indexing is a term that is used by plant pathologists to denote the monitoring of plants for the presence of pathogens in order to maintain and provide "healthy" plants for propagation and dissemination. The development of indexing procedures can be divided into four tasks. First, the target pathogens must be identified such as is described above. Second, potential techniques or procedures should be evaluated to determine the most effective strategy for that host-pathogen interaction. Third, the selected procedure should be tested thoroughly to ensure that it has a sufficiently broad spectrum of recognition to detect all forms of the target pathogen, yet retain enough specificity to minimize false positives. Fourth and most important, particularly for perennial crops or vegetatively propagated hosts, the life cycles of the pathogen and the host should be examined to determine when indexing can be conducted most reliably.

Assays

The approach in my program has been to use SPFMV as a model for the development of virus indexing procedures in sweet potato. Hopefully, much of this information will be applicable for the new viruses described in the previous section. Indexing for SPFMV and other graft transmissible agents has traditionally been conducted by grafting scions from sweet potato into I. setosa or other sensitive Ipomoea spp. (11). This procedure requires considerable time to conduct and for symptoms to be expressed. Symptom expression is dependent upon environmental influences (e.g. 1, 22) and we have recently found that there is also variability in symptom intensity and duration due to the strain of SPFMV (17).

In efforts to improve indexing procedures for SPFMV (2, 7), we have emphasized an evaluation of methods which detect the presence of viral proteins through serological assays. Currently, we are focusing on the problems of extremely low virus titer and the presence of substances in sweet potatoes (e.g. phenolics, phenol oxidases, quinones, latex and carbohydrates) which interfere with these assays. At the present time conditions which favor optimum sensitivity and reduced nonspecific interactions have been identified (18); and we have produced an antiserum which has been shown to be specific for all known strains of SPFMV (3, 18, 20). We anticipate that many of the sweet potato related problems have been elucidated and that modifications will be required for new viruses.

Pathogenesis

The temporal cycle of SPFMV in sweet potato is of both practical and theoretical interest. It is of practical importance to know when and/or under what environmental conditions virus titer (accumulation) is favored in order to develop the most effective procedures for indexing. (See above). SPFMV is acknowledged to be highly "variable" and unevenly distributed in its occurrence during the life of the sweet potato plant (2, 7, 10). Ourselves (7) and others (2) have repeatedly demonstrated that the use of arbitrary sampling procedures used to assay other crops are inadequate to insure detection of SPFMV in sweet potato. It is my opinion that reliable indexing procedures for sweet potato viruses must be based on a thorough understanding of the biology of these viruses in sweet potato.

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