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Sterility Mosaic Disease—the "Green Plague" of Pigeonpea

Advances in Understanding the Etiology, Transmission and Control of a Major Virus Disease

Sterility mosaic (SMD) is the most damaging disease of pigeonpea (*Cajanus cajan* (L.) Millsp.) in the Indian subcontinent. After seven decades of research, vital breakthroughs made on the identification, detection, and transmission of the causal agent of this major disease are enabling the development of broad-based durable resistant pigeonpea cultivars. These breakthroughs will contribute greatly to sustainable pigeonpea production and enhance the income and livelihood of poor farmers in the semiarid tropics of the Indian subcontinent.

The Crop

Pigeonpea (Fig. 1) is the most versatile grain legume crop grown in the semiarid tropical and subtropical regions between 25° N and 30° S in Asia, Africa, and the Americas (Fig. 2) (76). It is cultivated on 5.25 million hectares, with annual production of over 3 million tonnes contributing to about 5% of the total world production of pulses (Food and Agriculture Organization of the United Nations [FAO], published online). Nearly 90% of the global pigeonpea cultivation is confined to India and Nepal, the remainder is in Africa (6%), the Caribbean (2%), and other Southeast Asian countries (Fig. 2).

Pigeonpea is a very important subsistence crop in marginal farming systems adopted by millions of smallholder farmers in the Indian subcontinent (Figs. 2 and 3). The fast-growing, deep, extensive root system allows plants to grow and produce grain in very arid conditions and in drought years when no other crop can survive. Furthermore, the slow growth of the plant above ground during its early phase offers very little competition to other crops and allows productive intercropping with virtually any crop. It is grown as a sole crop or as an intercrop mixed with cereals (maize, sorghum, pearl millet, finger millet), fiber and other legume crops (groundnut, soybean) under wide climatic conditions in rain-fed low-input agricultural systems.

Pigeonpea is grown for its seed for human consumption and for income generation by trading surpluses in local and commercial markets, but it is widely used for diverse purposes (11; Fig. 1). Pigeonpea assimilates more nitrogen per unit of plant biomass than most other legumes, can nodulate in most soils, and mobilizes soil-bound phosphorus. This benefits both the pigeonpea crop and subsequent crops in rotation, thus contributing to increased productivity and soil amelioration (1,12). Because of these features, pigeonpea has recently been used to restore soil fertility and to prevent soil erosion (4,62,78).

Pigeonpea seed contains 20 to 30% protein, is rich in essential amino acids, carbohydrates, minerals, and high amounts of vitamins A and C (17,63), and is the principal source of dietary protein for an estimated 1.1 billion people, most of whom are vegetarian and poor (Fig. 3). Because pigeonpea is a low-input rain-fed crop with characteristics that provide economic returns from each and every part of the plant, its cultivation has a direct bearing on the overall economic and financial well-being, and on the nutritional status of subsistence farmers in the subcontinent.

The Disease

Sterility mosaic disease (SMD), first described in 1931 from Pusa, Bihar State, India (39), and subsequently from the rest of India, and other pigeonpea-growing countries in Asia, is not known to occur in Africa or the Americas (56). SMD is the major constraint on pigeonpea production in the Indian subcontinent and occurs with regularity and, under suitable conditions, spreads rapidly, leading to epidemics. Yield losses depend on the growth stage at which infection occurs. The disease is sometimes referred to as the "Green Plague" because at flowering time, affected plants are green with excessive vegetative growth and have no flowers or seed pods; under congenial conditions, it spreads rapidly like a plague, leading to severe epidemics (69). SMD infection at an early stage (<45-day-old plants) results in a 95 to 100% loss in yield (25,59), while losses from late infection (>45-day-old plants) depend on the level of infection (i.e., number of affected branches per plant) and range from 26 to 97% (25). Seeds from partially affected plants are discolored and shriveled, with about 20% reduction in dry weight (Fig. 4C), and attract only a poor price. Maximum virus incidence and yield losses occur in ratooned and perennial pigeonpea (59). In addition, SMD infection predisposes plants to powdery mildew (*Oidiopsis taurica*) (54) infection and infestation by spider mites (*Schizotetranychus cajani*) (71), compounding the damage.

Precise data on the impact of SMD and its socio-economic importance are limited, but in assessing the economic importance of various biotic problems of pigeonpea, reports indicate that SMD causes greater yield losses than any other disease affecting pigeonpea in India (25). In India alone in 1984, losses due to SMD were estimated

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Publication no. D-2004-0308-01F

at 205,000 tons of grain valued at US$76 million (25), and in India and Nepal in 1993, losses were US$280 million (58). More recent studies on the economic impact of SMD are lacking, but the disease is endemic in the subcontinent and continues to be responsible for greater losses than ever before (10,56,80).

The disease is characterized by one or more of the following features: complete or partial cessation of flower production (sterility), mosaic or chlorotic ringspot symptoms on leaves (Figs. 4 and 5A, B), excessive vegetative growth, stunting, and reduction in leaf size (56,59). The precise symptoms vary depending on the pigeonpea genotype and are categorized into three types: (i) severe mosaic (Fig. 5A, B) and sterility (Fig. 4); (ii) mild mosaic (Fig. 5A) with partial sterility; and (iii) chlorotic ring spots (Fig. 5A) without any noticeable sterility. Symptoms also depend on the time of infection. Infection in susceptible genotypes at an early stage of crop growth

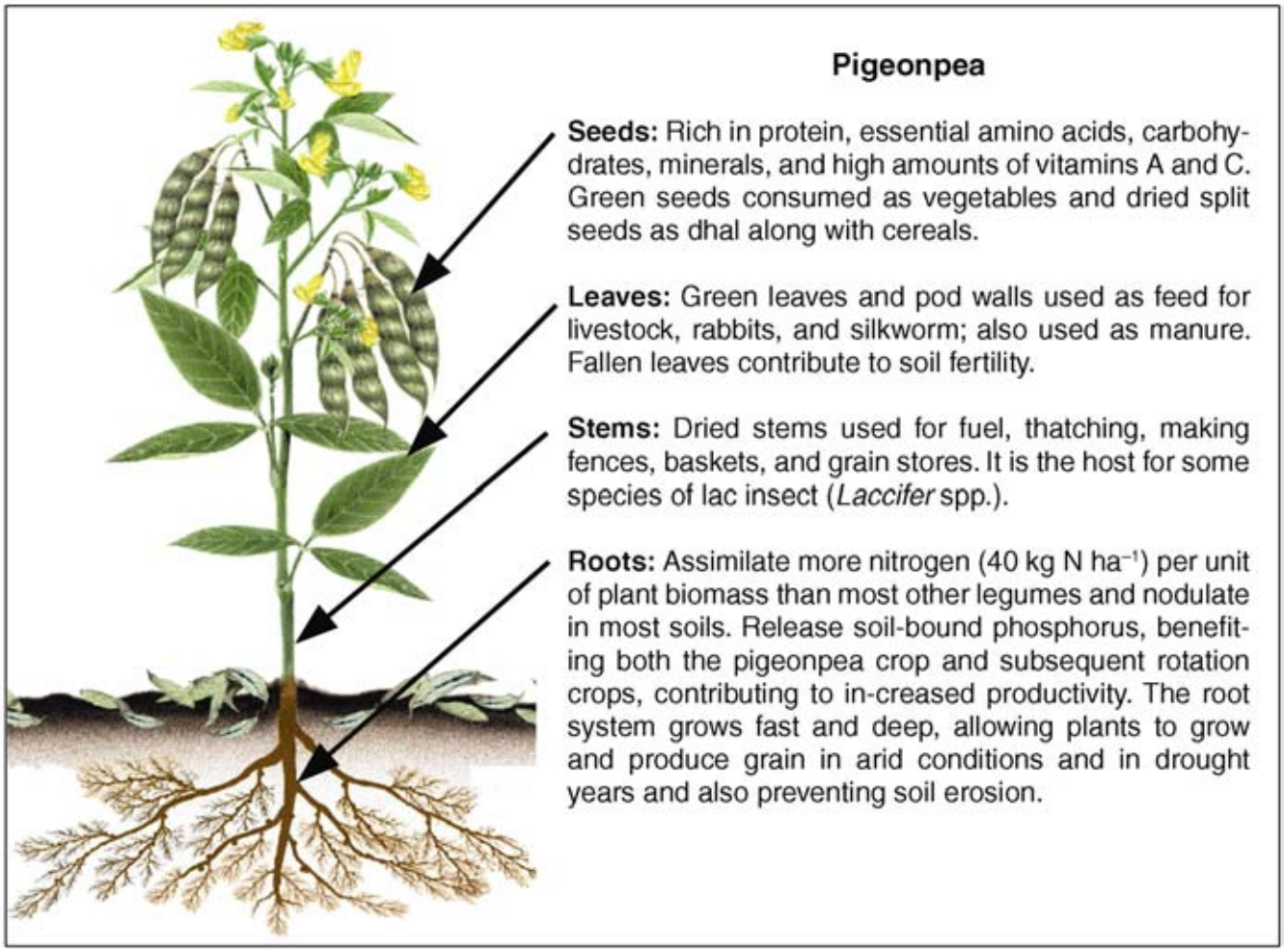

Fig. 1. Pigeonpea – a multipurpose grain legume.

Fig. 2. Worldwide distribution of major pigeonpea growing areas (red dots); sterility mosaic disease (SMD) endemic regions indicated within the blue rectangle.

(<30- to 45-day-old plants) results in the expression of characteristic disease symptoms in 10 to 15 days and almost complete cessation of flowering, but leaf symptoms become masked as plants grow. Later infection in susceptible cultivars (>50- to 60-day-old plants) results in slightly delayed symptom development and then only mild mosaic symptoms on only a few branches or parts of branches, and such plants show reduced flowering (20 to 50%). However, after ratooning (severe pruning), new growth from such plants shows severe mosaic symptoms and complete sterility (Fig. 5B).

The SMD causal agent was not known, but graft transmission experiments showed that it was an infectious agent (7) that, under natural conditions, is transmitted by the eriophyid mite, *Aceria cajani* Channabasavanna (Acari: Arthropoda) (Fig. 6) (27,53,65). Susceptible genotypes inoculated either with mites or by grafting show similar symptoms, but genotypes that show only chlorotic ring spots express symptoms when infected with mites but seldom when graft-inoculated (N. K. Kulkarni and P. L. Kumar, *unpublished data*).

The disease agent has remained elusive to identification and characterization over many decades, but studies demonstrated that it is not a fungus, bacterium, or phytoplasma-like agent (20,42,69). The invariable association of vector mites with diseased plants led to a speculation that the symptoms were the result of mite toxemia, but this was excluded by critical experiments using SMD-agent-free mite colonies on SMD-susceptible pigeonpea cultivars (20,27). In the absence of other likely causes, and on the basis of symptoms and transmission by mites, the SMD agent was therefore assumed to be a virus, but despite several attempts, especially during the past 20 years, the disease agent has remained uncharacterized and has posed a major challenge to the scientific community (35,42,69).

SMD diagnosis and the selection of germ plasm resistant to it have therefore been based solely on symptom expression. However, such diagnosis is complicated by the fact that symptom expression is governed by many biotic and abiotic factors and that pigeonpea is cross-pollinated so that genotypic variability, induced as a result of cross-pollination, also plays an important role in symptomatology. The unambiguous identification and utilization of resistant genotypes has therefore been hindered severely by the lack of sensitive diagnostic tools for the unequivocal identification of the disease agent.

Fig. 3. Pigeonpea supplies much-needed dietary protein to more than 1 billion people, most of whom are poor and vegetarian.

Fig. 4. Farmer holding a branch from a healthy pigeonpea plant (left) and branch from a sterility mosaic disease (SMD)-affected pigeonpea plant (right). Note the lack of flowers and increased vegetative growth on the infected branch. Because SMD inhibits flower production, there is 100% crop loss when infection occurs at an early stage of crop growth.

Advances Allowing Development of Disease Control Strategies

Isolation, purification, and properties of the causal virus. Previous work showed that the causal agent of SMD was transmitted by eriophyid mites (20,53,65). As these microscopic animals, particularly *A. cajani,* possess only a short stylet of ~2.03 µm, this allows penetration into only epidermal and, at most, underlying mesophyll cells (60). They can therefore only acquire the disease agent if it is present in such plant cells. Viruses occurring in such tissues are usually transmitted mechanically in sap extracts, and all the characterized mite-transmitted viruses are transmissible in this way, although for some, only with great difficulty (21,23,24,44). Against this background, we assumed that host polyphenolic compounds interfere with the stability and/or the infectivity of the SMD agent, thereby inhibiting virus infectivity and/or purification of virus particles. A purification procedure to minimize the influence of such components was therefore developed involving extraction of infected leaves in buffer containing chelating and reducing agents, high concentrations of nonionic detergent, and the precipitation of virus particles by polyethylene glycol. Further purification was achieved by quasi-equilibrium zonal centrifugation in sucrose and in CsCl gradients (30,34,36). Preparations made in this way contained aggregates of highly flexuous, apparently irregularly branched, filamentous virus-like particles (VLPs) of 8 to 11 nm diameter and of undetermined length, resembling in appearance particles of tenuiviruses (Fig. 7) (16). Comparable preparations from healthy pigeonpea leaves were free from such particles. The purified virus preparations contained a major protein of 32 kDa and up to seven segmented RNA species of size 6.8 to 1.1 kb. Such particles were isolated consistently from all SMD-affected plant samples collected from different locations of peninsular India and from SMD-affected pigeonpea samples infected by graft inocu-

lation, and by infective mites (*A. cajani*). Because of this very close association, the virus was named *Pigeonpea sterility mosaic virus* (PPSMV) (30,34,36) and was the first evidence of a causal agent for SMD.

Although the purified PPSMV VLP preparations were not infective to pigeonpea plants, PPSMV was transmitted experimentally, but with difficulty, by mechanical inoculation of fresh leaf sap extracts of SMD-affected pigeonpea to *Nicotiana benthamiana* and *N. clevelandii*. However, it was not possible to transmit the agent from infected *Nicotiana* species to pigeonpea by mechanical inoculation of sap. Systemically infected leaves of the *Nicotiana* species developed mild chlorosis and some necrotic spots (Fig. 8) (30,33,34).

Diagnostics for the causal virus. Polyclonal antibodies to PPSMV VLP preparations produced in a rabbit were very effective in detecting PPSMV in plant tissues by the double antibody sandwich (DAS) and direct antigen coating forms of ELISA using enzyme-labeled (alkaline phosphatase or penicillinase) immuno-gamaglobulin. These assays were simple, sensitive, and cost effective, and easily adaptable to conditions in developing countries (34). PPSMV was detected by ELISA in all SMD-affected pigeonpea plants infected experimentally by *A. cajani* and by grafting, and naturally in the field at several different locations in India, and in infected accessions of wild pigeonpea, *Cajanus scarabaeoides* (L.) Benth. Furthermore, it was detected in various pigeonpea genotypes showing the different symptom forms of the disease, and from *N. benthamiana* and *N. clevelandii* infected by mechanical inoculation. Leaves from hundreds of healthy or uninoculated pigeonpea plants were negative in ELISA (28,34). Taken together, these data demonstrated the complete and specific association of PPSMV with SMD, and provide very strong circumstantial evidence that PPSMV is the causal agent of the disease, ending the search for one of the most elusive plant pathogens. Unequivocal evidence that PPSMV is the causal agent depends on fulfilling Koch's postulates, but several technical difficulties prevent this, including the unstable nature of the virus and the difficulty of infecting pigeonpea by mechanical inoculation.

Novelty and relationships of the causal virus. The properties of PPSMV indicate that it is a previously undescribed virus with an unusual combination of properties. In the size and appearance of its VLPs and the number and sizes of its protein and RNA components, it is similar to viruses in the genus *Tenuivirus* (16). However, all tenuiviruses are phloem limited, transmitted by *Delphacid* planthoppers, and infect plant species in the Poaceae (16).

Ultrastructural studies of PPSMV-infected pigeonpea and *N. benthamiana* plants identified 100- to 150-nm quasi-spherical membrane-bound bodies (MBBs) and fibrous inclusions (FIs) (Fig. 9A, C) (30). The MBBs were labeled in situ specifically with antiserum to PPSMV, indicating that they contain the PPSMV-specific 32-kDa antigen (Fig. 9B). The FIs found in PPSMV-infected cells are possibly a nonstructural inclusion protein of PPSMV (30). Although tenuiviruses do not produce cellular inclusion bodies that resemble the MBBs found in PPSMV-infected cells, their nonstructural protein inclusions (NCP) in infected cells resemble the FIs of PPSMV (14,15,30).

PPSMV also resembles tospoviruses that share many properties with tenuiviruses. Thus, the filamentous VLPs of PPSMV resemble the nucleoprotein particles of *Tomato spotted wilt virus* (TSWV), and the MBBs of PPSMV are similar to, although larger than, those of TSWV (26). Despite these similarities, serological tests failed to detect any relationship of PPSMV to *Maize stripe virus* (MSpV) and *Peanut*

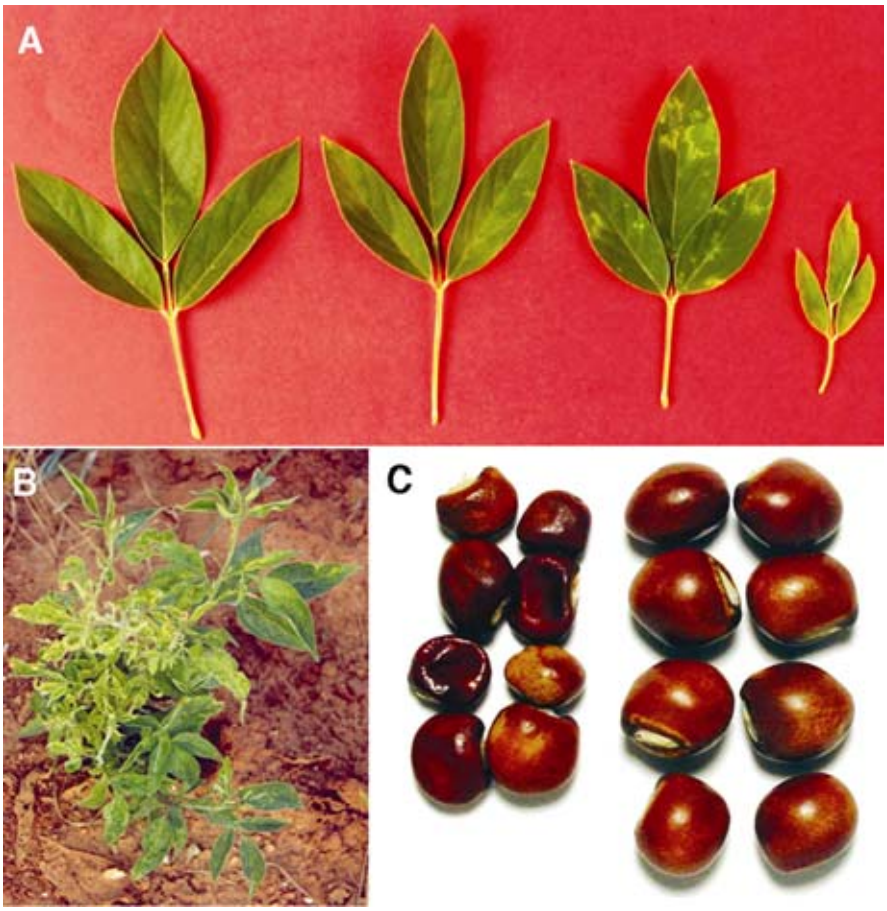

Fig. 5. A, Leaf symptom types of sterility mosaic disease (SMD) induced as a result of genotype–virus interactions. From left to right: a healthy pigeonpea plant, a genotype showing mild mosaic symptoms, a genotype showing chlorotic ring spots, and a genotype showing severe mosaic symptoms. B, New growth from ratooned pigeonpea showing severe SMD symptoms. C, Pigeonpea seed from healthy (right) and SMD-affected plants (left).

Fig. 6. Scanning electron micrograph of *Aceria cajani,* the vector of *Pigeonpea sterility mosaic virus* (PPSMV). It is an obligate pest dependent on pigeonpea during all stages of its life cycle, and it inhabits the underside of leaves. In nature, PPSMV and the vector coexist. Bar = 10 µm.

bud necrosis virus (PBNV), a tenuivirus and a tospovirus, respectively, that are endemic in the Indian subcontinent (18,48,52). Furthermore, whereas tospoviruses, tenuiviruses, and several other membrane-associated plant viruses are transmitted in a persistent and often propagative manner by their invertebrate vector species (77), PPSMV is transmitted by an eriophyid mite in a semi-persistent manner (27). Moreover, the nucleotide sequence of c. 2 kb of PPSMV-RNA and the monoisotopic masses of the 32-kDa nucleoprotein show no similarity with these viruses, or with any other organisms in databases (34; P. L. Kumar and A. T. Jones, *unpublished data*).

The VLPs of PPSMV show some morphological similarity to species in the genus *Ophiovirus*, but members of this genus differ from PPSMV in the number and sizes of their protein and RNA components (77), and there is no serological relationship detected between PPSMV and three members of this genus (30,34).

PPSMV shows most similarity with High Plains virus (HPV) (23), as each virus is transmitted by eriophyid mites, has 4 to 7 RNA species, a virus-specific 32-kDa protein, MBBs of similar size and morphology, and is mechanically transmitted with difficulty in sap extracts but not in purified preparations (2,23,30,37,38). However, no serological relationship was detected between these two viruses (34). MBBs similar to those detected in PPSMV- and HPV-infected plants are also detected in plants affected with other eriophyid mite-transmitted agents that cause fig mosaic, wheat spot mosaic, thistle mosaic, and rose rosette diseases (3,6,19). These agents, together with PPSMV and HPV, probably represent species in a new genus of plant viruses.

In leaf sap extracts and in purified preparations of PPSMV and HPV, no structures comparable to MBBs were detected (23,30), but for each virus, their MBBs were heavily labeled with their respective antiserum to purified VLPs. It is therefore possible that these particles are released from ruptured MBBs during the purification process. However, attempts to detect MBBs in purified PPSMV preparations concentrated without the use of detergents and organic solvents, or by fixing sap extracts in glutaraldehyde, were not successful. It is likely that the lack of infectivity of purified preparations could be due to the length of time that the VLPs are in vitro, for we have shown that PPSMV infectivity is short-lived in sap (15 min) (33,34). Alternatively, intact MBBs may be required for infectivity, and these are destroyed in the purification process. Presently, therefore, it is unclear if either the MBBs or the filamentous VLPs represent the infective particles of PPSMV.

Transmission of the causal virus by the eriophyid mite vector. PPSMV is transmitted by the eriophyid mite, *A. cajani* (Fig. 6) (20,65). This mite is highly host-specific and is largely confined to pigeonpea and its wild relatives, *C. scarabaeoides* and *C. cajanifolius*. Adult *A. cajani* measure 200 to 250 µm and have a very short life cycle of about 2 weeks comprising egg (30 × 40 µm) and two nymphal stages (45). Mites inhabit the lower surface of leaflets and are found predominantly on symptomatic leaves of PPSMV-infected plants (31,41,55). Their feeding causes no obvious damage to the host. Once established on PPSMV-susceptible genotypes, mites can multiply to high densities within a few weeks. Their dispersal is passive, assisted mainly by wind currents.

Little was known about the mechanism of transmission of PPSMV by *A. cajani* because studies were hindered by difficulties in rearing healthy mite colonies, manipulating individual *A. cajani,* and the lack of information on the causal agent of SMD and its unequivocal detection in plants. However, we have now overcome most of these difficulties and have shown that the virus is transmitted by the vector mite in a semi-persistent manner (27). Studies on two other mite-transmitted viruses also indicate a semi-persistent mode of transmission (21,40,72).

Our studies showed that single *A. cajani* transmit PPSMV, but the maximum transmission achieved with single mites was about 50%, which is high compared with the efficiency reported for the few other mite-transmitted viruses studied in detail (21,46). To transmit PPSMV, *A. cajani* required a minimum 15-min acquisition access period and 90-min inoculation access period, but these times were decreased to 10 and 60 min, respectively, when mites were starved prior to feeding. Viruliferous mites lose the ability to transmit PPSMV after feeding for 2 to 10 h on healthy plants, and there is no apparent latent period. As with other eriophyid mite–transmitted viruses, there is no evidence for transovarial transmission (44).

A. cajani retain PPSMV for up to 6 h when feeding and for more than 13 h without access to a susceptible host, explaining the ability of *A. cajani* to transmit PPSMV after being carried in wind currents to new plants. Although *A. cajani* remain alive without feeding for up to 30 h in a moist chamber, they do not survive when transferred to plants, so that in nature it is unlikely that mites survive for very many hours without feeding.

Efficient screening methods for virus resistance. Based on the above findings, a protocol for rapid screening of pigeonpea genotypes under laboratory conditions was developed. Pigeonpea plants raised in growth chambers were inoculated at the two-leaf stage with viruliferous mites by stapling SMD-affected pigeonpea leaves containing mites onto leaves of test plants (43). About 2 to 3 weeks later, plants were assessed for disease symptoms on leaves and tested for PPSMV in DAS-ELISA. Resistant genotypes (asymptomatic and ELISA negative) were inoculated again by petiole grafting (51) with mite-free SMD-

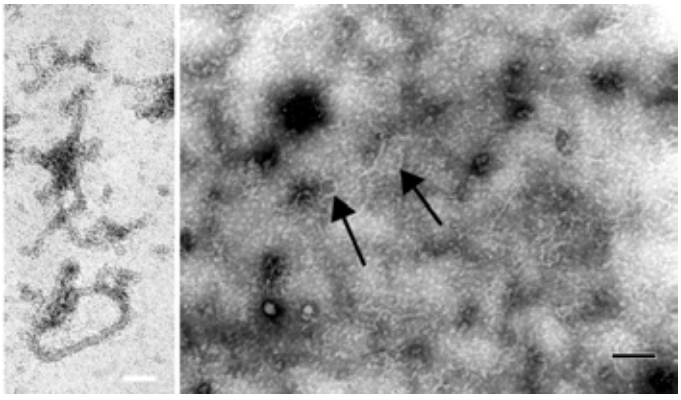

Fig. 7. Electron micrographs of the flexuous filamentous virus-like particles of *Pigeonpea sterility mosaic virus* (PPSMV) in a preparation purified from infected pigeonpea in uranyl acetate, pH 3.5, showing their linear (right, arrows) and branched and circular (left) structure. Bars represent 100 nm.

Fig. 8. Symptoms in *Nicotiana benthamiana* following mechanical inoculation with sap from a *Pigeonpea sterility mosaic virus*–infected pigeonpea plant.

infected pigeonpea material to identify their resistance to the virus. Screening in this way demonstrated that resistant genotypes were either (i) resistant to PPSMV only, (ii) resistant to the mites only, or (iii) resistant to both PPSMV and mites. It proved possible to determine the nature of the resistance to SMD in individual pigeonpea genotypes within 6 to 8 weeks.

Variation in host plant resistance. In screening trials initiated in 1975, ICRISAT and the Indian Council of Agricultural Research identified some pigeonpea cultivars with some field resistance to SMD infection. Although the resistant lines performed well in field trials at ICRISAT, Patancheru, and surrounding regions, their resistance elsewhere in the Indian subcontinent was much less effective (57). There are three possible reasons for this location-specific resistance in genotypes: variation within the mite vector, the virus, or both organisms. In order to produce durable resistance to SMD in pigeonpea genotypes, it was essential that the reason(s) for this location-specific resistance was determined. Analysis was therefore made of variation in the pigeonpea mites and PPSMV isolates present in the regions of the subcontinent where SMD resistance had been overcome.

Analysis for mite vector variation. Eriophyid mites (Arthropoda: Acari) are among the smallest arthropods known, and accurate identification of species, particularly by morphological characters, is very difficult because of their very small size (~200 µm) and their morphological similarity. Because of the microscopic size and soft body of these tiny animals and because single individuals are difficult to manipulate, we used a polymerase chain reaction (PCR)-based rDNA analysis technique that we showed previously to be rapid and sensitive in the unambiguous identification of morphologically closely related *Cecidophyopsis* mite species (18,32). Scanning electron microscopy was also used to study morphological features of *A. cajani* from India, Nepal, and Myanmar based on the first description of *A. cajani* by Channabasavanna (8).

These analyses of mite populations obtained from SMD-affected plants from several locations in India, Nepal, and Myanmar indicated that there was no significant variation in rDNA regions, or in the morphological features studied by scanning electron microscopy (31). Such results suggested that *A. cajani* on pigeonpea across the Indian subcontinent constituted one population and that no other *Aceria* species (and probably no *A. cajani* biotypes) are involved in PPSMV transmission. This indicated that the breakdown in SMD-resistance in pigeonpea genotypes across the Indian subcontinent is not influenced by variation in the mite vector, *A. cajani*.

Analysis for virus variation. To assess the possibility of virus variation as the cause of breakdown in resistance, a set of differential pigeonpea genotypes were planted at different field locations (57). Based on symptoms in these differential pigeonpea genotypes, the PPSMV isolates present on pigeonpea at Patancheru, Andhra Pradesh (P), Bangalore, Karnataka (B), and Coimbatore, Tamil Nadu (C), were found to be different (Table 1).

Analysis of the virus isolates at these locations showed that they were indistinguishable serologically in ELISA and at the genome level using reverse transcription–polymerase chain reaction (RT-PCR) with primers based on the sequence to part of RNA 5. However, in Western blot analysis to detect the PPSMV-specific 32-kDa protein in leaf sap, isolate C was clearly distinguished from the other two isolates in reacting only weakly with antiserum to isolate P. Furthermore, the protein specifically associated with the VLP preparations of isolate C had a size of c. 35 kDa, and not 32 kDa as with isolates P and B. This difference in the size of the VLP-associated protein may be due to the fact that they do differ in size or that all three isolates have a protein of 35 kDa but that those in preparations of isolates P and B are readily degraded to 32 kDa. Whatever the precise reason for the difference in size, isolate C is clearly different chemically as well as biologically from the other two isolates. The basis for the breakdown in PPSMV-resistant genotypes seems therefore to be due to regional variation in PPSMV isolates (P. L. Kumar and A. T. Jones, *unpublished data*).

The nucleotide sequence of the genome of the three PPSMV isolates is necessary to identify those sequences contributing to biodiversity. Once this is obtained, the sequence of the genome regions that differ will be used to develop sensitive nucleic acid–based methods, such as RT-PCR and nonradioactive nucleic acid probes, to detect and differentiate PPSMV isolates.

Durable SMD Resistance and Disease Management

Identification of broad-based SMD-resistant genotypes. Adequate levels of SMD resistance are scarce in the cultivated pigeonpea gene pool, but wild relatives of pigeonpea have been shown to possess high levels of resistance to several pigeonpea biotic constraints (61). Among these, *C. scarabaeoides,* which is widely distributed predominantly in Asia and Australia, is placed in the secondary gene pool and is cross-compatible with cultivated pigeonpea via traditional breeding or introgression through backcrossing methods (64). ICRISAT maintains accessions of this species, and 110 of them were tested against PPSMV isolates P, B, and C. Some were resistant to only isolate P, others to only isolate B, but a few were resistant to isolates B, P, and C, and no symptoms or

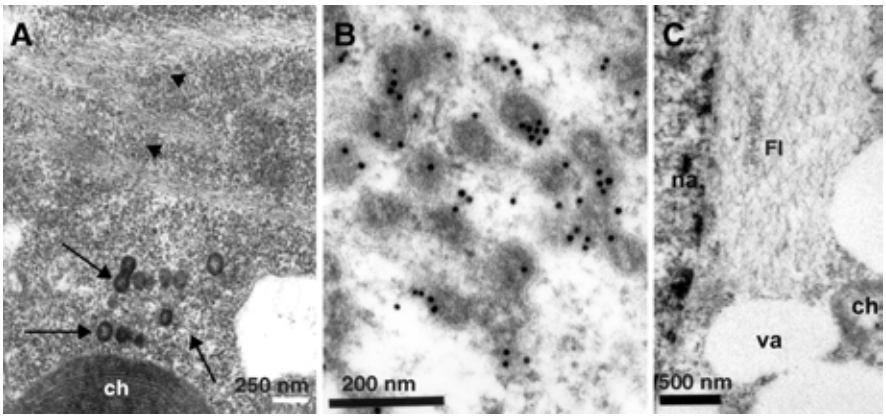

Fig. 9. A, Cytopathology of *Pigeonpea sterility mosaic virus* (PPSMV)-infected pigeonpea cells showing membrane-bound bodies (MBB; indicated by arrows) and fibrous inclusions (FI; indicated by arrowheads). B, Immuno-gold labeling of PPSMV polyclonal antibodies to MBBs in PPSMV-infected pigeonpea cells. C, FIs in SMD-affected cells. ch, chloroplast; na, nucleus; va, vacuole.

Table 1. Symptom response of some pigeonpea genotypes to infection with three isolates of *Pigeonpea sterility mosaic virus* (PPSMV)

Genotype	PPSMV-P	PPSMV-B	PPSMV-C
ICP 2376	**Ringspots**[a]	Severe mosaic	Severe mosaic
ICPL 7035	No symptoms	No symptoms	No symptoms
ICP 10976	Mild mosaic	Mild mosaic	**Severe mosaic**
ICP11164	**No symptoms**	**Mild mosaic**	**Severe mosaic**
PURPLE 1	Severe mosaic	Severe mosaic	**Mild mosaic**

[a] Bold font indicates differences in reaction from the other two isolates.

mites were noted on these plants. In addition, some resistant accessions also possessed resistance to pest damage, cyst nematode, and wilt (29). These sources of broad-based SMD resistance identified in *C. scarabaeoides* are being used for incorporating SMD resistance into existing cultivated pigeonpea.

Mechanisms and inheritance of resistance to SMD. Our studies have indicated that SMD resistance in some genotypes is due to immunity to PPSMV, in others to resistance to *A. cajani,* and in a few others to resistance to both organisms. With regard to mite resistance, it is known that some SMD-resistant genotypes have a thicker leaf cuticle and epidermal cell wall than those of mite-susceptible genotypes (60). Conceivably, the thick cuticle prevents the short mite stylets reaching epidermal cells, preventing feeding altogether.

A complicating factor in determining the precise nature of the resistance mechanism is our finding that the reproduction of *A. cajani* is much greater on PPSMV-infected plants than on healthy plants of the same genotype, confirming some earlier field observations (27,28,41,55). There seems therefore to be a beneficial relationship between the vector mite and the virus it transmits, and this may explain why mites are rarely found on PPSMV-resistant pigeonpea genotypes. Interestingly, compared with healthy plants, greatly increased numbers of the eriophyid mite, *Cecidophyopsis ribis,* occur on blackcurrant plants infected with the *Blackcurrant reversion virus* that it transmits (24,75), and of *Phyllocoptes fructiphylus* on multiflora rose infected with the agent of Rose rosette disease that it transmits (13).

Earlier studies indicated that susceptibility to SMD is dominant over tolerance and that resistance and disease response to SMD infection is under the control of independent nonallelic genes (66,70,73,74). However, our finding of several distinct resistance mechanisms to SMD infection, the occurrence of at least three PPSMV strains, the close relationship between mite numbers and PPSMV infection, and the wide array of pigeonpea lines, makes the interpretation of this earlier data more difficult.

SMD epidemiology. SMD occurs in every year in most of the pigeonpea growing regions in India, but the incidence in different regions and seasons varies widely (69). The epidemiology of SMD is complex, involving the virus, mite vector, pigeonpea cultivar, diverse agriculture systems, and the unpredictable environment of the semiarid tropics. Conflicting reports exist on the influence of climatic conditions on the epidemiology of SMD (9,57,68), but it is accepted that crops grown under irrigation or near irrigated fields are the most vulnerable to early SMD infection (47).

As the disease is not seedborne, it has to be introduced into the field by the mite vector. Information on the sources of primary inoculum is limited owing to the diversity of crop seasons in marginal farming systems. However, likely sources include diseased plants left in fields after harvest, on field banks, or in kitchen gardens, and the presence of perennial pigeonpea or wild relatives of pigeonpea such as *C. scarabaeoides*. The most common source of inoculum in rain-fed pigeonpea agriculture is the stubble left in the field after harvesting and plants near water sources such as canals and wells, or in the shade, as these plants maintain their foliage and harbor mites as well as virus. Following early rains, such plants start growing and provide an inoculum source for newly emerging crops nearby. Such early primary infection provides the maximum opportunity for repeating the cycle of infection. Disease spread within fields in a season depends on proximity to the source of inoculum, plant age, pigeonpea cultivar, climatic factors, and mite populations.

Factors governing the appearance of SMD in areas with only one pigeonpea crop followed by a long temporal gap, or where volunteer pigeonpeas are not common, are unknown. Nevertheless, it is likely that in such regions wild *Cajanus* species play a significant role in harboring the virus and its vector mite. Additionally, as pigeonpea cultivation in the subcontinent often overlaps in different geographical regions and as cultivars differ in maturity periods and include perennials, mites from such plants may possibly be carried long distances by wind currents to serve as inoculum sources. However, this explanation must be tempered by the knowledge that mites survive for only a few hours in the absence of feeding hosts and are highly sensitive to fluctuations in relative humidity and temperature.

SMD management. In the past, several methods have been studied to reduce SMD incidence. The application of pesticides to delay the onset of infection and disease spread was found to be critically dependent on the timing and dosage of applications (20,49,50), but even if these are optimized, the economics preclude it as an option for subsistence farmers.

Several studies to determine the effect of sowing dates, plant density, plant spacing, intercropping with millets, the use of border and inter-cropping with millets and fiber crops found no significant decrease in SMD incidence (5,22,49,67,79). Furthermore, in rain-fed systems, farmers are preoccupied with other revenue generating activities and lack resources and labor, so that crop management practices are not likely to be effective in reducing SMD incidence. For such subsistence farmers, the most feasible and cost-effective means of controlling SMD is the production and cultivation of pigeonpea cultivars resistant to the disease.

Future Developments

These very recent advances in our understanding of the etiology, detection, and transmission of PPSMV and of resistance to it in wild *Cajanus* species, some of which are also resistant to Fusarium wilt and pod borer, has been a major step towards an integrated approach to manage these serious disease problems in pigeonpea. The immediate outputs from our studies are two very promising pigeonpea genotypes with broad-based resistance to SMD that are currently being evaluated in farm trials in several states in India. The results to date indicate that their resistance to SMD is stable and that the genotypes are acceptable and suitable to farmers cultivating in different geographic locations and under different cropping systems, climatic conditions, and soil type (Fig. 4). Indeed, the demand from farmers for seed of this material currently greatly exceeds supply. Quality seed of these selections is being bulked up for more widespread release.

This work program, involving partnerships between international institutes and national centers to address strategic and applied research, coupled with technology development and transfer to farmers through strong links to extension agencies in NARS, NGOs, and farmer communities, has led to a significant potential increase in the profitability and competitiveness of pigeonpea farmers in the Indian subcontinent in a relatively short time. Consequently, such farmers can now see a possible end to the Green Plague that has so devastated this crop over many decades. For those rural communities in isolated arid areas in which this crop is pivotal to human survival, this prospect has not come too soon.

Acknowledgments

This work was funded by the United Kingdom Department for International Development (DFID) under the Crop Protection Programme for the benefit of developing countries. Work at the Scottish Crop Research Institute is grant aided by the Scottish Executive Environment and Rural Affairs Department (SEERAD) and was done under the conditions of a SEERAD license for studies on nonindigenous organisms. The International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) is a Future Harvest Center supported by the Consultative Group on International Agricultural Research (CGIAR). The views expressed are not necessarily those of DFID. We thank D. V. R. Reddy, Frances Kimmins, Jill Lenné, and Simon Edengreen (Natural Resource International, UK) for their encouragement and support.

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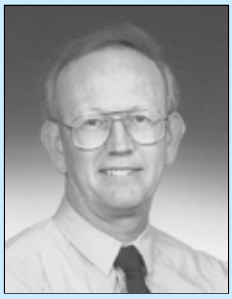

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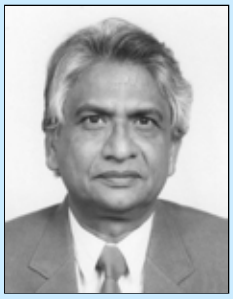

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