Plant Virus Disease Problems in the Developing World

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I. Introduction

The problem of plant virus diseases in the developing world is intimately related to the nature of the primary food crops grown in these areas and the agricultural practices used. The Food and Agriculture Organisation (FAO) has defined the major primary food crops (in order of volume grown) in the developing world to be: (1) rice, (2) wheat, (3) maize, (4) cassava, (5) fresh vegetables, and (6) sweet potatoes. Other crops of major importance are sugarcane, oil palm fruit and soybeans (see Table I). The most important crops in the developing world as far as local populations are concerned, however, are bulk foods such as rice, maize, cassava, bananas, and sweet potatoes; vegetables such as beans and pumpkins; and fruits such as mangoes and coconuts (see Tables I and II). The developing world (see FAO Web site for definitions; http://www.fao.org) has a population of 4.473 billion out of a total world population of 5.767 billion. Thus, as far as this review is concerned, the viruses of greatest importance are those affecting the crops that feed the greatest number of people rather than those destined for export.

Other important considerations in discussing the most important viruses affecting a developing country's agriculture are agricultural practices in this country, and the depth/coverage of reporting of plant virus diseases. Of necessity, most agriculture in developing countries is devoted to feeding people rather than developing commodities for export; the farming practices are also usually smaller in scale and of lower input cost than those in developed countries. Thus, pesticide/herbicide use is less extensive or less effective, and far less inorganic fertilizer is used than in extensive commercial farming. Additionally, people often use cultivars and varieties of crop plants that are not improved, resistant, higher yielding types: the effect of viruses in such plants is likely to be more severe than it is in more sophisticated varieties. All these considerations indicate that there is a greater chance of infection of crops by disease agents in developing countries, so that these countries probably have a greater risk of crop failure/reduction due to virus diseases than do developed regions.

A complicating factor in recognizing the disease problems facing plant production in developing countries is that the recognition of, and reporting of, viral diseases of crops in most of these nations is sadly lacking. Most of the personnel capable of pursuing such
activities do not live or work in these areas; nor are there particularly good facilities or resources to support the type of work necessary for even rudimentary virus disease surveillance. Whole countries like Zambia, for example, have no resident virologists, very limited access to facilities such as electron microscopes and almost no resources to purchase even rudimentary virus detection kits. Thus, the surveys of virus diseases that have been done are either extremely sketchy or have been done on a sporadic basis by outsiders. Consequently, there is in fact only a very limited understanding of the overall picture of the incidence of specific virus disease problems in most of the developing world. Beacons of light in this landscape are, however, the 16 international centers supported by the Consultative Group on International Agricultural Research (CGIAR; http://www.cgiar.org/). These centers maintain databases of information on agriculture within their specific mandates, several of which will be referred to below.

Inasmuch as information is available, then, this review will focus on virus diseases affecting only major crops. Nor will any attempt be made to be encyclopedic in coverage; the subject matter is sufficient for a book rather than an article. Consequently, we will concentrate on (1) major disease problems, especially those affecting the major food crops for subsistence agriculture, and (2) important emerging diseases in the developing world. We will also concentrate on recent information and/or information on less-well characterized viruses.

**II. Virus Diseases in Rice**

Rice (*Oryza sativa, Oryza* spp.) is the staple food of perhaps the largest single group of people on this planet; although it is apparently not the food crop grown in the greatest abundance worldwide, it is certainly the largest in the developing world (Table I). Although developing countries may not be major rice exporters, they are certainly among the chief growers and consumers; thus, any disease that affects rice directly affects the well-being of a significant proportion of the world’s population. Mainland China accounts for 197 million tonnes (Mt) out of a total of 545 Mt (Table II); other major producers are India (120 Mt) and Indonesia (51 Mt). The largest Latin American producer is Brazil, with 10 Mt; the largest African producer is Egypt, with 5 Mt. Thus, rice production is overwhelmingly concentrated in Asia.
Viruses described with rice as their principal host are rice black streaked dwarf (RBSDV), rice dwarf (RDV), rice gall dwarf (RGDV), and rice ragged stunt (RRSV) viruses (Reoviridae); rice grassy stunt (RGSV), rice hoja blanca (RHBV) and rice stripe (RSV) viruses (Tenuivirus); rice necrosis mosaic (RNMV, Potyvirus); rice stripe necrosis (RSNV, Furovirus); rice transitory yellowing (RTYV, Rhabdoviridae); rice tungro bacilliform (RTBV, Badnavirus); rice tungro spherical (RTSV, Sequiviridae, Waikavirus); and rice yellow mottle (Sobemovirus) (Murphy et al., 1995). Brunt et al. (1997; VIDE Web database, http://biology.anu.edu.au/Groups/MES/vide/) list 23 other viruses to which O. sativa is susceptible, such as barley yellow dwarf virus (BYDV, Luteovirus). These viruses infect rice under natural conditions; however, their effects are minor compared to those of the group named above. All of the viruses named above have insect vectors except for the furovirus; and as there is an excellent review on insect-borne viruses of rice (Hibino, 1990), insect-vector relations will not be discussed further. Additionally, Hibino (1996) has written a comprehensive review of the biology and epidemiology of rice viruses, so only selected examples of rice disease in developing countries will be discussed here. Further information is available at the International Rice Research Institute in Manila, the Philippines (http://www.cgiar.org/irri/).

A. Rice Tungro Disease

Perhaps the most important disease affecting rice is rice tungro disease (RTD). This is a leafhopper (Cicadellidae, Nephotettix spp.) transmitted virus complex consisting of rice tungro bacilliform virus (RTBV, a dsDNA badnavirus) and rice tungro spherical virus (RTSV, an ssRNA waikavirus) (Jones et al., 1991). Leafhopper transmission of the disease complex is dependent on the presence of RTSV; however, both agents contribute to symptoms and severity. The dependent transmission of RTBV can be explained by the association of both viruses with an inclusion body matrix in infected cells (Medina et al., 1994). The disease is confined to Asia and there are reports from India (Nagarajan, 1993) through Southeast Asia to China (Zhou et al., 1992). In Mindanao (the Philippines) RTD was listed as the most destructive disease on rice (Sanchez and Obien, 1995); also in the Philippines, Cabauatan et al. (1995) described a variety of different strains of both viruses with differing pathogenicity. In Thailand Parejarearn et al. (1990) investigated the susceptibility of weeds and wild rice to the RTD complex: 3 of 10 weed species got RTSV
alone; 9 wild rice species got both, one each got RTSV or RTBV alone, and one got neither; 27 wild rice lines got both. Druka et al. (1996) presented evidence of a distinction between Indian strains of RTSV and Southeast Asian isolates; Fan et al. (1996) showed evidence from DNA sequencing and restriction enzyme analysis for a similar division between RTBV isolates. This variation is probably related to vector biology, which needs to be understood better in order to effect better disease management (Chancellor and Cook, 1995). Hibino (1996) made the point that intensification of rice cultivation, and in particular, practices such as double-cropping, has significantly increased the incidence of virus disease. This is borne out by Rao and Hasanuddin (1991), who noted that RTD was endemic in double-cropped rice areas. A polymerase chain reaction (PCR) test for RTBV may have wide application in the timely detection of the disease complex (Dasgupta et al., 1996). Development of breeding material with genetic resistance to RTD is proceeding (Cabautan et al., 1995); however, there are problems with resistance breaking by both viruses and leafhoppers (Nemoto et al., 1985).

B. Rice Yellow Mottle

An important and relatively new disease in rice that has emerged in Africa is rice yellow mottle. The causative agent, the spherical ssRNA sobemovirus rice yellow mottle virus (RYMV), is found in all rice environments in West Africa and has the potential to seriously affect rice production, especially on small farms. Graminaceous weeds, including some wild rice species, are alternative hosts for the virus. Disease severity appears to depend on the type of rice planted, the vegetation zone, and the environment. The virus is transmitted by the beetle Chaetocnema zeae (Fomba, 1990; Awoderu, 1991a, b). The first appearance in the Ivory Coast was reported in 1987 (Awoderu et al., 1987), when its shift from infecting mainly lowland rice to include upland rice was noted. RYMV infections in Sierra Leone caused a yield reduction of up to 82% (Taylor et al., 1990). A serological study has mapped 73 isolates into three distinct serogroups, which are related to their probable ecological origin. The serogroups were also correlated with two RYMV pathotypes on a panel of rice varieties. The results are relevant to development of resistant lines, as area variation will have to be taken into account (Konate et al., 1997).
C. Rice Hoja Blanca

Another area-limited virus is rice hoja blanca virus (RHBV, *Tenuivirus*). This was one of the first tenuiviruses to be described (Shikata and Galvez, 1969). It is a thread-like virus with a divided ssRNA negative-sense genome, and is the major viral disease agent of rice in Latin America. It is closely related to the second most important rice disease agent, Echinochloa hoja blanca virus (EHBV) as well as being serologically and sequence related to rice and maize stripe tenuiviruses (Espinoza *et al.*, 1992; de Miranda *et al.*, 1994; 1996a, b, c, d, e. Tenuiviruses are transmitted by leafhoppers in a persistent, circulative manner.

D. Rice Grassy Stunt / Rice Stripe

Many strains of another tenuivirus, rice grassy stunt virus (RGSV), have been found in the Philippines (Miranda *et al.*, 1992); a new strain of the virus was also implicated in a severe disease of rice in Kerala, India, in 1988 (Ghosh and Venkataraman, 1994). In the latter case, rice that was resistant to an earlier-described RGSV (I) was susceptible to the new RGSV-II. This implies local sources of viral diversity and a potential threat to rice cultivation in these areas. Toriyama *et al.* (1997) have suggested that RGSV should be made a prototype of a new genus because it has more genome components and little sequence similarity to other tenuiviruses.

Rice stripe tenuivirus (RSV, RStV) has the potential to cause severe crop loss, as shown in Korea by Chen and Ko (1988), who noted up to 100% yield loss in field tests with early (10-30 days) infections and considerably lower losses with later (>100 days) infections. The virus occurs more often in temperate (developed) rice-growing areas. However, inasmuch as China is a developing country, it is worth noting that RNAs 3 and 4 of a Chinese isolate were different in size from those of two Japanese isolates; nevertheless, no differences consistent with geographic separation were seen (Qu *et al.*, 1997).

E. Rice Dwarf / Rice Ragged Stunt

Rice dwarf virus (RDV), a phytoereovirus (spherical particles, and a multicomponent dsRNA genome), has been well characterized over many years, mainly from Japan and other more sophisticated research centers. However, it has been noted to be a new agent of rice disease
in the Philippines, where it caused severe disease symptoms that were visible even when mixed with RTD (Cabauatan et al., 1993).

Another reovirus, rice ragged stunt (RRSV; Reoviridae: Oryzavirus), emerged in Sri Lanka in 1984, with a 20-70% disease incidence (Jayasena and Hibino, 1987).

F. Prevention of Disease in Rice

The overall impression conveyed by the literature on rice disease is that this can be curtailed significantly if planting dates are moved (Bae and Kim, 1994); if resistant cultivars are used; if correct cropping patterns are used; and if favorable soil conditions and nutrient status are achieved (Sanchez and Obien, 1995). The latter three factors will be the hardest for small farmers to achieve.

The good news about prevention of viral diseases of rice is that Agrobacterium tumefaciens and especially biolistic transformation of rice varieties are now semi-routine (Hiei et al., 1997; Chen et al., 1998). In the latter paper, for example, the authors biolistically transformed rice simultaneously with 14 transgenes: 17% of regenerated plants contained more than nine transgenes, and most of these plants were fertile. Thus, genetic engineering approaches to introducing viral resistance into rice will undoubtedly gain momentum. With RTD, for example, both genome components have been completely sequenced (Hay et al., 1991; Shen et al., 1993), providing material for plant transformation. IRRI currently has projects based on assessing transgenic resistance to RTD (http://www.cgiar.org/irri/DGReport99/Ce1.pdf). The entire genome of RYMV has been sequenced (Yassi et al., 1994); in addition, infectious clones exist (Brugidou et al., 1995), so that developments using molecular biotechnology are undoubtedly imminent. Fang et al. (1997) have already biolistically transformed two rice cultivars with the nucleocapsid gene of rice yellow stunt virus (RYSV, Rhabdoviridae), and have shown resistance in the regenerated plants to leafhopper-borne virus infection. The virus is given as being important in China and other Asian countries; thus, a new resource is now available for combating disease, at least for commercial farmers.
III. Virus Diseases in Wheat

Wheat (*Triticum aestivum* L.) is mainly a cash crop in the developing world. China is again the largest producer by far (111 Mt, Table II), followed by India (63 Mt); the next biggest producer is Turkey (18 Mt), followed by other better-developed producers, with a long gap to the largest African producer outside South Africa (Egypt, 5 Mt) and other smaller producers.

Viruses found in wheat under natural conditions include BYDV, wheat chlorotic streak (WCSV) and winter wheat Russian mosaic (WWRMV, *Rhabdoviridae*); wheat dwarf (WDV, *Geminiviridae*, *Mastrevirus*); European wheat striate mosaic (EWSMV) and winter wheat mosaic (WWMV, *Tenuivirus*); wheat soil-borne mosaic (WSBMV, *Furovirus*); wheat spindle streak mosaic (WSSMV) and wheat yellow mosaic (WYMV, *Potyviridae*, *Bymovirus*); wheat streak mosaic (WSMV, *Potyviridae*, *Bymovirus*); and wheat yellow leaf (WYLV, *Closterovirus*) (Brunt et al., 1997; Murphy et al., 1995). Another 44 viruses to which wheat is susceptible are listed by Brunt et al. (1997); however, most of these viruses have been characterized from temperate developed countries and are of little relevance to developing country agriculture. CIMMYT (the International Maize and Wheat Improvement Center; CIMMYT, 1992) maintains a Web site at http://www.cgiar.org/cimmyt/. There is almost no information on viruses in wheat outside of Europe and North America, especially from poorer countries.

A notable exception to this situation, however, is the aphid-transmitted spherical ssRNA luteovirus BYDV. This virus has been reported from all over the world and is associated with crop loss everywhere it is reported. CIMMYT in Mexico has published two books on barley yellow dwarf (BYD) (see Burnett, 1990). These books discuss the incidence of the virus worldwide, as well as breeding efforts, vector relations and so on. Bertschinger (1994) also described CIMMYT’s efforts to combat BYD worldwide.

A survey from China (Qingzhou et al., 1995) correlated long-distance *Schizaphus graminum* migrations with the incidence of BYDV in the Ningxia region. This sort of work should, as it has elsewhere, allow relatively accurate forecasting of disease incidence.
IV. Virus Diseases of Maize

Maize (*Zea mays* L.) is the third largest crop in terms of volume grown in the developing world (Tables 1 and 2); again, China heroically out-performs the rest of the developing world, producing over half the total crop (128 Mt vs. 254 Mt). The next largest producers are the relatively well-developed countries of Brazil (32 Mt) and Mexico (18 Mt); Nigeria is the sixth largest producer, with 6 Mt. It is obvious, therefore, that maize is probably one of the most important subsistence-level foodstuffs outside of Asia.

The Sixth Report of the International Committee on Virus Taxonomy (ICTV) (Murphy et al., 1995) lists maize chlorotic dwarf (*MCDV*, *Sequiviridae*, *Waikavirus*); maize chlorotic mottle (*MCMV*, *Machlomovirus*); maize dwarf mosaic (*MDMV*, *Potyvirus*); maize mosaic (*MMV*) and maize sterile stunt (*MSSV*, *Rhabdoviridae*); maize rayado fino (*MRFV*, *Marafivirus*); maize rough dwarf (*MRDV*, *Reoviridae*, *Fijivirus*); maize streak (*MSV*, *Geminiviridae*, *Mastrevirus*); maize stripe (*MSIV*, *Tenuivirus*) and maize whiteline stripe viruses (*MWLMV*, unassigned) as viruses mainly occurring in maize. The VIDE database (Brunt et al., 1997) lists another 57 viruses as commonly infecting maize, including cucumber mosaic virus (*CMV*, *Bromoviridae*, *Cucumovirus*) and BYDV, as well as maize yellow stripe (*MYSV*, *Tenuivirus*). McGee (1988) gives another listing, with some overlap, but with a considerable amount of information on virus distribution and severity. Useful surveys of maize diseases in developing countries have been reported: Thottappilly et al. (1993), Tsai and Falk (1993), Marchand et al. (1995), and Ammar et al. (1987). Maize as a crop falls under the mandate of the International Institute for Tropical Agriculture based in Ibadan, Nigeria (IITA, http://www.cgiar.org/iita), as well as of CIMMYT.

A. Maize Streak Virus

Perhaps the most destructive viral disease of maize is the leafhopper-transmitted (Cicadellidae, *Cicadulina* spp.) maize streak disease (MSD), caused by maize streak virus (MSV). The virus is limited to Africa and neighboring Indian Ocean islands (Madagascar, Mauritius, La Réunion), where it can cause up to 100% yield losses in early-infected maize.

The virus has been worked on for over 90 years. It was described by Claude Fuller in 1901 and characterized biologically by H.H. Storey and others from the 1920s through the 1960s
(see Rybicki, 1988). However, only in 1974 was the unique geminate particle visualized (Bock et al., 1974), and only in 1977 was it shown that it has a single-stranded DNA genome and not one of RNA (Harrison et al., 1977). More historical material may be found at the Maize Streak Virus Home Page (http://www.uct.ac.za/microbiology/mastrevirus.htm), together with a comprehensive bibliography.

Important developments in recent years have been the complete genome sequencing of at least fourteen MSVs found in severe infections in maize and of six MSVs infecting wheat and grasses, as well as the determination of many partial MSV sequences and the complete and partial sequencing of other related mastreviruses (see Briddon et al., 1994, Rybicki et al., 1997, and the NCBI sequence database, http://www.ncbi.nlm.nih.gov/Entrez/nucleotide.html). These sequences show that virus isolates causing severe disease in maize are very closely related to one another regardless of their geographical origin. The genome of the most different maize isolate (from La Réunion; Peterschmitt et al., 1996) differs by only 4% from that of any other isolate. All maize isolates from continental Africa are within 2% of one another, whereas MSV strains from grasses and other cereals differ by 10-25% (D. Martin, E.P. Rybicki, J.A. Willment, W.H. Schnippenkoetter, unpublished 1999; see also Rybicki et al., 1997).

Maize was introduced as a crop in Africa and its neighbors. Thus, MSV “emerged” into maize crops from natural reservoir sources. The narrow range of viral genome diversity in isolates causing severe disease could be due to these being the only ones in a wide range of viral genotypes in grass hosts that were capable of replicating sufficiently well in maize, a host upon which the leafhopper vector does not breed, in order to be picked up for further transmission. A number of groups have studied vector-virus-host relations and have accumulated important data for the institution of good disease management. Mesfin et al. (1995) studied the feeding activities of Cicadulina mbila on different hosts and gathered a great deal of useful data on vector preferences, which may be put to good use in understanding the epidemiology of MSD. A major study on the biology of Cicadulina species determined that these have significantly different transmission efficiencies ranging from 15 to 45%; other characteristics related to vector potential were also discussed (Asanzi et al., 1995). Asanzi et al. (1994) noted interactions between vector populations, disease incidence, maize types and rainfall in the humid forest and Guinea savanna zones of Nigeria,
with important implications for control of disease. A study of planting density (Alo, 1993) concluded that disease incidence increased as plant separation decreased, which could have a dramatic impact on disease management.

Another important factor in MSV infections is reservoirs of the virus. Mesfin et al. (1992) investigated MSV isolates from maize and other grass species in Nigeria and concluded that only a small subset of isolates from grasses were capable of causing severe maize disease. Konate and Traore (1992) surveyed MSV isolates from Poaceae in Burkina Faso, and found 35 of 41 symptomatic plants contained ELISA-identified MSV-like viruses, from 32 of which virus could be transmitted to maize. They concluded that early planting of maize would decrease disease incidence, as reservoir infection - and availability of virus to leafhoppers - increased rapidly 3-4 weeks after the rains started. A later serological study (Konate and Traore, 1994) found three serotypes of MSV among 1240 samples from 36 naturally-infected hosts. A subset of one of these serotypes was evidently the maize isolate, as it masked symptoms caused by the other types in mixed infections. It has been pointed out elsewhere that serology is of very limited use with MSV strains, as they are all so similar antigenically. It is far more useful, for the sake of differentiation, to combine the use of a panel of maize, cereals, and grasses as differentials, with nucleic acid-based tests such as restriction fragment length polymorphisms (RFLPs), restriction mapping, or limited sequencing (Hughes et al., 1992; Briddon et al., 1994; http://www.uct.ac.za/microbiology/msvvariation.htm).

Breeding for resistance to MSV has been actively pursued in a number of locations for over 30 years. Presently there are programs in South Africa, Zimbabwe, Nigeria, Kenya, La Réunion, and elsewhere that are meeting with some success in significantly reducing the incidence of the disease in commercial plantings. Rodier et al. (1995) described some of the French efforts in this regard; CIMMYT (Zimbabwe) has also produced extremely resistant breeding lines, as have the Grain Crops Institute in South Africa (see http://www.uct.ac.za/microbiology/msv_session_2.htm). Kairo et al. (1995) discussed evidence of useful resistance mechanisms against the vector in maize tested for resistance to MSV that would be a very useful adjunct to virus resistance per se. Use of resistant varieties, together with sound crop management practice, may be the only long-term means of successfully combating MSV infections. The traditional recourse of the commercial farmer has always been spraying to control or eliminate the vector; however, this option is not open to subsistence farmers, who
have to use less costly practices. The possibility of using genetic engineering to produce MSV-resistant maize is also being actively pursued in at least two locations.

B. Maize Rayado Fino

Maize rayado fino disease (MRFD) is caused by the maize rayado fino virus (MRFV, *Marafivirus*). This is an isometric ssRNA-containing virus, limited to North and South America. It is persistently transmitted by the corn leafhopper *Dalbulus maidis* (among others) and may replicate in the vector (Tomaru *et al.*, 1995). It has the potential to cause severe crop losses (up to 100%). Three strains are recognized: the original MRFV, and Colombian and Brazilian strains (McGee, 1988). A 1992 survey of MRFD in Latin America found the virus in all eight countries surveyed, and in all life zones. Use of an MRFV-specific cDNA probe detected virus in 75% of symptomatic plants tested; symptom expression and symptomatology also varied widely, possibly indicating wide genetic diversity of the viruses involved (Kogel *et al.*, 1996). In Brazil MRFV was found to be the main maize virus, and, together with sugarcane mosaic virus (SCMV, *Potyvirus*), caused 29% yield loss. Experiments with different hybrids showed a wide range of susceptibilities, from 0 to 100% (Waquil *et al.*, 1996).

Hammond *et al.* (1997) studied the molecular epidemiology of MRFV by determining the coat protein (CP) gene and 3' non-coding region sequences of cDNA clones of 14 isolates from the Americas. They determined that the isolate sequences were related to within 88 to 99%, and that there were three phylogenetic clusters: the northern, southern, and Colombian isolates (formerly named "maize rayado colombiana virus"). The latter were deemed sufficiently different from the other two groups to be termed a distinct strain of MRFV.

The virus is also involved in a disease syndrome called "maize stunt disease", or “achaparramiento”, which is caused by corn stunt Spiroplasma, maize bushy stunt phytoplasma, and MRFVs, and is transmitted by *D. maidis*. The disease incidence in Nicaragua rose abruptly to very high levels over a few years and then decreased, correlating with a government-sponsored increase in irrigated maize production that “bridged” the vector populations between traditional growing seasons (Hruska *et al.*, 1996). The disease syndrome has apparently caused severe yield losses in maize throughout Central America.
Work in Nicaragua on the timing and extent of vector infestation of maize has shown that disease management should concentrate on reducing vector infestations from the seedling through the midwhorl growth stages, as this lessens disease severity and yield loss (Hruska and Gomez-Peralta, 1997).

C. Maize Dwarf Mosaic / Sugarcane Mosaic

Among the viruses infecting maize, MDMV and SCMV are probably the most widespread. These filamentous, aphid-transmitted, ssRNA potyviruses essentially have a worldwide distribution and are often associated with severe disease (McGee, 1988). The often confusing nomenclature of maize-infecting potyviruses has been clarified, with MDMV-B being designated as an SCMV (strain MDB) and MDMV-A being called MDMV (Shukla et al., 1992).

Apparently MDMV and SCMV-MDB cause significant cultivar-specific disease problems in maize in Shaanxi, China (Wang et al., 1987). Chen et al. (1995) reported that in 1993 in the Shaanxi region, MDMV was responsible for up to 30% of all maize disease (the majority being maize rough dwarf virus, MRDV), and the source was mainly perennial grasses. The first report of MDMV in maize in Pakistan, published in 1990, involved a naturally-infected commercial hybrid (Aftab et al., 1990). MDMV has been identified in maize and sorghum in Uttar Pradesh, causing significant disease (Rao et al., 1996). Venezuela seems to have an ongoing problem with MDMV-A. Garrido and Trujillo (1988) identified a new strain of MDMV in a serious viral disease of sorghum and maize; the virus also infected sugarcane. Pineda et al. (1991) noted severe yield losses in Portuguesa State due to either SCMV or MDMV. In 1995, Rangel et al. (1995a) identified two isolates of MDMV-A from Venezuela (one in johnsongrass), and in the same year they reported evaluations of a range of sorghum and maize cultivars for MDMV-A resistance (Rangel et al., 1995b). SCMV is an important pathogen of maize in the former Yugoslavia (Krstic and Tosic, 1995), and often occurs in mixed infections. Chen et al. (1996) discussed problems with SCMV and MDMV in Taiwan, where several strains of SCMV were found in sugarcane and where SCMV-MDB was found to predominate in maize. Waquil et al. (1996) found that SCMV infection reduced maize yields by 48% in certain areas of Brazil. Ammar et al. (1987) reported
SCMV in sugarcane and MDMV on maize, together with a mixture of other viruses, causing a high incidence of disease.

As a disturbing endnote, von Wechmar et al. (1992) noted the apparent transmission of both MDMV and SCMV-MDB by uredospores of the maize leaf rust *Puccinia sorghi*. This was the first “non-traditional” transmission mechanism noted for a potyvirus, and one that may explain a number of hitherto perhaps puzzling features of natural infections.

D. Other Maize Virus Diseases

In 1993, maize rough dwarf fijivirus (MRDV) comprised 70 to 80% of all maize disease in Shaanxi, China. In South America, apparently “Mal de Rio Cuarto”, or Rio Cuarto disease, is the most important viral disease of maize in Argentina and is caused by MRDV. Predictive models based on vector studies have apparently been validated over several years, and accurate forecasts of disease can now be made (March et al., 1995).

In Mauritius, maize stripe tenuivirus (MStV) can occur in mixed infections with MSV in maize, in which case MSV symptoms can mask those of MStV. Yield losses are worse than with either virus alone (de Doyle and Autrey, 1992). MStV infections occurred in Taichung in Taiwan in 1986 and 1987, causing severe disease symptoms in maize (Chen, 1990; Yang, 1990). The three cereal-infecting tenuiviruses in Taiwan are RSV, MStV and rice wilted stunt (RWSV). They may be successfully and simply differentiated by inoculation onto rice, maize, millet, and wheat (Chen et al., 1996). Aboul-Ata and Ammar (1989) noted that early sowing was recommended in the Giza area of Egypt to avoid infections with maize yellow stripe tenuivirus (MYStV) and MDMV, as vector numbers would be lower. Marchand et al. (1995) reviewed the incidence of MSV, MStV and the planthopper-transmitted maize mosaic (MMV, *Nucleorhabdovirus*) in Africa and Indian Ocean islands, with coverage of vectors, epidemiology, and resistance breeding.

From personal observations in South Africa and Kenya, it is obvious to the two of us, at least, that the incidence of virus disease in maize is severely under-reported. For instance, in one field in western Kenya near Kisumu in June 1997, it was possible to see evidence of three different virus diseases: symptoms of MSV, MDMV, and probably MMV infections were
obvious, and their incidence was relatively high (P.G. Markham and E.P. Rybicki, personal observation, 1997). Similarly, in one field in Mpumalanga in north-eastern South Africa in September 1997, it was possible to see MSV, MDMV, and another unidentified infection (G. Pietersen, personal observation, 1997). In neither location had anyone reported anything other than MSV. It is apparent that even a little training in symptom recognition would probably hugely increase disease reporting, even in some of the apparently better-developed countries of the developing world.

V. Virus Diseases of Sweet Potato

A. Common Viruses

Sweet potatoes (*Ipomoea batatas*) are grown the world over, but China grows more than 90% of the total (Table II). The International Potato Center (CIP, http://www.cgiar.org/cip/) in Peru has sweet potatoes as one of their four main crop interests. CIP Director General Hubert Zandstra has said: "Sweetpotato will play a major role in feeding the world population in the years ahead… with its high yields and ability to grow in poor soils under drought conditions, [it] is an excellent crop for the job" (http://www.cgiar.org/cip/new/new.htm). It is significant that although the crop originated in Latin America, its production outside the Americas (excluding the United States) is now far higher than it is within (Table II).

Virus diseases in sweet potato that are taxonomically accepted are the aphid-transmitted potyviruses sweet potato feathery mottle (SPFMV) (synonyms: SP virus A, SP chlorotic leafspot, SP russet crack, SP internal cork), sweet potato latent (SwPLV), and sweet potato vein mosaic (SPVMV), and the whitefly-transmitted “Ipomovirus” (*Potyviridae*) species sweet potato mild mottle (SPMMV) and SP yellow dwarf (SPYDV) (Barnett *et al.*, 1995). In addition, the VIDE Web database (http://biology.anu.edu.au/Groups/MES/vide/famly045.htm#Ipomoea batatas) lists cassava green mottle nepovirus, SP caulimovirus, SP leaf curl badnavirus, SP ringspot nepovirus, and the more recently-described sweet potato sunken vein closterovirus (SPSVV), which is whitefly (*Bemisia tabaci*)-transmitted.

Of these viruses, SPFMV is apparently the single most important disease agent, and the IPC has made it a focus of attention because of its impact on resource-poor farmers in sub-Saharan Africa. The Center cites sweet potato virus disease (SPVD) as the main problem in
the region, and says it is caused by “...the synergism between the aphid-borne sweetpotato feathery mottle virus (SPFMV) and a whitefly-borne virus (WBV)....CIP researchers developed cultivars resistant to SPFMV, but the plants were found to be susceptible to [SPVD].” (http://www.cgiar.org/cip/ipm/diseases/plant2.htm).

An Israeli study has shown that SPFMV-infected sweet potato cuttings showed no yield loss relative to controls; plots planted with SPSSV showed up to 30% yield loss only in the second year; however, doubly-infected plots have had yield reductions of 50% or more (Milgram et al., 1996).

A major review has highlighted the significance of SPFMV in subsistence production of the crop in Africa. Karyeija et al. (1998) discussed the impact and management of SPFMV, and in particular of SPVD, in sub-Saharan Africa. They describe the disease complex as SPFMV plus sweet potato chlorotic stunt virus (SPCSV, a putative Closterovirus); it is quite possible that the latter is in fact SPSVV (Karyeija et al., 1998; Brunt et al., 1997, and http://biology.anu.edu.au/Groups/MES/vide/descr787.htm). Infections of SPFMV alone are apparently low titer, and aphids have difficulty obtaining it; moreover, natural selection means that most cultivars are resistant to SPFMV infections. In double infections, however, the titer of SPFMV is markedly increased, and it is much more easily acquired. The authors speculated that SPFMV and SPCSV/SPSVV originated in Africa, as the whitefly vector (B. tabaci) is supposed to have done. They made the point that the disease is under-studied, and that there is a strong need for increased research on epidemiology in both crops and wild hosts. It is illuminating that the CIP database contains six references to SPFMV in Africa from 1985 to 1996 compared to 59 references from the Americas, despite their far smaller share of the world crop (see Table II) (Karyeija et al., 1998).

A Kenyan isolate of SPSSV was used for cDNA cloning and sequencing, as well as for production of antisera to CP produced in Escherichia coli (Hoyer et al., 1996). The CP was closely related to that of SPSVV-Israel and to those of SPVD-associated closteroviruses from Nigeria (SPCSV?) and the United States. This indicates that there is a widespread family of viruses capable of associating with SPFMV in SPVD and that the African experience should perhaps be taken seriously in the prevention of similar disease elsewhere.
Pozzer et al. (1995a) describe a Brazilian isolate of SPFMV found in a germplasm collection. Perhaps not coincidentally, Pozzer et al. (1995b) described yield differences between heat-treatment and meristem-tip culture derived virus-free material compared to naturally-diseased material infected with SPFMV. An increase in yield of over 100% was noted for virus-free plants compared to infected plants; however, after one crop cycle, the incidence of virus in both sets of plants was similar.

The IPC has claimed that the use of healthy planting materials is a highly effective control measure for SPVD: in addition to the evidence presented above, Chinese field experiments showed that virus-free planting materials yielded two to three times more than those infected with the virus (http://www.cgiar.org/cip/ipm/diseases/plant2.htm). To this end, researchers in Brazil have developed a more efficient protocol for production of virus-free sweet potatoes, through direct regeneration of shoot tips (Torres et al., 1996).

B. Other Viruses

Sweet potato latent virus (SwPLV) was unambiguously assigned to the genus Potyvirus after partial sequencing of the 3’ termini of a Taiwan isolate and an SwPLV-like Chinese isolate (Colinet et al., 1997). Fuentes et al. (1996) characterized a novel Luteovirus, sweet potato leaf speckling virus (SPLSV), from an accession in the IPC germplasm collection. It was also found on farms in northern Peru, indicating that it was in the environment. It was transmitted persistently by the aphid Macrosiphum euphorbiae.

The perils of importing sweet potato material, even from reputable sources, was demonstrated by Marinho and Dusi (1995), who reported detection of SPFMV, SwPLV, SPMMV, and SP chlorotic fleck virus (SPCFV) in two varieties imported from Japan; none of the viruses except SPFMV have been reported from Brazil.

VI. Virus Diseases of Cassava

“Manihot esculenta” Crantz, or cassava, provides food and a livelihood for about 500 million people in the developing world. The crop is relatively tolerant of poor soils and seasonal drought...Because it is well suited to harsh conditions, cassava is grown mostly in marginal
environments by poor farmers. Used mainly for food in sub-Saharan Africa…Cassava is one of the few crops that offer the possibility of linking small-scale farmers in marginal areas to expanding markets.” (http://www.ciat.cgiar.org/frames/fra_proy.htm). Certainly cassava production is far more widespread out than - for example - sweet potato, with three African nations featuring among the six largest producers (Table II).

Virus diseases affecting cassava, according to the ICTV, are African cassava mosaic (synonym: "cassava latent") (ACMV) and Indian cassava mosaic (ICMV, Geminiviridae, Begomovirus); cassava American latent (CALV) and cassava green mottle (CGMV, Comoviridae, Nepovirus); cassava brown streak-associated (CBSAV, Carlavirus); cassava common mosaic (CsCMV) and cassava virus X (CsVX, Potexvirus); cassava symptomless (CasSV, Rhabdoviridae); cassava vein mosaic (CsVMV, Caulimovirus) (Murphy et al., 1995). The VIDE database (http://biology.anu.edu.au/Groups/MES/vide/famly058.htm#Manihot esculenta) additionally lists cassava brown streak potyvirus (CBSV), cassava Caribbean mosaic and cassava Colombian symptomless potexviruses, and cassava Ivorian bacilliform ourmiavirus. To this may be added East African cassava mosaic (EACMV, Begomovirus).

The International Center for Tropical Agriculture (CIAT, http://www.cgiar.org/ciat) is the “home” of cassava; however, the IITA also looks after the crop in Africa. CIAT has an excellent database of publications on cassava diseases, with over 100 listings on cassava viruses alone (http://www.ciat.cgiar.org/frames/fra_inf.htm).

A. African / Indian Cassava Mosaic

The begomovirus African cassava mosaic virus (ACMV) was the first geminivirus sequenced. Stanley and Gay (1983) determined that the presumed agent of cassava mosaic disease (CMD) from western Kenya (then termed "cassava latent virus", CLV) was a two-component ssDNA virus. Morris et al. (1990) followed with the sequence of a very similar Nigerian isolate that had >95% sequence identity over both components with the Kenyan isolate. CMD in India was presumed to be simply a regional variant of the African disease until the genome of an Indian isolate was completely sequenced. It was found to be a distinct two-component begomovirus, no more closely related to ACMV than to other begomoviruses.
The same authors discovered another new begomovirus of cassava in Malawi. This was named East African cassava mosaic (EACMV), as it too showed no more affinity with Nigerian or Kenyan isolates than with (for example) tomato yellow leaf curl begomovirus (TYLCV). Subsequently another EACMV was sequenced from Tanzania (Zhou et al., 1997), and was found to be related to, but distinct from, the Malawi strain. Berrie et al. (1997, 1998) have described yet another distinct begomovirus infecting cassava in Africa: South African cassava mosaic virus (SACMV), a 2-component virus with closer affinities to TYLCV than to ACMV or EACMV (Berrie et al., 1998). The virus is associated with severe disease in cassava in northeast South Africa, but its distribution is unknown.

The whitefly-transmitted (B. tabaci) African cassava mosaic disease (ACMD) is described as “…an under-estimated and unsolved problem” by Thresh et al. (1994), who reviewed the history of research on the virus (stretching back to one O. Warburg, in 1894), as well as epidemiology, control, and yield reductions. They point out that mosaic in cassava probably costs Africa 28-49 Mt per year; they also described the possibilities of disease control and acknowledged that adoption of control measures on a sufficiently wide scale will be very difficult.

Gibson et al. (1996) pointed out that the current epidemic of ACMD in Uganda was associated with unusually severe symptoms. This was further investigated by Otim-Nape et al. (1997), who described how the disease was spreading across Uganda into western Kenya at the rate of 20 km/year along a broad front. Initial distribution of ACMD-free material resulted in its being rapidly infected; subsequent control attempts involved destruction of all diseased material before planting of clean material. This was apparently fairly satisfactory as a control measure. Zhou et al. (1997) showed evidence that the virus causing the pandemic is a natural recombinant between EACMV and ACMV, which are different species of Begomovirus. The Ugandan variant is essentially EACMV with two parts of the coat protein gene replaced by ACMV sequences. The same recombinant was detected in widely separated sampling sites within the severely diseased area (most of Uganda), indicating a single origin for the pandemic. This recombinant has since reached into western Kenya (E.P. Rybicki and P.G. Markham, personal observation, 1997), and is causing severe crop damage (see Fig. 1). It is quite remarkable to pass within a few kilometers from areas with mild ACMD to areas where there are almost no cassava plants left growing. The inevitable lag in
replacement of the crop by sweet potato, for example, results in severe hardship for farming families accustomed to using it as a staple in their diet. The wave of ACMD across Uganda may be a good example of the devastating effect of a plant virus on the human population. Otim-Nape *et al.* (1994) have described how the epidemic caused food shortages and even famine in some areas of Uganda.

A valuable research and extension resource was the generation of a panel of monoclonal antibodies (MAbs) to ACMV; this has been used in the differentiation of cassava isolates and the sensitive detection of the virus (Thomas *et al.*, 1986). Other research on ACMD in Africa includes a temporal disease progress study in the Ivory Coast, which indicated that the maximum rate of disease increase peaked 2 months after planting and had a seasonal component; disease increased markedly between November and June and less so between July and October. A reduction in the rate of spread with plant age was noted (Fargette *et al.*, 1994).

ICMV infections in India were apparently a problem in the past. Menon and Raychaudhuri (1970) cited it as a serious problem in Kerala, India. Interestingly, the virus infected cucumber as an alternate host. It has not been much discussed in recent years; however, in Tamil Nadu in 1986, the virus incidence was estimated at 5% (Jeyarajan *et al.*, 1988). Malathi *et al.* (1989) noted that the use of ELISA for virus detection showed up virus in symptom-free plants, demonstrating the need for good surveillance to avoid dissemination of diseased material. Mathew and Muniyappa (1991) transmitted ICMV using *B. tabaci* to cassava, ceara rubber, *Nicandra physalodes*, 18 different *Nicotiana* species, and 23 different cultivars of *N. tabacum*, illustrating the potential of the virus to find reservoirs in cultivated areas.

**B. Other Viruses In Cassava**

Cassava brown streak disease (CBSV, *Potyvirus*) has been known in eastern and central Africa for many years (see Storey, 1936). It apparently no longer causes particularly severe disease, and is not widespread. It occurs largely in lowland areas of Kenya, Malawi, Mozambique, Zimbabwe, Tanzania, and Uganda and causes necrosis of tubers (although apparently not in highland sites). Although it was tentatively classified as a potyvirus, the
vector may be a whitefly (*Bemisia afer*), and particles are apparently carlavirus-like (Bock, 1981; 1994). Two distinct strains were distinguished in Bock's 1994 study.

Costa and Kitajima (1972) described the occurrence of cassava common mosaic virus (CsCMV, *Potexvirus*) and cassava vein mosaic virus (CVMV, *Caulimoviridae*) in Brazil; the former caused some symptoms, the latter very few. Neither was seen to be vector-transmitted and both have since been sequenced (Calvert *et al.*, 1995, 1996). CsCMV is a typical potexvirus, though distinct from other sequenced exemplars; CVMV, which is widespread through the north-east part of Brazil, is a “pararetrovirus” (reverse-transcribing dsDNA genome) distinct from other well-characterized *Caulimoviridae* such as generic caulimoviruses and badnaviruses, and probably deserves its own genus. CVMV did not significantly affect plant yield in a number of trials in Brazil (dos Santos *et al.*, 1995).

CsCMV is listed as perhaps the worst virus problem in South America (Fauquet *et al.*, 1992). A survey in Colombia, however (Nolt *et al.*, 1992), found Caribbean mosaic disease in northern Colombia, CsVX in up to 50% of plants surveyed at a single location, and in all parts of south-central Colombia, and frogskin disease (FSD) in two of three growing areas. No CsCMV was found. A number of unidentified dsRNAs were identified in plants in addition to the characterized viruses.

FSD is endemic in areas of Colombia, where it causes some economic damage. It is apparently associated with CsVX and a “mosaic” agent; CsVX infection is symptomless, and the mosaic-causing agent has not been identified. CsVX is apparently transmissible by whiteflies, which is unusual for a potexvirus (Angel *et al.*, 1987).

A novel cassava virus was described from the Ivory Coast. Cassava Ivorian bacilliform virus has distinctive bacilliform particles and three ssRNAs and a number of strains have been found (Fargette *et al.*, 1991). The virus was presumed to be an *Alfamovirus* (*Bromoviridae*) by the authors; however, it is listed as an *Ourmiavirus* in the VIDE database. It causes symptomless infections in cassava and has not been described elsewhere.

That there are probably still some viruses to be characterized in cassava was shown by Cuervo (1990), who extracted a variety of dsRNA species from diseased cassava samples at
CIAT, Palmira. The dsRNA sizes ranged from 1.6 to 9.4 kb for plants infected with Cali mosaic, Caribbean mosaic, FSD, Quilace mosaic, and “latent” (CsVX?) agents.

C. Control of Cassava Disease

Nolt (1990) pointed out that because cassava originates in the Americas, its genetic diversity there is rich, and this means that it is a source of germplasm for breeding elsewhere. Unfortunately, it also means that pathogens of cassava are more diverse in the Americas, which necessitates strict quarantine measures when importing cassava into other areas. Economically important virus diseases of cassava, including cassava common mosaic virus (CCMV), CsVX, FSD and Caribbean mosaic disease, have been described in Latin America. These viral agents pose a potential threat if they are inadvertently introduced into new regions. However, through the use of thermotherapy and meristem-tip culture for virus eradication, and virus diagnostic methods such as ELISA and dsRNA detection, it should be possible to guard against this while disseminating valuable germplasm. Breeding efforts in Africa (IITA) and South America (CIAT) are also making headway in reducing the effects of particular viruses; crop management practices are also being taught worldwide.

Another potentially very useful avenue for crop improvement is genetic engineering. Fauquet et al. (1992) described a model system for demonstrating CP-related resistance to cassava viruses with the use of ACMV and CsCMV CPs (as examples of most disastrous viruses of cassava in Africa and the Americas) in *Nicotiana benthamiana*. Other avenues may also be explored, such as the exploitation of defective interfering forms of the virus genomes (Stanley, 1990), or the use of virus-induced expression of toxic substances to generate an artificial “hypersensitive response” (Hong et al., 1996, 1997). Given the subsequent success of biolistic and other transformation techniques and regeneration for cassava (Li et al., 1996; Schopke et al., 1996), it is hoped that this is just a matter of time.

VII. Virus Diseases of Bananas

The banana (*Musa* sp.) is one of the most important subsistence crops in the world. It is widely grown in the tropics and subtropics in all types of agricultural systems, from small, mixed subsistence gardens to large multinational commercial monocultures. In many developing countries the crop serves as the staple food for the population or the cornerstone
of the country’s economy; thus, production figures (see Table II) are often not reflected in export figures, as most of the production is consumed locally. The largest producers are all located in Latin America and Asia; however, much of the American production in particular is an export crop to the developed world. The International Network for the Improvement of Banana and Plantain (INIBAP) is a division of the International Plant Genetics Resource Institute (IPGRI) (http://www.cgiar.org/ipgri/inibap/index.htm, inibap@cgnet.com), and has bananas as its mandate. INIBAP has as its mission to improve the productivity and yield stability of banana and plantain grown on smallholdings for domestic consumption and for local and export markets, and supports various initiatives on viruses of bananas. The IITA (http://www.cgiar.org/iita/home.htm) also includes responsibility for bananas, as does the Centre de Recherches Regionales sur Bananiers et Plantains (CRBP), Cameroon.

An increase in the international movement of banana germplasm has occurred in recent years, much of it to developing countries, particularly in the form of tissue culture propagated plants. The presence of any virus in such material therefore poses a risk as new viruses or strains may be distributed in large quantities to new sites.

Bananas are affected by five known, relatively well characterized viruses: banana bunchy top (BBTV, proposed “Nanovirus”); banana streak (BSV, Badnavirus); cucumber mosaic (CMV, Cucumovirus); banana bract mosaic (BBrMV) and Abaca mosaic (AbaMV, Potyvirus). New filamentous virus particles have also been noted in bananas from Africa, the Americas, Southeast Asia, and Australia (Lockhart, 1995; J.E. Thomas, personal communication, 1998). Another new virus, banana dieback virus, has also been described from Nigeria (Hughes et al., 1998).

A. Banana Bunchy Top

Of the known virus diseases of bananas, banana bunchy top disease (BBTD, caused by BBTV) is the most serious. BBTD seriously affects banana production in many areas of Southeast Asia and the Pacific (Thomas et al., 1994). The disease has been identified in numerous developing countries in Oceania, Africa, and Asia (Dooen, 1991; Thomas et al., 1994; Diekmann and Putter, 1996; Othman et al., 1996; Kenyon et al., 1997). BBTV is still absent from the countries of Central and South America. Strains of BBTV causing mild or
latent symptoms have been detected (Wu and Su, 1992), and the virus may occur at higher incidences than was previously believed.

BBTV has caused some devastating epidemics, the latest in Pakistan, where the disease was observed for the first time in 1988. By 1992 the disease was widespread in a number of districts in Pakistan, with an incidence ranging from 0.5 to 100%, and about one-half of the plantations had already been destroyed (Soomro et al., 1992; Khalid et al., 1993).

Successful methods of control, namely, early identification, eradication of infected plants, and the use of virus-free planting material, were successfully applied in Australia (Dale, 1987). However, these are unlikely to alleviate the problem in developing countries where farmers lack the organization to apply eradication programs throughout affected districts and funds are lacking to enforce such programs. Additionally, no incentives or alternate food sources exist, so a farmer will not sacrifice the small source of sustenance the family may have in order to save a larger area. Furthermore, virus-free planting material may not be readily available.

B. Banana Streak

Banana streak virus (BSV) is believed to be distributed worldwide on *Musa* sp. (Lockhart and Olszewski, 1993). It was not considered a serious problem of bananas until the 1980s. It is significant that the IITA is making development of tolerance and resistance to BSV one of its priorities in Africa. The disease was first noted in the Ivory Coast in 1966 (Lockhart and Olszewski, 1993), but the causal virus was not isolated until 1986 (Lockhart, 1986). Since then the disease has been and continues to be reported from many new countries (Jones and Lockhart, 1993; Diekmann and Putter, 1996; Tushmereirwe et al., 1996, Pasberg-Gauhl et al., 1996; Reichel et al., 1996; Vuylsteke et al., 1996). Evidence suggesting that the virus is integrated in the genome of *Musa* species and may give rise to episomal viral infection following *in vitro* propagation (Ndowora et al., 1999; Harper et al., 1999), has cast a spotlight on the role of *in vitro* propagation of *Musa* for low input production in developing countries. The virus causes a wide range of symptoms and damage ranges from mild to very severe. The virus is transmitted mostly by planting materials and also by mealybugs (Jones and Lockhart, 1993). It appears to spread in some countries but not in others (B.E. Lockhart
personal communication, 1998). In Uganda, a serious outbreak of the virus was reported in 1996, with some plantations containing 100% infected plants (Tushmereirwe et al., 1996). Damage appeared to be most severe when the virus was associated with an unidentified filamentous virus particle.

C. Cucumber Mosaic

Cucumber mosaic virus (CMV) is the type member of the genus *Cucumovirus* (family *Bromoviridae*), is worldwide in its distribution; has the largest host range of any plant virus, infecting more than 800 species; and is transmitted by more than 60 aphid species in a non-persistent manner (Palukaitis et al., 1992). The virus exists as a large number of strains that can be subdivided into two major subgroups, I and II, by various methods (Devergne and Cardin, 1973; Piazzolla et al., 1979; Edwards and Gonsalves, 1983; Rizos et al., 1992; Singh et al., 1995; Hu et al., 1995), all of which yield essentially the same subdivisions of isolates (Rizos et al., 1992). Subgroup I, typified by isolate DTL, occurs predominantly in the tropics and subtropics, whereas subgroup II, typified by isolate ToRS, is prevalent in temperate regions (Hasse et al., 1989). Both subgroups, however, occur on bananas (Diekmann and Putter, 1996). Strains on banana vary from those causing no symptoms to those inducing mild to severe symptoms (Diekmann and Putter, 1996). The heart-rot strain found in Morocco causes particularly severe disease (Wardlaw, 1972).

The virus may cause chlorosis, mosaic, and heart rot and is the etiological agent of infectious chlorosis disease in this crop (Niblett et al., 1994). In general, CMV does not have a major impact on banana production, but serious outbreaks have occurred (Bouhida and Lockhart, 1990; Li, 1995). The virus is currently considered an emerging threat to the cultivation of bananas in Kerala, India, especially where cucurbitaceous vegetables are cultivated as intercrops in banana (Estelitta et al., 1996). It is especially important to control CMV where mass propagation of *in vitro* banana material is employed, as levels of CMV can be high (Thomas, 1993), and bunch weight reductions of between 45 - 62% have been reported (Estelitta et al., 1996).

Although considered worldwide in their distribution, CMV and CMV strains have been reported for the first time on bananas in a number of developing countries since 1990. This is
probably due to the relatively recent availability of efficient detection and identification methods (Doon, 1991; Zambolim et al., 1994; Castano et al., 1994; Rivera et al., 1992; Osei, 1995; Srivastava et al., 1995; Allam et al., 1995; Li et al., 1996; Pietersen et al., 1998).

Infectious chlorosis disease symptoms of banana can be confused with those of BSV, BBrMV (Thomas, 1993; Thomas et al., 1997) or zinc deficiencies (Singh et al., 1995).

D. Other Viruses

Banana bract mosaic disease was first noted in 1979 in the Philippines, at Davao on the island of Mindanao (Thomas and Mangnaye, 1996). It was subsequently been shown to be widespread throughout the Philippines and also to occur in India, Sri Lanka, Vietnam and Western Samoa (Thomas and Mangnaye, 1996). Banana bract mosaic virus (BBrMV, Potyvirus) has been isolated from infected plants and is assumed to be the causal agent (Thomas and Mangnaye, 1996). Yield losses of up to 40% have been recorded in the Philippines on cultivars Cardaba and Lakatan (Magnaye, 1994). The symptoms of Kokkan disease of Nendran (AAB “French Plantain”) in India are the same as those described for banana bract mosaic, and BBrMV has been detected in these plants (Thomas and Mangnaye, 1996). The virus has also been detected in plants of Embul (AAB “Mysore”) with the same symptoms in Sri Lanka (Thomas et al., 1997). The virus has also been detected in India (Rodoni et al., 1997), Vietnam and Western Samoa with CMV-like symptoms but without the typical mosaic pattern on the bracts of the inflorescence (Thomas and Mangnaye, 1996). The virus has been found in symptomless infections (Thomas and Mangnaye, 1996). It is thus possible BBrMV is much more widespread than was previously believed. With the detection methods now available (Bateson and Dale, 1995; Rodoni et al., 1997; Thomas et al., 1997), a clearer picture of the distribution and importance of the virus is likely to emerge.

Nucleotide sequence information and serology (Bateson and Dale, 1995; Thomas et al., 1997) have confirmed that in spite of similarities in symptoms caused by AbaMV and BBrMV in bananas (Magnaye and Espino, 1990; Stover, 1972), the two viruses are distinct. This suggests that both have to be taken into account in surveys of viruses of bananas and in the indexing of Musa germplasm.
AbaMV, belonging to the sugarcane mosaic (SCMV) subgroup of the genus *Potyvirus*, was first found in the Philippines on *Musa textilis* (abaca or Manila hemp) (Eloja and Tinsley, 1963). The virus can be transmitted to edible banana and causes symptoms similar to those of BBrMV infections (Stover, 1972), from which it is, however, distinct (Thomas *et al.*, 1997). AbaMV has not been reported from any areas other than the Philippines, where it has significantly constrained abaca production (Diekmann and Putter, 1996). It must be assayed for in *Musa* germplasm to prevent spread of the virus to other areas through infected planting material. Further studies on the virus are currently underway in Queensland (Thomas *et al.*, 1997).

Unidentified filamentous virus particles have also been noted in bananas from Africa, the Americas, Southeast Asia and Australia (Lockhart, 1995; J.E. Thomas, personal communication, 1998). These appear to be related and are considered a probable potexvirus (J.E. Thomas, personal communication, 1998). The widespread nature of the virus, so soon after being detected for the first time, suggests that it may have a worldwide distribution. The virus has not been associated with a specific disease of banana so far, but it appears to increase the severity of the symptoms of BSV when it co-infects plants (Tushmereirwe *et al.*, 1996).

Banana dieback virus, a probable nepovirus, is a new virus of banana that has been identified in Nigeria (Hughes *et al.*, 1998). The virus causes leaf crinkling, leaf necrosis, and cigar-leaf die-back, and suckers from the same mats are progressively more stunted, ultimately resulting in severely stunted banana plants. The extent of the disease and the implications for banana/plantain production in sub-Saharan Africa are unknown, and are the subject of current investigation (Hughes *et al.*, 1998).

**VIII. Geographic Problems**

In a survey of peppers in Zambia, at least four viruses were identified: tobacco mosaic virus (TMV) and pepper mild mottle virus (PMMV; both tobamoviruses), potato virus Y (PVY, *Potyvirus*), and alfalfa mosaic virus (AMV, *Alfamovirus, Bromoviridae*). This apparently constitutes the first report of these agents in peppers in Zambia and is
therefore an important finding that may well influence future agronomic policy (Ndunguru, 1997).

We also solicited anecdotal evidence, from people working in given geographical areas and familiar with the problems there. Dr Josias Correa de Faria (Brazil; josias@cnpaf.embrapa.br) wrote that “a very recent outbreak of a geminivirus disease in cowpeas (Vigna unguiculata) in the northeast coast of Brazil appears to be caused by a virus distinct from either of the begomoviruses bean golden mosaic Brazil (BGMV-BR), or the newly described BGMV-BRII from the Pernambuco State (from Phaseolus lunatus); as well as from geminiviruses found on tomatoes in the same region. The so-called Bemisia argentifolii (=B biotype of Bemisia tabaci) is now being reported throughout the tropical and semi-tropical areas of Brazil in horticultural crops and on soybean, causing considerable damage to soybean during the current growing season in the state of Sao Paulo. Traditionally, BGMV-BR is the main viral disease of beans in the planting season which starts in February to March. Losses are variable with [the] year and [e]specially with [the] time of infection, but if disease incidence is high between 30 to 40 days after planting, losses can go from 40 to even 100%, if extremely severe. Bean common mosaic virus [BCMV] and bean common mosaic necrotic virus [BCMNv, Potyvirus] are well under control in Brazil through the use of the II gene”.

Dr Francisco Morales (CIAT, Colombia; F.MORALES@CGNET.COM) wrote: “I have witnessed the emergence of geminiviruses in Latin America almost from the beginning of the GV problem. My main line of research has been bean golden mosaic virus [BGMV, Begomovirus] in Brazil, Argentina, Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica, Dominican Republic, Haiti, Cuba and Mexico. This virus is by far the most destructive pathogen of beans in the lowlands of these countries. I guesstimate that over one million hectares traditionally planted to beans in Latin America cannot be cultivated or suffer 80 to 100% yield losses during the summer months of the year, when whitefly populations are at their peak. We (CIAT) have
worked in collaboration with national program scientists in the region to develop BGMV-resistant bean cultivars. Their development and adoption are the reason beans are still cultivated and consumed in the BGMV-affected areas. Severe outbreaks of bean dwarf mosaic geminivirus [BDMV, *Begomovirus*] in Argentina used to destroy more than 20,000 hectares every year in 1979 - 1982. Now, our resistant varieties and control measures protect Argentina's 120 000 ha bean crop.

"There are currently over 15 geminiviruses affecting tomato production in the Americas. I can inform you that geminiviruses have practically annihilated tomato production in the lowlands of tropical America, just like they did to beans over a decade ago. All of these problems are man-made and have been greatly exacerbated by the tremendous abuse of pesticides used to control whiteflies on these crops."

Dr Yule Liu (Beijing; liuyl@sun.im.ac.cn1) writes that tomato [yellow] leafcurl geminivirus was discovered in GuangXi Province, but that it was not very severe. Tobacco leaf curl virus disease, caused by tobacco leaf curl virus (TobLCV, *Begomovirus, Geminiviridae*), occurs in southern China, especially in Yunnan and GuangXi Provinces, and is becoming increasingly severe. Squash leaf curl disease (SLCD), caused by SLCV-Chi (*Begomovirus*), had been discovered in GuangXi Province, but was now inapparent. MDMV was very severe, especially in northern China; in 1996 in some parts of Shaanxi and Hebei provinces, up to 100% of plants were infected. Maize rough dwarf disease (MRDV) was becoming more and more severe in Hebei, ShangDong, Shaanxi, and Yunnan provinces; in some regions more than 40% of maize plants were infected, and up to 90% during 1994 to 1996. CMV was an important virus in vegetables. BYDV was an important virus in wheat in China. In rice, RDV and RStV were important, but probably less so than fungal and bacterial diseases.
IX. Emerging Disease Problems

There are three major groups of viruses that seem to be most important among new disease-causing agents worldwide, and that could reasonably be termed "emerging" in terms of apparently being new viruses causing new and serious diseases. The most important of these are in the taxonomic families Potyviridae and Geminiviridae, and the Tospovirus genus of the Bunyaviridae.

A. Potyviridae

The Potyviridae are the largest single taxonomic group of plant viruses, and the subject of more scientific papers per year than any other plant virus taxonomic group. It seems that potyviruses, and specifically aphid-transmitted viruses of the genus Potyvirus, are one of the most successful groups of plant pathogens in the world. The genus is apparently almost as ancient as flowering plants (Ward et al., 1994) and has a worldwide distribution throughout higher plants. New potyviruses seem to appear with new crop introductions just about anywhere these occur. In South Africa, for example, Passiflora edulis (passionfruit) cultivation in the 1980s was severely limited by diseases, many of which appeared linked to a mixture of potyvirus-like agents. One of these viruses has been characterized in detail (Brand et al. 1993) and found to be not the passionfruit woodiness virus (PWV) characterized in Australia and the Far East, but a distinct species now known to be a strain of cowpea aphid-borne mosaic virus (CABMV; Huguenot et al., 1996; Sithole-Niang et al., 1996). It is very interesting that a similar disease in Brazil seems to be caused by a closely-related strain of the same virus (M. Zerbini, personal communication, 1998), whereas woodiness disease in Australasia and Asia is caused by distinct species of viruses (Rybicki and Shukla, 1992). Several unidentified potyvirus-like agents have been found to be associated with diseased small grains in South Africa (Rybicki, 1984); beans and groundnuts in South Africa have also produced a number of new potyviruses (e.g., van Tonder, 1997; Cook et al., 1998).

Another intriguing aspect of emerging potyviral plant diseases is the appearance of hitherto latent virus infections. A well-documented case was the appearance in cultivated specimens of the horticulturally important flowering bulbs Ornithogalum and Lachenalia species of at least two distinct potyviruses. One of these, Ornithogalum mosaic virus (OrMV), first described in South African-derived bulbs in Holland, was found in plant specimens collected...
in localities where the plants were endemic. However, it caused apparent or overt infections only when plants were kept in cultivation (Burger and von Wechmar, 1988; 1989).

B. *Geminiviridae*

Viruses in the family *Geminiviridae*, and specifically the whitefly-transmitted begomoviruses, are presently associated with severe diseases in tomatoes the world over (Czosnek and Laterrot, 1997), with cotton disease in India and elsewhere (Padidam *et al.*, 1997), and with disease problems and food shortages due to disease in cassava in central Africa (Gibson *et al.*, 1996). Infections with bean golden mosaic virus (BGMV) are the single largest limiting factor to bean production in Central America (Faria *et al.*, 1994).

Polston and Anderson (1997) covered the Caribbean basin and the Central American region in an excellent review of the emergence of whitefly-transmitted geminiviruses (WTGs) in tomatoes. They estimated that 20 - 100% of crop loss throughout these areas has been caused by epidemics of WTGs, ranging from tomato golden mosaic (TGMV) to tomato yellow leaf curl (TYLCV) to potato yellow mosaic (PYMV). They listed 17 distinct viruses known to affect tomatoes in this region, and this is probably a conservative estimate, as more are constantly being found.

Much of the apparently emerging nature of the virus diseases is due to the worldwide spread of a new "silverleaf" or B biotype of the vector *Bemisia tabaci*, which is now being touted as the new species *Bemisia argentifolii*. The new vector has a much wider range of preferences for feeding than older vector types, which has apparently resulted in the spread of viruses that normally infect only weed or endemic plant species into adjacent, previously untargeted crop species. Roye *et al.* (1997) surveyed geminiviruses in weeds (notably *Sida* and *Wissadula* species) in Jamaica and showed a population of viruses distinct from crop-infecting geminiviruses - and one perhaps poised to enter crops once a suitable vector biotype appears. Although the problem also exists in the developed world, and most notably in the southern United States and Europe, it can be dealt with, to at least some extent, by changing cultivation practices. The same option does not exist in the developing world, however, due to the expensive nature of the measures (screen houses, heavy spraying regimes). Thus, the inexorable spread of the vector into these areas will almost certainly mean heavy crop losses.
This may have already happened in cassava in Uganda and western Kenya (Zhou et al., 1997, 1998a); however, the disease problem there is also associated with a natural recombinant virus (between ACMV and EACMV) with increased virulence. The scale of the problem was aptly illustrated by an anonymous agricultural researcher from Uganda, who said (to E.P. Rybicki, in 1997) that there was no cassava disease problem in eastern Uganda, as there was no more cassava to be infected!

Begomovirus diseases also seem to exemplify a phenomenon first noted with potyviruses; unrelated or distinct virus species in the genus cause very similar disease syndromes in the same crop plants, usually in widely separated geographical areas but sometimes in the same small area. Thus, TYLCV-Israel, -Sardinia, and -Thailand are distinct species of virus despite causing the same disease; however, so are the several viruses causing cotton leaf curl disease in India and Pakistan (Nateshan et al., 1996; Zhou et al., 1998b) and the several distinct viruses causing tomato leaf curl in India or even in Bangalore alone (Padidam et al., 1997). This is undoubtedly related to their worldwide distribution and also to their apparent antiquity. Rybicki (1994) has linked geminivirus variation to geographic separation and host plant genetic divergence, as well as to continental drift. All these factors indicate that the youngest age for the family Geminiviridae is hundreds of millions of years and that of begomoviruses is at least tens of millions of years. This diversity and ubiquity guarantee that a wide variety of virus genotypes exists, probably as well-adapted and essentially symptomless infections in endemic species or weeds, and that any change in transmission characteristics of the vector will result in appearance of the virus in crop plants almost instantly.

Whereas begomoviruses have been making their presence felt as they spread out of endemic hosts into cultivated species, viruses in the genus Mastrevirus continue to emerge into crop species in Africa. MSD was discussed earlier, and can hardly be considered as emerging. However, more recent findings indicate that a hitherto unsuspected and distinct group of MSV strains cause disease in wheat and some grasses, rather than the disease being caused by the same closely-related group of virus isolates and strains as are found in maize (Rybicki et al., 1997). Sugarcane streak virus(es) (SSVs) also still seem to be present in Africa. Whereas the better characterized variants SSV-Natal and SSV-Mauritius (Hughes et al., 1992) have not been a problem in southern Africa for over 30 years, and especially not in
commercially-grown sugarcane varieties, distinct mastrevirus species have been characterized from mildly diseased “traditional” material from Egypt (L. Bigarre and M. Peterschmitt, personal communication, 1999).

During the early 1990s a serious outbreak of a “yellow dwarf” disease known locally in South Africa as “oupatjies” occurred in the Phaseolus vulgaris L. (dry bean) winter seed production areas of Mpumalanga (northeast South Africa). Symptoms of the disease are brittle and leathery primary leaves, thickened and shortened internodes, and downward curling of trifoliolate leaves. Affected plants remain stunted and die after a few weeks. Although most plants die before producing seed, those infected at a more advanced stage bear only one or two pods, generally with only a few seeds, and seed reduction, by number, is between 85% and 92%. The seed produced is shrivelled and small, and the yield by weight is reduced by 93-95%. There was a 20% infection incidence per field throughout the seed production area in 1992 and 30% in 1993 but individual fields in the Malelane area were infected at up to 45% incidence (G. Pietersen, 1993, unpublished observations). The disease also occurred in the commercial bean production area of the highveld, but at relatively low rates (less than 1% of plants per field were infected). A new mastrevirus, for which the name "bean yellow dwarf virus" (BeYDV) has been proposed (Liu et al., 1997), was shown to be the causal agent. The virus was instrumental in the termination of dry-bean seed production in the winter seed area and may pose a serious threat to similar production in neighboring territories.

After anecdotal reports in 1997 of a typical TYLCV-like disease in the north of South Africa as well as in neighboring Mozambique, a survey was done in the Onderberg area. Disease was detected at a number of producers at an incidence between <1% and 50%, and yield losses on individual plants ranged from 0% to 100%, apparently depending on time of infection. The disease could be transmitted to healthy glasshouse-grown tomatoes by grafting and by field-collected whiteflies, but not by transmitted by mechanical inoculation. Typical geminivirus-like particles were detected in a number of plants; however, infected plants reacted very weakly with TYLCV antiserum in ELISA. Polymerase chain reaction (PCR) DNA amplification from infected plants using begomovirus-specific primers yielded a DNA product. Comparison of the nucleotide sequence of the fragment with cognate regions of other begomoviruses suggested that this was a new virus. It can be concluded that a
TYLCV-like geminivirus disease, new to South Africa, is prevalent in the Onderberg. The disease, with its associated virus, is a new threat to tomato production in South Africa as well as its neighbors (G. Pietersen, J. Brown, unpublished results, 1999).

A ray of hope in the struggle to combat TYLCV-like diseases is the fact that genetic engineering approaches using pathogen-derived resistance appear to be quite promising. Tomato plants transgenic for the TYLCV coat protein appear to be resistant to the virus (Kunik et al., 1994). Additionally, transgenic *Nicotiana benthamiana* plants expressing antisense RNA to the *Rep* gene were resistant to TYLCV infection (Bendahmane and Gronenborn, 1997). These developments, coupled with traditional and marker-assisted breeding techniques, may allow rapid dissemination of improved material to farmers.

C. Tospoviruses

The genus *Tospovirus* (family *Bunyaviridae*) is an extremely important group of plant viruses capable of infecting a large range of important crops. The type member, tomato spotted wilt virus (TSWV), has one of the broadest host ranges among plant viruses (Peters et al., 1991). Tospoviruses are transmitted exclusively by thrips in a circulative propagative manner, meaning that the virus multiplies in the vector (Wijkamp et al., 1993). TSWV was thought to be the only member of this genus until recently, and has had a serious impact on many crop species worldwide (German et al., 1992). It causes severe outbreaks in a large variety of crops grown in tropical and subtropical climate zones. Since 1990, other related but distinct viruses in this genus have been identified. The following members or tentative members are described: TSWV, groundnut ringspot tospovirus (GRSV), tomato chlorotic spot virus (TCSV), *Impatiens* necrotic spot virus (INSV), watermelon silver mottle virus (WSMV), and groundnut bud necrosis virus (GBNV) (de Avila et al., 1993a, b; Heinze et al., 1995).

Owing to the worldwide spread of the Western flower thrip (*Frankliniella occidentalis* Pergande), the most efficient vector of tospoviruses (Peters et al., 1991; German et al., 1992), TSWV and some of the other tospoviruses are increasing being reported as causing problems in developing countries (Theron and Pietersen, 1992; Dewey et al., 1995; Resende et al., 1997). These “new” tospoviruses often originate in developing countries, and some appear to occur only in these countries. For example, GRSV was first detected on peanuts during a survey of the viruses of this crop in South Africa, and isolate SA-05 now
serves as the type isolate of GRSV (Theron and Pietersen, 1992; de Avila et al., 1990; Adam et al., 1991). Thus far, this virus has been detected only on peanuts in South Africa, where it is relatively common (G. Pietersen and G. Cook, unpublished results, 1998), and on tomatoes in Brazil (Resende et al., 1997) and Argentina (Dewey et al., 1995). TCSV was first reported from Brazil (de Avila et al., 1990), and GBNV was first detected in India (Ghanekar et al., 1979).

Research on this important virus group has increased dramatically, and new tospoviruses are constantly being found (Adam et al., 1997; Resende et al., 1997; Yeh et al., 1997). It could be that only the lack of resources in developing countries for the diagnosis of hard-to-identify viruses prevents even more reports of new tospoviruses.

D. Banana Streak Virus / Pararetroviruses

BSV may be considered an emerging problem in light of findings suggesting, first, that it occurs in an integrated form in Musa (Lockhart, 1996); second, that it has been found in this form in all Musa germplasm tested thus far (R. Hull, B. Lockhart, personal communications 1998); third, that it is seed transmitted (Daniells et al., 1995); and fourth, that the integrated virus can be activated to form the episomal form through stress (Anonymous, 1997). Stress can possibly include tissue culture propagation (B. Lockhart, personal communication, 1998). Thus, it may be that initial BSV infections may largely be the result of plant stress, which means that BSV infections could occur anywhere, at any time, in the absence of any vectored introduction. It may be that genetic engineering in the form of a “knock-out” of the integrated viral genome can come to the rescue, at least of major commercial varieties.

X. Concluding Remarks

One of the most pressing reasons for comprehensive surveys of viruses affecting plants in the crop-growing areas of the developing world is the need to be aware of factors limiting crop yields in these areas. Although in many instances virus infections are suspected, these infections are never satisfactorily identified, and in many other cases it is not known whether crops are infected, let alone what effect the infection has on yield. Without a thorough understanding of the incidence and variety of virus infections in food crops in the developing
world, it is hard to plan for improved crop yields. However, developing countries are often unable to do such surveys on their own due to lack of expertise and resources. Given the lack of respect of borders by plant (or any other) viruses, it is hoped that the more developed neighboring countries will assist those less well endowed in these endeavors.

Another more future-oriented reason for surveys of viruses is the need to understand virus diversity in order to be able to combat virus diseases by genetic engineering of crop plants. Although there are many examples of crop plants transformed with viral genes or sequences exhibiting wide-spectrum resistance or tolerance to virus infection (e.g., Hackland et al., 1994), there are also many examples of an unexpected diversity of a given virus type being discovered within the crop in the same area or even within the same plants. A good example in this regard is the multitude of "cotton leaf curl" begomoviruses in Pakistan (Padidam et al. 1997; Zhou et al., 1998b). Thus, it is quite possible that the use of transgenic material in areas where the virus diversity is not known could still result in crop failures.

Another unifying concept emerging from the welter of different viruses and transmission mechanisms that cause severe plant disease in plants in developing countries is that the main problem is often one of insect/vector control. If the vector populations or the interaction of vector populations with crop plants could be controlled or managed better, the incidence of severe disease could be drastically reduced.

As a final note, it is well to realize that there are more direct threats to human health inherent in changing farming practices than simply a reduction in the amount of food being produced due to plant virus infections. For example, development of rice paddies in areas not previously used for this sort of agriculture can have profoundly damaging effects on local arbovirus epidemiology (Surtees et al., 1970). This could affect humans and their animals directly, in terms of increased incidence of insect-borne viral or other diseases.

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### Table I

**Primary Food Crop Production (Millions of Tonnes, 1997)**

<table>
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<th>PRODUCT</th>
<th>DEVELOPED COUNTRIES</th>
<th>DEVELOPING COUNTRIES</th>
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<tr>
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<td>284</td>
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<tr>
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<tr>
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<tr>
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All data ©Food and Agriculture Organisation; obtained from FAO Web site database
(http://apps.fao.org/cgi-bin/nph-db.pl?subset=agriculture)
Table II  
Top Developing Country Producers of Selected Food Crops in 1997

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<tr>
<th>COUNTRY</th>
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<td><strong>TOTAL</strong></td>
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<td><strong>TOTAL</strong></td>
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<table>
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<td>BRAZIL</td>
<td>6</td>
</tr>
<tr>
<td>CONGO (DR)</td>
<td>19</td>
<td>ECUADOR</td>
<td>6</td>
</tr>
<tr>
<td>THAILAND</td>
<td>17</td>
<td>INDONESIA</td>
<td>5</td>
</tr>
<tr>
<td>INDONESIA</td>
<td>16</td>
<td>PHILIPPINES</td>
<td>3</td>
</tr>
<tr>
<td>GHANA</td>
<td>7</td>
<td>BURUNDI*</td>
<td>1.5</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>166</strong></td>
<td><strong>TOTAL</strong></td>
<td><strong>57</strong></td>
</tr>
</tbody>
</table>

\(^*\)=included as biggest producer in Africa (not in order of production)

\(^1\)=excluding plantains

All data from the FAO Web site (http://www.fao.org)
Legend:
Main picture: cassava plant showing the effects of severe ACMD. Note lack of leaves, and of neighbouring plants. Insets, top: healthy leaves; middle, mild infection; bottom, severe infection. All photographs by EP Rybicki, taken in western Kenya, June, 1997.