Persley, G.J. and De Langhe, E.A.L. (eds.). 1987. Banana and plantain breeding strategies: ACIAR Proceedings No. 21. 197p.

Disease Susceptibility and Genetics in Relation to Breeding of Bananas and Plantains

Ivan W. Buddenhagen *

It is realistic to state that the real reason for both this conference and the interest today in breeding bananas and plantains lies in only one crop attribute — disease susceptibility. Hence the title of this paper, and its emphasis: *susceptibility*. Although resistance may be the goal of research investment, the real driving force for investment is susceptibility, which has been, or is perceived to be, changing in the last decade or two. The most important change has been the appearance and spread of more virulent forms of Sigatoka leaf spot which are now present in the Pacific Islands, Latin America, and Africa. The 'new' forms are not only more virulent on their traditional banana hosts but they now endanger the plantains, unlike the old Sigatoka. Thus they generate much more interest among organisations concerned with peasant agriculture and local food supplies in the lowland wet tropics.

The second change is the appearance of a race or races of the *Fusarium* wilt pathogen on Cavendish bananas formerly considered resistant. This has occurred not only in widely separated locations in the subtropics (Taiwan, Australia and South Africa) but also in the equatorial climate of Mindanao in the Philippines. Although it has long been considered that the great banana export industries in the tropics were not endangered by wilt after successfully converting to resistant Cavendish clones, the present experience in Mindanao belies that assumption. However, the fact that wilt has not appeared in Cavendish clones in Latin America or Africa, and that the AAB plantains remain unaffected everywhere, tends to reduce the urgency of concern. There is localised concern in a few places, but largely, the problem is seen as a potential threat rather than an actual one. Brazil is seen as a special case, where there is a need for wilt resistance for the widely planted and susceptible AAB cultivars.

Once one considers embarking on a resistance breeding program, driven by the actual or perceived threat by only these two pathogens, other useful objectives become apparent, especially, reduced susceptibility to various nematodes, and in some areas, reduced susceptibility to bacterial wilt (Moko disease) and to fruit spotting fungi. Then one may question if resistance to bunchy-top or other viruses should be considered. That leads to the question of resistance to the virus vectors and even to other insects. Does one include such objectives in a breeding program? Such reasoning may lead one to question to what degree susceptibility to minor pathogens may be *increased* by any breeding effort targeted only at the two major pathogens, and how much one should be concerned with 'preventive' breeding.

And last, but not least, is the need to be concerned with all of the diverse agronomic and quality aspects of a highly sophisticated industry for dessert bananas and of different consumer groups. It becomes apparent that beyond the worldwide needs there are localised and specific needs in different regions. Given the diversity of needs and the difficulty of obtaining recombination in this parthenocarpic triploid crop and the very limited number of sustained breeding programs, it is not too surprising that success has not yet been achieved: All the bananas and plantains that we grow and eat were selected in prehistory by primitive peoples!

Breeding for Resistance: General Considerations

It is important to know as much as possible about the existing variability of the pathogens against which one is trying to breed, and also about their potential variability and the ease with which a new virulent form, once selected, can replace the old pathogen population and thereby render ineffective the results of a resistance breeding program.

For *Musa* the world may be divided into two parts: 1) a coevolved pathosystem region of Southeast Asia; and, 2) the rest of the world where *Musa* is of fairly recent introduction.

* Agronomy Department, University of California, Davis, CA 95616.

The areas of recent introduction are Latin America and Brazil, the Caribbean, and Australia. These represent the locations of most research on bananas. Pathology research has been most intensive in Central America but with some research also connected with the export trades in the other new areas and in the Philippines and Taiwan. The new areas are all radically different from the coevolved pathosystem areas in that most research has been confined to only two basic AAA clones: Gros Michel and Cavendish (all Cavendish clones are grouped in this paper as one for pathosystem analysis). A small amount of research has been conducted on Silk/Pome (AAB) types in Brazil and on Bluggoe (ABB) and plantains in Central America but basically only a few clones have sampled the environment for local non-coevolved but potential pathogens over wide areas. Moreover, it means that any pathogens occurring in the new areas following their limited introduction from the centre of host origin have had very little host diversity on which to propagate and diversify.

This means that any sampling of Cavendish, for example, for the Sigatoka pathogen, in any new region should result in cultures with identical pathogenicity genes. At least there would have been no reason for selection and/or preservation of pathogen diversity. Now that Sigatoka forms exist that attack plantains, one could consider that the plantains might select a different pathotype; at least the possibility exists.

The presence of a sexual stage and the rapid aerial dispersion of the Sigatoka pathogen, which has been amply demonstrated with Black Sigatoka in Africa and Latin America recently, show that deployment of a new cultivar, with new and different resistance genes, would exert great selection pressure on the existing Sigatoka population. A 'breakdown' of resistance would be limited only by the flexibility of that pathogen population in mutating to new virulence. Once mutated, a new pathogenic form would be dispersed rapidly, especially since, on the new 'resistant' clone, spraying would have been stopped or be at a low level.

In thinking about breakdown potential, one must consider pathogen population size and location. In the case of Sigatoka, if a banana industry converted to a resistant clone, there would be only a short period of time (a few years at most) for complete replacement, during which abundant spores (from the old variety) would be challenging the new one, mostly at the transition zone. After that, challenge inoculum would no longer exist within the plantation and the risk of breakdown would be extremely low. Presumably the region would preserve a limited inoculum on household plantains scattered about. If a resistant plantain were also introduced, the Sigatoka population would become so low that it could become locally or regionally extinct.

The point of importance right now in assessing Sigatoka variability in the laboratory is that cultures collected from the *same host* over a regionally intact area outside the area of coevolution should have very low variability. Thus, their analysis will tell us little about potential variability. What is needed is to collect anew, from as many *different* genotypes as possible, in each region. The best place is a banana collection. Lacking that, a diverse set of genotypes could be planted in each region purposely to act as a trap for pathogen variability. Such a genotype set should contain the major parents being used in breeding programs around the world, and be updated periodically.

For the *Fusarium* wilt fungus, the story is somewhat different. The presumption is strong in Central America, the Caribbean, West Africa, Australia, etc. that the wilt fungus was moved about extensively in seed pieces of Gros Michel. Whether or not the fungus was originally introduced from Asia or had a separate origin in Latin America, etc. is not known. New research could provide a probability answer. If it is an introduction, then one would expect little variability; again, because it had only one static host. Cultures from banana from one introduced region should be very limited in pathogenic variation. If, on the other hand, indigenous wilt fungi in the new areas evolved to attack the introduced host, there should be much more variation.

Certainly, the very discovery and existence of the races of *Fusarium oxysporum* f.sp. *cubense* were due to appearance of wilt symptoms on banana/plantain cultivars earlier considered resistant to the fungus attacking Gros Michel. The exception is race 3 which was discovered on *Heliconia*, where a *Fusarium* was purposely sought after it was discovered that *Heliconia* was the original host of the bacterium that caused the new-encounter Moko disease of banana in Latin America (Buddenhagen 1964). There is no question that the *Heliconia* wilt Fusaria are locally evolved in Latin America on this genus. Whether their diversity includes forms that created the American Panama wilt of banana was never proven, but it still remains a good possibility. New research should be able to answer this question.

The question of separate origin in Latin America for the banana wilt *Fusarium* is not all irrelevant to a consideration of breeding for resistance because it should influence greatly the possibility of resistance breakdown of new cultivars bred from Gros Michel.

Unfortunately, a further unknown is the level of challenge that will confront a new cultivar. After 25 years of cultivation of resistant Cavendish on soils where wilt devastated Gros Michel, what has happened to the inoculum? Is it still there at high or

moderate levels, even now, and more genetically diverse due to its life as a saprophyte or rhizosphere organism? I am not aware of information that answers this question, important to a banana breeding program that is considering the deployment of a new cultivar.

If a new cultivar became widely planted and a single rare mutation (or even a few) to new virulence then occurred, spread to become important should be slow if what we think we know about spread is correct.

In the region of the world where *Musa* and its two great pathogens are coevolved the situation should be very different in terms of diversity of genes both for pathogenicity and for resistance. But exactly what part of the *Musa* homeland also contains these pathogens as indigenous in any active coevolving relationship? Does it extend to India, to Fiji, to Papua New Guinea? Much pathosystem analysis is needed in the presumed centres of origin. Since *Fusarium* wilt is endemic in Southeast Asia in AAA, AAB and ABB karyotypes (Vakili 1965), it is highly probable that its variability there is much greater than is represented by our concept of the four races.

Clonal replacement Although those in the banana business have some idea of the cost and magnitude of effort required to restock lands with a new clone, newcomers may underestimate how these considerations may influence strategy and feasibility of banana breeding schemes.

For the established banana trades where Cavendish is not apparently threatened by *Fusarium*, a *Cercospora*-resistant clone based on *Fusarium* wilt susceptible Highgate would find considerable opposition in company management. They would be concerned with the risk of breakdown of the *Fusarium* wilt resistance, and have to balance that against the financial advantages of Sigatoka resistance.

Plantain culture, now threatened in many places by Black Sigatoka, is in a completely different position. If a new plantain can be constructed from two *Fusarium* wilt resistant (both races 1 and 2) parents, it would be accepted immediately and would be pushed by agencies concerned with local farmer welfare.

For Pacific Islands where black leaf streak or Black Sigatoka devastates formerly successful banana export production and where *Fusarium* wilt is not a threat, a *Cercospora*-resistant clone somewhat less perfect than ideal might still work and be a great boon.

In Brazil, where special clones of AAB are desired, a *Fusarium*-resistant clone with good Pome/Silk type taste would be readily adopted. The question of Black Sigatoka resistance has to be handled by the breeding program outside Brazil since it does not yet occur in the country.

Is it really realistic to think of a continuing breeding program that would result in one improved variety after another — or the need for replacement of 'collapsed resistance'? Or are we indeed confined to a 'one-shot' deal for either or both *Fusarium* and *Cercospora* resistance? Would sufficient financial support continue for maintaining a good breeding program? The answers are not clear but they will differ from place to place and they require local analysis.

The economics and the feasibility of variety replacement should be studied in each region as a prerequisite to making decisions on breeding and on breeding strategy.

In judging breeding for resistance and the benefits of resistance to the one or two major pathogens, one should also estimate the cost of nematodes to the local industry in terms of control, yield reduction, and either field shifting or replanting frequency. Certainly for plantains, resistance to several nematodes, and probably to their accompanying fungi, needs to be considered a primary target, along with resistance to *Cercospora*.

The potential for increases or decreases in yield and quality must also be considered in breeding and selection for resistance. Each region has different standards and different possibilities and dangers. If yield and quality are high, as for Grande Naine in Latin American export trades, the difficulties of matching these standards are great. For less sophisticated industries, the situation is different. For plantains, inherently low yielding, the possibilities and needs for combining both higher yielding ability with higher nematode and *Cercospora* resistance offer added incentives for breeding.

The *Cercospora* Pathogens

The banana breeding programs in the Caribbean and Honduras were, prior to about 1970, confronted only with Yellow Sigatoka (YS). Presently, only the breeding program in Honduras has Black Sigatoka (BS) challenging its parents and progenies. The programs in Jamaica, Brazil and Guadeloupe are located in areas where neither Black Sigatoka nor Black Leaf Streak (BLS) exist. The nascent program in Guadeloupe plans to use Cameroon as a screening site for Black Sigatoka. Thus, the only breeding program with considerable experience and information on resistance to Black Sigatoka is that in Honduras. However, collections exist in several areas where BLS/BS occurs in favourable environments and these either have been scored for relative susceptibility, or they could be (Pearson et al. 1983; Foure 1985). Local reports exist for scoring of collections in the Philippines, Nigeria, Cameroon and Honduras. Relative susceptibility of

the same material in these diverse sites should be assessed. Apparently genotypes rank in the same order of susceptibility for Black Leaf Streak as for Black Sigatoka. On limited observations to date there is no evidence that these two names represent different virulence or pathogenicity genes. Contrarily, both, when compared with Yellow Sigatoka, have different genes which increase their host range and their virulence. General observations indicate similar ranking but less susceptibility with YS, but careful comparisons have not been made.

Almost all our knowledge of the Sigatoka pathogens and their biology comes from their existence in banana plantations, on Gros Michel or Cavendish. An exception is the work of Brun (1963) in Guinea. Another exception is Meredith's work (1970) in Hawaii where his studies were based on an isolated varietal collection. In this case, however, he was working only with *Mycosphaerella fijiensis*, the Black Leaf Streak pathogen. A flurry of work followed the appearance of Black Sigatoka in Honduras wherein the essentially identical disease symptoms were considered to be caused by a form (var. *difformis*) of *M. fijiensis* (Stover 1976).

Of considerable scientific interest would be to determine if the occurrence in Honduras was an introduction or a new and separate evolutionary event. I have hypothesised that since it occurred in the only location in Latin America where great host genetic variability existed, it would have been possible for new and different mutants to have been conserved on different hosts and for them to have recombined virulence genes to create a 'new' pathogen. It should still be possible to investigate this hypothesis by assaying the fungal variability in this site of possible evolution and comparing it with that in both the distant areas into which the 'new' form subsequently spread and the areas from which it might have been introduced (such as the Pacific Islands). A key question also is to determine how genetically different the BS pathogen is from the YS one. Alternative to the idea of a new evolutionary event would be that of introduction from the original coevolved centre. This clearly happened for BLS in West Africa (from Taiwan) and it could well have been so for the Honduran event since the germplasm collection and breeding program are the site for international visitors and traffic as well as for planting stock introductions. At first, the difference from BLS in sporodochial production indicated a new evolutionary event, but the findings by Stover (1976) that pathogens indistinguishable from the Honduran BS were widespread in the Pacific Islands strengthens the introduction hypothesis.

The most interesting and perplexing phenomenon has been the replacement (and apparent extinction) of the Yellow Sigatoka pathogen by the BLS/BS pathogens as they have spread in Fiji, Central and South America and Africa. Stover (1976) travelled through the Pacific area and Asia in 1975 and concluded that the BLS pathogen was present (and not BS) in Hawaii and the Philippines; that the BS pathogen was present (and not BLS) on all other Pacific Islands (including the Solomons, Papua New Guinea) and Taiwan. His collections from Fiji revealed only BS, but he cites Meredith and Firman (1970) wherein they found in the Nadi area of Fiji the Yellow Sigatoka pathogen as well as that of BLS. At that time the BS pathogen (distinguished from that of BLS only by sporodochial production) was not known. Stover found that the BLS and BS pathogens could not be distinguished in culture and that they were clearly separate from the Yellow Sigatoka pathogen. Collections received by Stover in 1975 from Bogor (Java), Indonesia and Kuala Lumpur in Malaysia revealed only *M. musicola*. My observations in Sri Lanka in 1984 were that only *M. musicola* was present. Of considerable interest is Stover's re-examination of herbarium material dated 1927 from Taiwan, material which proved to have the pathogen of BS. Pearson et al. (1983) confirmed the presence of only BS in the Port Moresby area of Papua New Guinea.

No one has gone back to the pathogens' homeland in Southeast Asia and either collected extensively for mycological and genetic work or has studied the diseases and pathogens in situ in scattered domestic plantings or in the wild. Thus, we do not know if the three Sigatoka forms, or many more, are living together there in a compatible fashion. We also do not know the most important point of all — if they can interbreed. My prediction is that the BLS and BS pathogens will intercross freely and that they will segregate the sporodochial trait. If this is so, then collections from BLS/BS symptoms in the original area of evolution will reveal both sporodochial and non-sporodochial isolates. If indeed there are islands or continents where only one type occurs it would mean limited introduction and then spread of only one or the other. Although the probability of crossing of BLS/BS with YS is less, on observations which indicate a lack of intermediate symptom forms, crossing should certainly be considered and attempted. It could be that simple 1:1 inheritance occurs (in symptom expression) and that the extinction of YS is purely epidemiologically based.

The Yellow Sigatoka pathogen (*Mycosphaerella musicola*) was proven to be hermaphroditic but heterothallic, with a single pair of alleles controlling ascospore production (Stover 1963). These findings were from pairings conducted on banana leaves, since ascospore production in culture has not been obtained. The BLS/BS pathogen also produces abundant ascospores in nature. The type of its

sexuality has not been reported.

There is little work on genetics of resistance/pathogenicity with other *Cercospora/Mycosphaerella* species. Thus, we have no guidelines to help us as we approach the subject for *Musa*.

Susceptibility to the Cercosporas

Apparently all *Musa* can be infected even by YS but subsequent fungal growth is more rapid and extensive in some than in others. The more rapid and extensive the fungal growth the greater the symptoms. Resistance is relative and is seen as lesions appearing on lower (older) rather than upper leaves, and fewer rather than more lesions in a certain group of numbered leaves. Various refinements based on timing of the key stages of the infection cycle and on sporulation have been used, but basically, *relative* susceptibility is easy to estimate. FAO (1971) has produced a rating system. The key need is to remove environmental variance across sites and dates, by the use of a set of standards with known levels of resistance/susceptibility. No one has used such clones as standards in evaluating progeny in breeding programs. A new set of differentials is now needed for BLS/BS. This is especially important since environmental conditions markedly influence lesion numbers and development time. Much less disease occurs in the dry season. Even temperature alone changes the host-parasite interaction to the extent that AAB plantains, disease-free in the lowlands, show considerable Sigatoka (YS) at 1000 m in the coffee-growing areas of Colombia.

It is generally considered that ABB cultivars are more resistant, but too much reliance on genomic origin obscures the point that plenty of resistance occurs within the species *M. acuminata* and this frequency varies by subspecies and original location. The most extensive and interesting work on comparative susceptibility was done 20 years ago in connection with the early breeding work in Honduras (Vakili 1968). Seventy per cent of seeded AA diploids were classed as resistant. This decreased to 26% for edible AAs and 19% for AAAs (Table 1). The frequency of resistance (to YS) based on geographical origin differed markedly: 94% of entries originating from Southeast Asia/Philippines were resistant versus only 19% of those from Papua New Guinea/Solomons (Table 2). This must tell us something about the original area of evolution of YS and/or of contrasting environments. The frequency of resistance in edible diploids decreased compared with seeded diploids and the frequency of resistance in triploids decreased even more. Although dosage effects for increased susceptibility might be implicated in the triploids, the lower frequency in edible diploids must indicate that either the resistant wild progenitors gave people fewer 'good' edible types and/or that Yellow Sigatoka was not important enough in scattered village plantings to be selected against. All of this should be reexamined for BLS/BS with more diploid collections. The areas of coevolution of BLS/BS forms on *Musa* are possibly different from areas of YS evolution.

Table 1. Yellow Sigatoka resistance/susceptibility of *Musa acuminata* in relation to parthenocarpy and ploidy.*

Type	Resistant (%)	Susceptible (%)
Seeded AA	70	19
Edible AA	26	60
Edible AAA	19	76

* After Vakili 1968. Based on 341 accessions.

Table 2. Yellow Sigatoka resistance/susceptibility of *Musa acuminata* in relation to geographic origin of accessions and their ploidy and parthenocarpy.*

Region	Resistant (%)	Susceptible (%)
Southeast Asia/Philippines		
Seeded AA	94	6
Edible AA	34	66
Edible AAA	27	73
New Guinea/Solomons		
Seeded AA	19	81
Edible AA	14	86
Edible AAA	10	90

* After Vakili 1968. Based on 433 accessions.

Regarding genetics, little is known publicly beyond the work of Vakili (1968) on YS with crosses of susceptible *M. acuminata banksii* with a resistant clone of *M. acuminata microcarpa* and with resistant *M. acuminata errans*. He concluded that multiple factors were involved and that resistance was partially dominant. Both resistant parents were partially heterozygous for resistance and the Zebrina clone of *microcarpa* had more resistance genes than the *errans* parent. It is of considerable interest that some resistant clones are carrying genes for susceptibility.

A cross between the homozygous susceptible *banksii* and a resistant edible diploid, 'Lidi,' gave all resistant F_1 progeny, indicating that at least one dominant gene conditioning resistance is present in the homozygous condition in 'Lidi.' Yet, when 'Lidi' was crossed with a short Gros Michel (Cocos, AAA) most progenies were susceptible. Although Vakili speculated that this was due to a dosage effect, additional reasons could be advanced. Since some F_1 progenies were resistant it could be that resistance in 'Lidi' is conditioned by multiple factors, some of which are in the heterozygous state.

Unfortunately, no further information on genetics is available from this work. Twenty years earlier a report from Jamaica suggested that resistance to Sigatoka (YS) was a dominant character (Larter 1947 see in Rowe and Richardson 1975).

Without further genetic work on resistance and susceptibility, using diploids and carrying through to F_2 and backcross generations, it is impossible to be more precise about genes conditioning resistance. Moreover, such work needs to be redone now for BLS/BS as well as YS.

With the ready sexuality of the pathogens, the genetics of pathogenicity and virulence and the genetics of the various diploid hosts for resistance/susceptibility should be readily determinable. This is crucial to any banana/plantain breeding program and of considerable scientific value as well. If unpublished data are available on this subject, every effort should be made to review the work and publish it.

It is probable that the degree of susceptibility is conditioned by several or many genes since 'susceptibility' across genotypes appears to be a continuous spectrum of degree. It must reflect many host processes which interact, over considerable time, to limit or not limit fungal development. On the other hand, it is quite probable that resistance can be conditioned by a single gene, blocking fungal development at one of many potential interacting sites. Thus, 'resistance' will be found to be due to different single genes or combinations thereof, in different materials.

Toxins Pertinent to discussions of susceptibility/virulence is work on a toxin produced by many cercosporas (Daub and Briggs 1983; Assante et al. 1977). It is especially interesting in relation to possibilities of tissue culture selection for resistance (Daub 1986). The toxin, cercosporin, is unique among fungal toxins in being a photosensitising agent. In the presence of light, cercosporin causes the peroxidation of polyunsaturated fatty acids in membrane lipids, leading to rapid membrane leakiness. The symptoms of the Sigatokas, with tissue watersoaking and the suppression of the disease under shade certainly fit a hypothesis of cercosporin-mediated pathogenesis. Relative susceptibility and relative virulence (of BS vs YS) could reflect relative production of cercosporin in host tissue. Thus, resistance could be seen as a suppression of cercosporin production. Thus, one could believe that selection for cercosporin resistance in tissue culture would be a potential avenue to pursue. But this may be a trap for the unwary. First, studies on testing *C. musae* for cercosporin production (amongst 60 other cercosporas) listed it as negative (Assante et al. 1977). This cursory screen may need rechecking. Either cercosporin or some other secondary metabolite may be produced that governs relative host susceptibility. It would be of interest to see if the cercosporas which produce or do not produce cercosporin fit into different perfect genera of fungi.

If cercosporin is the important compound in pathogenesis, can one expect success using it as a screening agent? Daub (1986), in a review of tissue culture for selection of resistance to pathogens argues convincingly that the answer is No. This follows her own failure to select resistant cells of sugarbeet and protoplasts of tobacco. An appraisal of the original thinking of why cercosporin seemed an ideal candidate for in vitro selection reveals why this thinking was illogical (Daub 1986). Not only is cercosporin toxic to plants carrying resistance to the pathogen itself but it is toxic to all plants tested. Moreover, it is the basic structure of the polyunsaturated fatty acids that makes them susceptible to peroxidation and their essential function is tied to this basic structure. The idea that cells might be able to detoxify cercosporin is not favored by the nature and site of action (plasma membrane). If, indeed, cercosporin is effectively the mediator of pathogenesis, then increased resistance has to lie in host suppression of cercosporin production. Thus, resistance/susceptibility would have to be judged directly and we would not be able to direct the selection of higher resistance levels except through choice of parents and use of a standardised realistic challenge wherein the suppressive host effect can be judged accurately.

That somatic mutagenesis might result in resistance is not an impossibility and this could be pursued through tissue culture and somaclonal variation. No positive results are known to have been obtained so far, but would they be noticed in commercial operations where the plantings are sprayed anyway? An old report (Drummond 1964 — mentioned in Meredith 1970) mentioned suckers of a susceptible mother plant of an AAB clone being resistant to Sigatoka.

The *Fusarium* Pathogens

There is abundant literature on the fusaria in general (Nelson et al. 1981) and on *Fusarium oxysporum* f.sp. *cubense*, the banana wilt pathogen (Stover 1962, 1972), and on other formae specialis of *F. oxysporum*, the xylem-invading *Fusarium* causing wilt of many crops.

Much of the literature on pathogen variability both in culture and in nature is confusing and conflicting and for *oxysporum*, a species without a known perfect stage, genetic information is entirely lacking. Identification of fusaria is an art usually left to a few specialists, and *Fusarium* taxonomy is a continuing bone of contention among specialists.

It is conventional to throw all fusaria isolated

from plants suffering a wilt disease into the species *F. oxysporum*, but not all oxysporums induce wilt symptoms.

It is also conventional to consider that fusaria which wilt different host species are different themselves and they are usually given different names at the level of 'formae speciales.' If the hosts, however, are considered sufficiently related, their wilt fusaria may be left with the same name but given a 'race' designation. Thus, we have race 3 of the banana wilt fungus, which is really a pathogen of the different genus *Heliconia*. Most 'races' however are described when a variety or cultivar, earlier considered resistant to a known wilt disease, is seen as susceptible. If, on inoculation, the first host is not attacked by the isolates from the newly susceptible cultivar then a new race is established and one has two races and two differential cultivars. This is what happened when Bluggoe was first found diseased in Honduras and race 2 was established. However, if a cultivar is newly found to be susceptible and the isolates still attack the old cultivar, then a new race is still established. The two hosts do not differentiate the races; rather, only the new cultivar detects that there is a different pathogenicity. This is the case for race 4 in Taiwan (Table 3). The situation of *Fusarium* wilt of Cavendish bananas in Taiwan is reviewed by Su et al. (1986).

Table 3. Races of *F. oxysporum* f.sp. *cubense* as defined by the reaction of three cultivars. The differentiating characteristics are shown in dotted boxes.*

Cultivar	Cocos AAA	Bluggoe ABB	Cavendish AAA
Race 1	+	−	−
2	−	+	−
4	+	+	+

* Race 4 data are for the Taiwan strain.

The implications in the first case, if one considers the new race to be derived from the old, is that a single virulence gene mutated to avirulence for the first host while simultaneously acquiring virulence to the second. The implication in the second case is that the fungus has retained the old gene for virulence but has acquired another virulence gene at a separate locus, giving it new virulence. Of course this is pure speculation since no genetic studies have been conducted. Since race 4 is also virulent on the race 2 host, one could just as well suggest an alteration of the race 1 virulence gene to a third form, enabling it to expand its hosts without losing its race 1 virulence. Thus, without a set of differential hosts, the situation we have today is that any strain capable of readily attacking Cavendish clones would be called race 4. That essentially is the current definition of race 4.

The key questions needing answers in relation to a rational resistance breeding program for *Fusarium* wilt are:

(1) Are the strains from Cavendish from Taiwan, Philippines, Australia, South Africa, etc. the same, with the same virulence genes, or not?
(2) Do they represent different evolutionary events?
(3) Has Cavendish successfully selected its pathogen in each case due to environmental factors acting on the host/parasite system, or is it due to the greater genetic potential of the *cubense* already in those particular soils?
(4) If it is due to the greater genetic potential of the local *cubense*, was this potential really a result of the presence of prior different *Musa* hosts such as Abaca in the Philippines, Silk in Australia and Latundan and others in Taiwan?
(5) If it is due to the latter, were these host/cultivar systems indigenous or were they themselves brought from other centres of origin?
(6) What is the genetic variability of the wilt fusaria in the centre of origin of *Musa*? (a) In what areas is it indigenous? (b) Is selection pressure exerted on the wild species? (c) Do all the known races, and many more, representing different virulence and pathogenicity genes, occur? (d) What wilt fusaria occur on other Scitamineae in Asia?
(7) Are the *cubense* in Latin America indigenous and if so, are they coevolved with certain *Heliconia* species in certain places and have they spread from there?

Stover and Buddenhagen (1986) proposed a greater pathogen variation in the centre of origin and a need for testing identities of race 4 strains from different locations. They also suggested that the Philippines race 4 might have been introduced from Taiwan, but this is probably incorrect, based on the mild pseudostem symptoms and on colony cultural differences detailed by Dr Shirley Nash-Smith (pers. comm.).

Dr Nash-Smith believes that the Taiwan and Philippines isolates differ markedly from each other (in culture) but they are similar in each region. She believes that they are not related and that the Philippines Cavendish-attacking *cubense* derives from Abaca. She also believes that the Latin American cubenses have great variability (the old Gros Michel ones) unlike the Philippines and Taiwan Cavendish isolates. She has not seen the Australian isolates of race 4.

Thus, we need many more definitive studies, using modern methods, to explore genetic relatedness as well as virulence/pathogenicity genes of *F. oxysporum* f.sp. *cubense*.

One point remains clear. The reason Cavendish remains resistant in the vast areas of Latin America where it was introduced into soils containing untold billions of *cubense* propagules is that the gene or genes necessary for virulence to Cavendish are not easy to acquire. If the local fusaria had those genes, Cavendish would be attacked there as well. It speaks for tremendous stability of this resistance and it leads one to believe that the capacity for appropriate virulence genes must come from prior coevolved systems.

Information from other wilt/host systems Is there any information on other wilt/host systems which might influence our thinking? *Fusarium* wilt of peas in Washington State became important shortly after the pea industry started in 1924 and within 6 years resistance to race 1 was needed and shortly incorporated (Kraft et al. 1981). Race 1 resistance due to a single dominant gene was adequate for 30 years but then two new races became important. Subsequently, up to 1980 three more races or strains have appeared. Thus, under monoculture, the deployment of varieties with different genes for resistance resulted, with varying lag times, in the appearance of 'races' overcoming the resistance. Even in this easily bred crop no satisfactory variety exists carrying resistance to all the races, so soils are assayed to determine the race present to enable the right variety to be grown.

The tomato story is somewhat different. Wilt was once the most common and destructive disease of tomato (Jones and Woltz 1981). A wild species was found to carry a single dominant gene conferring resistance and by 1941 resistant commercial tomatoes were in use. Race 2 was first reported in 1945 but it did not become of widespread concern until 1961. It was then reported from Florida, USA and rapidly from other states in the USA and countries as distant as Australia, Brazil, Morocco, and England. This nonimportance for years and then rapid importance in widely separated areas may mean either (1) seed transmission, or (2) wide deployment of cultivars which act as a specific screen for the new race. If earlier cultivars were not selective and since all had the same dominant resistance gene, then this selectivity trait has to be due to background genes. Resistance to both race 1 and race 2 was found in an interspecies hybrid and it was quickly incorporated into tomatoes everywhere. Two dominant single genes are said to be responsible but all known lines resistant to race 2 also carry resistance to race 1. Jones and Woltz (1981) argue that these major genes are adequate and better than the use of polygenes conferring tolerance. They also argue that in spite of reports on nematodes reducing the resistance conferred by the major genes, there is no field evidence to support this contention. Since their 1981 report, a race 3 has been reported in Australia and elsewhere, so far with limited distribution.

The cotton wilt pathogens are designated as five races which are geographically separated (Smith et al. 1981). Armstrong and Armstrong (1978) reported six races on cotton. The pathogens are not confined to cotton and races 1 and 2 are differentiated artificially on soybean and tobacco. Seed transmission could explain the widespread presence of race 1 in USA and also in East Africa. Although breeding for resistance has been conducted since 1900 greater resistance is still needed in combination with high quality cotton. Studies on the inheritance of resistance have indicated everything from one dominant gene to additive polygenes, depending on the cross and the methods of challenge. It appears that adequate screening facilities and techniques are not available or used in many breeding programs and this lack limits progress.

The only paper wherein I have found a concern for and experimental work on what formae specialis and races really mean and where they come from is that of Bouhot (1981) in France, working with the melon wilt fungus. Salient points are that prior to 1963 only melons not carrying resistance genes were cultivated in France. Yet when varieties carrying either one or a second gene for resistance were introduced they were widely attacked in the second year. Moreover, some cultures made before introduction of the resistance genes were shown to carry virulence for the genes. Bouhot easily induced mutations in microconidia with nitro-soguanidine, and could create one race from another. He even made new races, not yet known in the field. He then attempted to change formae specialis. He was unable to mutate a gladiolus or leek *Fusarium* to virulence on melon. But when he used fusaria from Cucurbitaceae he was able to switch a f.sp. *niveum* (watermelon) to a race 0 of f.sp. *melonis*. Moreover he found that some isolates from either cucumber or melon contained spores that attacked watermelon. Thus, these cucurbitaceous pathogens are probably all one group, having minor differences in virulence genes only some of the time.

Although interesting, what do these examples really tell us? First, the durability of resistance seems to vary from short to long. There is no rule. It must depend on both the kind of gene added to the host as well as (or possibly even more so) on the variability of the pathogen. Obviously the ability to replace the old race depends upon dissemination as well as frequency of mutation of the new race, and both differ by crop and pathogen. There has been no concern for origin or coevolution and no attempt to determine the situation in the centre of origin of the crop species. Even where the cotton wilt races have such specific geographic locations I see no

speculation as to why or what it might mean to breeding. An enigma remains, the fairly long durability of resistance and then a report of a new race in one place followed by its appearance everywhere. Why is this? What does it mean? We do not know.

It seems to me that we must be more analytical for *cubense* and bananas. We must apply both good logic and modern techniques to understanding fungal variability in order to obtain durable resistance.

Susceptibility to *Fusarium oxysporum* f.sp. *cubense*

The classical breeding scheme employed since the beginning has had the underlying assumption that resistance will be dominant and of high penetration. Also it assumes that the resistance will be simply inherited and that the male parent will be homozygous for resistance. Also it assumed that the fungal pathogen is uniform everywhere for virulence genes and that it will remain that way. Larter (1947) reported that wilt resistance was dominant and that virtual immunity was imparted to Gros Michel in the cross with a resistant diploid (see in Rowe and Richardson 1975). Vakili (1965) gives considerable data on susceptibility/resistance based on many crosses; little else is available publicly. Vakili concluded that resistance to race 1 in the edible diploid 'Lidi' is due to a single dominant gene in the heterozygous state. Also, that different accessions of *M. acuminata* subspecies *banksii* differed in susceptibility, to both race 1 and race 2. Subspecies *errans* was susceptible to race 1 but resistant to race 2. *Mycosphaerella balbisiana* was susceptible as a seedling but resistant as an adult plant. Vakili suggested that the subspecies *banksii*, from New Guinea, is not attacked in its homeland and that probably the wilt fungus is not present nor has it influenced *banksii* evolution. The subspecies *burmanica*, *malaccensis*, *microcarpa*, and *siamea*, on the other hand, are highly wilt resistant and they originate from Burma to Vietnam, Malaysia and the Soenda Islands where *Fusarium oxysporum* f. sp. *cubense* is endemic. Seedlings from the majority of the accessions from these subspecies segregated for resistance to both races.

In Jamaica, various diploids were considered to have polygenic resistance and a seedling screen of the potential parents was necessary to ensure sufficient resistance in the tetraploids (Shepherd and Lacy 1968).

Waite (1977) reports on reaction of container-grown plants of Gros Michel and Bluggoe in Honduras to inoculations of fusaria from Australia, Philippines, Malaysia, Thailand and various Latin American locations. The results were consistent with expectations based on source of the isolates, but with only two differentials, unique pathogenicity genes would not have been detected. Waite concluded that further studies to determine the pathogenic characteristics of *Fusarium* clones attacking the Cavendish group and the AAB group in various countries is obviously needed. This statement is still true now, 10 years later. In fact, the cumbersome nature of tank-grown tests, and the reluctance to introduce foreign isolates for field tests and the uncertain correlation between pot-grown tests and field results and the lack heretofor of tissue culture 'plantlets' have all combined to limit knowledge of comparative virulence and pathogenicity of the banana wilt fusaria. Improved methods with small containers and tissue-grown plantlets such as that used by Sun and Su (1984) should be utilised not just for screening for resistance but for comparative virulence and pathogenicity studies to answer the questions posed above. Probably important and mostly neglected is to standardise temperatures for such tests, an emphasis for many years in *Fusarium* work in Wisconsin (Bosland and Williams 1984).

Modern Methods for Understanding *Fusarium*

The lack of definitive genetic information on virulence and pathogenicity of the wilt fusaria, and the lack of genetic meaning to the terms race and formae specialis leave the person working with the system in a quandary as to what is worth doing.

Obviously new techniques must be applied. Some information on electrophoresis (Glynn and Reid 1969; Reddy and Stahmann 1972) and restriction fragment work applied to the wilt fusaria exists. Both should be tried anew, but they must be tried with the right isolates which will give evolutionary and practical scientific information.

An interesting new technique has been applied to obtain information on possible evolutionary relationships. The basic idea is that only closely related isolates will form heterocaryons and isolates can be grouped into 'vegetative compatibility groups' (VCGs) that represent relationships (Puhalla 1985; Correll et al. 1986). Preliminary studies indicate that different formae specialis belong to different compatibility groups and that even some races may be distinguished. This latter means that some formae specialis must contain different compatibility groups. The new technique which makes heterokaryon detection easy is the formation at high frequencies of nitrogen reductase deficient (*nit*) mutants by growing the fungi on a potassium chlorate medium. When subsequently grown on a minimal medium, thick growth occurs where heterokaryons are formed. Apparently the heterokaryons are unstable, however, so that they are not automatically useful for subsequent

pathogenicity tests to determine different virulence or pathogenicity gene effects.

Although data are still limited, it would seem this compatibility grouping should be tried for *cubense* isolates from different continents and those considered to be different races. The inference is that relatedness and descent are revealed because compatibility relies on having all of the many (6 to 10?) genes governing compatibility identical in each strain, whereas the races could differ by only one pathogenicity gene. If the pathogenic mutation has been from different parental stocks it could be revealed by the compatibility grouping.

A recent paper describes linear mitochondrial plasmid-like DNA from *Fusarium oxysporum* f.sp. *conglutinans* (Kistler and Leong 1986). All isolates of races 2 and 5 contained the same plasmid, even though they were from locations as separate as Japan, California and Germany. All race 1 isolates (a pathogen of radish, not cabbage) had a different plasmid, similar in size but nonhomologous. The suggestion was made that these elements may be carrying genes for host specificity. Even if not, it was suggested their presence could be diagnostic for determining the fungal race. (But in reality the difference here reflects a formae specialis difference, not a race difference.) The two races attacking cabbage varieties which differ by a single resistance gene were not separated.

In fact, for the *conglutinans* group all three modern methods (VCG, electrophoresis and RFLPs) gave the same differentiation of the races (Bosland, pers. comm.). Races 1 and 5 (cabbage pathogens) were separate from races 3 and 4 (stock pathogens) and race 2 was separate from the other two groups (see Armstrong and Armstrong (1966) for a discussion of *conglutinans*). Although this would seem to be a beautiful confirmation of pathotype with modern molecular characterisation techniques, a careful review of the key paper (Ramirez-Villupadua et al. 1985) gives me pause. The label race 5 (the first race to break Walker's famous type A resistance in cabbage) was given to a fungus isolated from cabbage in California from a field where no crucifer yellows was known previously. Moreover, race 1 is not known in California and thus cabbage varieties susceptible to race 1 can and are being grown. The variety which became diseased in this one field is susceptible to race 1. Thus, the 'new' race was not selected by a 'resistant' cultivar containing the A or race 1 resistance nor did it appear from a population of race 1 in wilt-affected soils. Moreover, it is not just a converted race 1 because it also was highly virulent on broccoli, cauliflower and some stock varieties not attacked by race 1, as well as being virulent to both old and new cabbage varieties. Thus, it is not just a simple race 1 change and it is a very different pathotype in pathogenic capability. To me, the presumption is strong that it is a 'new' evolutionary event and that it arose out of saprophytic or other pathogenic (than race 1) fusaria. All the molecular methods, including the mitochondrial plasmid DNA homology work, could not pick up that it is a different pathotype with a different origin.

The work on compatibility groups and on plasmids is of interest but we do not know if it will prove to be general and useful for other formae specialis. It is certainly promising enough to examine for *cubense*.

Several laboratories are just beginning to work with ribosomal RNA homologies and with DNA restriction fragment length polymorphisms for *Fusarium* (Sally Leong and Tom Gordon, pers. comm.).

Much new work is underway in many laboratories, as is revealed by the recent book 'Molecular genetics of filamentous fungi,' (Timberlake 1985), and by papers such as that of Gilchrist and Yoder (1984). It would be well for those of us interested in practical but very scientific questions to interest molecular people in our questions so the elegant new techniques can be applied to our very important wilt disease pathogen.

Molecular biologists, however, want a clean system to work with, and one with a perfect stage would be chosen as a model unless special reasons existed for not so choosing. *Fusarium oxysporum* would not be an organism of choice. To clean up terminology in relation to communication and genetic research in this area, a very recent paper should be consulted, entitled 'Genetic terminology and practice for plant pathogenic fungi' (Yoder et al. 1986).

However, there are really two basic questions to explore. One concerns the genes for pathogenic specificity, the other concerns the genetic relatedness of the pathogens. This latter point could be addressed with existing modern methods, even for the wilt pathogens. Thus, the questions posed earlier on pathogen evolution and pathogen/host coevolution could be addressed for the banana wilt system.

Nematodes

Nematodes have received much less research than the other major pathogens, in spite of their importance. Million of dollars are spent on nematode control in bananas and undoubtedly many more millions are lost through their effect in lowering yields. The reasons for this neglect are the insidious and hidden nature of the injury and the ability of *Musa* in the right moist environments to bear and continue to grow with considerable nematode damage.

Although some research on nematodes has been

carried out in connection with the Honduran breeding program, there has been little in-depth research. Most research worldwide on nematodes in relation to bananas has been on pragmatic aspects of control. Chemical control is commonly practiced by the export industries but for the low resource banana farmer and the plantain farmer, such control is too costly. Moreover, control chemicals are environmentally damaging.

Although it has long been known that nematodes were moved about in planting stock, that knowledge has not prevented their continuing dissemination. How much the indigenous nematode fauna is really responsible for the depredations and how much is due to the introduced nematode biotypes has never been addressed in spite of the great opportunity to explore this question as new isolated jungle lands have been developed into plantations over the years.

Thus, we know even less of nematode 'races' on bananas in different parts of the world than we know of the other major pathogens. Some interesting work in Central America on variability of *Radopholus similis* indicates that two banana biotypes exist there, differing in virulence (Pinochet 1979; Tarte et al. 1981). Earlier work on 'races' was published in 1971 (Edwards and Wehunt). A more general account of nematode problems of bananas by Tarte and Pinochet (1981) is very useful. This bulletin includes 43 references to nematode papers, most of which involve nematodes on *Musa*.

The most extensive published information on resistance is that of Wehunt et al. (1978), reporting on some of the work carried out in the 1960s in Honduras. In the Honduran work, 64 clones of interest to the breeding program were screened and it is clear that the clones range in resistance to *Radopholus similis* from high susceptibility to virtual immunity. The cultivar group 'Pisang Jari Buaya' (PJB) was identified as having considerable resistance in some accessions, but not in all. Some *banksii* derivatives, and others, were more susceptible than the susceptible check, 'Valery.' The results confirmed earlier observations that the Cavendish group is more susceptible than the Gros Michel group.

In more recent work the *Radopholus* resistant diploid parent 'Pisang Jari Buaya' (PJB) was found to be highly susceptible to *Pratylenchus coffeae* (Pinochet and Rowe 1978). Valery had similar root lesion indices with *P. coffeae* and *R. similis* and both Valery and PJB were good hosts for *Meloidogyne incognita*. Plantains are a preferred host for *P. coffeae* so plantains also would need resistance to this nematode.

Very little information has been published on genetics of resistance but PJB apparently carries one or more dominant genes for resistance to *Radopholus* (Pinochet and Rowe 1979). This parent, crossed with SH1734 has produced an outstanding diploid (SH3143) with resistance even higher than that of PJB (see Rowe, these Proceedings).

Most of the comments made for the fungal pathogens apply also to the nematodes. It is not known how potentially variable they will be if resistant clones are deployed in one area, or if other areas will have different 'races,' differing in virulence genes. No one has studied their variability in the centre of origin of *Musa* and no one has assayed the genetics of the many wild subspecies in terms of nematode reaction. Much needs to be learned.

Moko Disease

Moko disease (bacterial wilt of *Musa*) caused by a special race (race 2) of *Pseudomonas solanacearum*, has received little attention from the banana scientific community because of its limited distribution and its ease of control by prophylactic measures. However, it is potentially a very destructive disease. One strain is readily transmitted among inflorescences by insects and has caused major epidemics (Buddenhagen and Elsasser 1962). If this strain were introduced into the homeland of *Musa* in Asia it would probably spread quickly throughout Asia and devastate much local production. The Moko pathogen evolved in Latin America on *Heliconia* and is absent from Africa and Asia, except for Mindanao in the southern Philippines where the Moko pathogen (a non insect-transmitted strain) was inadvertently introduced in seed pieces in the 1960s, from Honduras. The bacterial wilt pathogens of other crops are ubiquitous in Africa and Asia (Buddenhagen 1964; Buddenhagen and Kelman 1964). Some seeded diploids are attacked, however, by the ubiquitous and omnivorous *Pseudomonas solanacearum* race 1, in Honduras (Buddenhagen 1962), and thus they should be attacked in the Asian homeland as well, but there are no such reports.

Little is known regarding inheritance of resistance except for one seedling resistance study by Vakili. Resistance is mediated by plant response mechanisms which limit or slow down systemicity of the pathogen within the xylem vessels (Buddenhagen, unpublished data). Such limiting of systemicity is rare in *Musa* and it is not known if the resistance levels that exist would be useful in limiting the disease under natural field conditions. No one has bred for or selected progeny for resistance to Moko disease.

A second form of resistance occurs which operates as an escape mechanism for the insect-spread strain. This consists of persistence of small hemaphroditic fruits on the peduncle rachis, instead

of the conversion to male flowers which abcise. It is the site of male flower abscissism, which has open xylem vessels and which exudes a nectar attractive to many bees and other insects, where infection occurs during insect-transmitted epidemics. These sites and others on the peduncle, bud and fruits also exude the bacterial pathogen in great quantities.

These Bluggoe type cultivars, such as Pelipita, which hold their hemaphroditic fruits/flowers, escape the disease. In any breeding program for AAB silk types or ABB Bluggoe types, selection for this escape mechanism should be practised. This is especially important in Brazil and elsewhere in Latin America, where insect-transmitted strains are prevalent and expanding in area.

A recent collection of papers on bacterial wilt in Asia has appeared (Persley 1986) which covers bacterial wilt of non-*Musa* species. One paper in this collection (Buddenhagen 1986) reviews races, genetics, and resistance and includes a discussion of Moko disease over all crops.

Breeding

Breeding has been reviewed in two quite different treatments (Rowe 1984; Stover and Buddenhagen 1986). There are three basic breeding targets in terms of the final quality product: (1) Gros Michel/ Cavendish (AAA), (2) Silk (AAB), and (3) Plantain (AAB). At present there are three breeding programs for the first target and one in Brazil for the second. For the third target a small effort has been made in Honduras but no major program exists anywhere. A fourth breeding target that should merit consideration would be for a Bluggoe (ABB) type fruit from plants resistant to both *Fusarium* wilt and Moko disease.

Everyone essentially would be starting from scratch for objectives 3 and 4. For objective 1, those who have invested years and millions in background diploid breeding (in Jamaica and Honduras) are leagues ahead of anyone beginning, and thus whether or not the advanced diploids are to be shared is a critical question for consideration by anyone thinking of beginning breeding for target 1. In fact, I think the presence of excellent bred diploids inhibits the advanced breeding programs from considering going back to any wild species or any cultivated diploids for use in breeding. This effectively cuts off a broader germplasm base. (In fact, for most crop species, this is also the case.)

For objective 2 (Silk types) Brazil should be ahead of everyone, but their lack of Black Sigatoka is a major difficulty. No one else has been very interested in investment into 'Silk' types.

The problem with too few localised breeding programs is that they easily become stultified if not connected with current good university research advances and at the same time their target horizons may be much narrower than real needs in different parts of the world. To back these points, one may ask how many doctoral theses have been produced at a good university since the United Fruit Company cut back its research in the late 1960s? Also one could cite lack of any concerted effort to utilise the good dessert tetraploids resistant to BS and *Fusarium* for local use in low resource farming anywhere. Jamaica is just starting. Also there has been a lack of any real concerted effort to produce a BS-resistant plantain, now some 16 years after BS invaded Latin America.

Above and beyond resistance considerations is a consideration of the agronomic status of the existing cultivars. If Grande Naine is the excellent and ideal ideotype already existing for the AAA target, then matching it is, indeed, difficult (Stover and Buddenhagen 1986). However, for breeding targets 2, 3, and 4, existing desired clones are agronomically quite deficient from the ideal in terms of yielding ability and stature, so matching or improving them should be relatively easy.

We have argued that the classical breeding approach of creating a tetraploid from an intact 'Highgate' genome plus a gamete from a resistant diploid should be changed (Stover and Buddenhagen 1986). Very good tetraploids have been produced which would be very useful in low resource agriculture in many parts of the world. However, the approach is a dead-end since any further attempt to use the tetraploid in breeding results in break-up of the 'Highgate' genome. After reviewing breeding for *Fusarium* resistance in other crops I am convinced that the *Fusarium* susceptibility of the 'Highgate' genome is a major danger and a more important concern than the slightly poorer crop physiological characters of known tetraploids. The same may be true for making tetraploids with the AAB Brazilian bananas.

Certainly for breeding targets 2, 3, and 4 there is no need to even start with the tetraploid approach. Rather, one should start by exploring where the centres were for natural synthesis of those triploids and utilise diploids in great numbers from those regions. They could be screened for resistance, improved themselves, and utilised for resynthesising the desired triploids.

In such a program, various advantages accrue which are common in other crop-breeding programs. Progenies can be large enough for heritability studies, tester lines can be used and breeding values estimated, inbreds can be made, heterotic values calculated, and most important, recurrent selection can be carried out. Moreover, the strong need to have absolute parthenocarpy can be backed away from, making for much greater flexibility in breeding at the diploid level.

No one has mentioned inbreeding depression in *Musa*. Vakili (1968) showed that wild accessions were heterozygous even for resistance genes but there is no mention of general effects of inbreeding. Could inbreeding be practiced to 'clean up' materials so that superior breeding value can come from the surviving inbreds, then homozygous for better alleles? Although high heterozygosity is essential in the final product (Bingham 1980), having many inbreds as parents may enhance identification of, and obtaining, superior heterotic products.

In the past, some diploids were known to undergo natural restitution at a moderate level and the idea of utilising restitution to produce triploids was thus limited to these. Secondary triploids resulting from a breakup of the 'Highgate' genome in tetraploids also was very limiting. But now with tissue culture and the production of meristems of small size the possibility of inducing tetraploids with colchicine, as was done for true seedlings (Vakili 1967) is a major opportunity. It opens great opportunities for resynthesis of new superior triploids, starting from scratch. This could be done for all the breeding targets.

The other great advantage of using only diploids in breeding is that their resistance genetics can be studied and different resistance genes can be pyramided before triploid synthesis. This will be enhanced if diploids are bred which are more fertile, a definite possibility if the final product is a triploid. Earlier, with a tetraploid target, parthenocarpy had to be intense to preclude an occasional seed in pollen fertile and potentially female fertile tetraploids. This strong parthenocarpy at the diploid level limited (and still limits) flexibility in diploid improvement.

Tissue Culture Techniques

Somaclonal variation so far obtained in banana is significant and, thus, this new methodology should be examined for breeding. No one has attempted gametoclonal work. A good overview of somaclonal and gametoclonal variation up to 1984 is provided by Evans et al. (1984).

The more recent review by Daub (1986) is directly pertinent to tissue culture in relation to resistance to pathogens and it includes references to three successes with *Fusarium oxysporum*, two of which were proven to be transmitted to progeny. It also gives a good discussion of possibilities and problems in using selective agents versus no selective pressure.

Tissue culture techniques should be applied to obtain dwarfness, possibly with the addition of mutagens. Dwarfness is an important need in breeding and it would be most useful to have dwarfness more general in diploids, to be not confined to only a few dwarf donors. The stature series of the Cavendish group should also exist with the 'Silk' types, the plantains and with 'Bluggoe.' Although a few dwarfs are known, they are not abundant and these groups need short clones as breeding goals, just as is needed for the dessert bananas. It would be most interesting to do abundant somaclonal work with the diploid parents, not only to obtain dwarfs but other variations as well. As one approaches biotechnology in relation to plant breeding one should be aware of some of the implications in relation to public versus private research support, propriety rights, patents, and the social and political impact (Hansen et al. 1986).

Cytology

The cytological work with *Musa* has been very limited and it is now 40–60 years old. Could new cytological work on parents of interest and on Cavendish be helpful in breeding? If one wishes to continue with triploid restitution breeding the logical first step would be to apply cytology and physiology to understand sporogenesis and pollen tube growth in Grande Naine in order to enable it to enter into breeding. It may well be possible to manipulate the system to get either 'n' or '3n' products in Grande Naine. No one has really tried, using modern methods. Even without a restitution approach it would certainly be of interest to have Grande Naine enter into the breeding parentage in some way. Indeed, Cavendish is in the parentage through the improbable single case of trapping a single haploid gamete (Rowe 1984). If this could be done with Grande Naine (with many haploid gametes), there could be many recombinants of interest.

Conclusion

The practical and scientific worth of a much more in-depth approach to banana and plantain breeding and genetics is very great. With the abundant talent available today wishing to work on interesting problems in breeding, genetics, molecular genetics, and pathology, great progress could be made if there were funds, a focus, good leadership and sufficient communication between the past knowledge and the present and future technology. It is amazing how neglected *Musa* has been for basic studies. It provides very important crops for people, yet, like so many tropical crops, it has been badly neglected. I think the failure to support and carry out basic work has to be laid at the lack of a strong connection of good universities in the tropics with the botany and agriculture of their locales. In addition, the corporate management of profitable production has not been wise enough to see the needs and advantage of long-term basic research and thus their support has been sporadic. Moreover, they have not linked up with universities for a better addressing of their

problems and their opportunities. And finally, the international agricultural research system has largely ignored *Musa* due to the agronomic bias of original leadership and the bias against any 'commercial' crop.

The opportunities are there. I hope this wonderful crop can now receive its just due from science.

References

Armstrong, G.M., and Armstrong, J.K. 1966. Races of *Fusarium oxysporum* f. *conglutinans*; race 4, new race; and a new host for race 1, *Lychnis chalcedonica*. Phytopathology 56, 525-30.

1978. A new race (Race 6) of the cotton-wilt *Fusarium* from Brazil. Plant Disease Reporter 62, 421-23.

Assante, G., Locci, R., Camarada, L., Merlini, L., and Nasini, G. 1977. Screening of the genus *Cercospora* for secondary metabolites. Phytochemistry 16, 243-47.

Bingham, E.T. 1980. Maximizing heterozygosity in autopolyploids. In: Lewis, W.H. ed., Polyploidy-biological relevance. New York, Plenum Press, 471-90.

Bosland, P.W., and Williams, P.H. 1984. A new pathotype of the crucifer yellows organism capable of causing disease in cool soils. Crucifrae Newsletter 9, 95.

Bouhot, D. 1981. Some aspects of the pathogenic potential in formae speciales and races of *Fusarium oxysporum* on cucurbitaceae. In: Nelson, P.E., Tousson, T.A., and Cook, R.J. ed., Fusarium: Diseases, Biology and Taxonomy. Pennsylvania State University Press, 318-26.

Brun, J. 1963. La cercosporiose du bananier en Guinee. Etude de la phase ascosporee du *Mycosphaerella musicola* Leach. Paris, Institut Francais de Recherces Fruitieres Outre Mer, 196 p.

Buddenhagen, I.W. 1964. The relation of plant-pathogenic bacteria to the soil. In: Baker, K.E., and Snyder, W.C. ed., Ecology of soil-borne plant pathogens. Berkeley, University of California Press, 269-84.

1986. Bacterial wilt revisited. In: Persley, G.J. ed. Bacterial wilt disease in Asia and the South Pacific: proceedings of an international workshop held at PCARRD, Los Baños, Philippines, 8-10 October 1985. ACIAR Proceedings No. 13, p. 126-143.

Correll, J.C., Puhalla, J.E., and Schneider, J.E. 1986. Identification of *Fusarium oxysporum* f. sp. *apii* on the basis of colony size, virulence, and vegetative compatibility. Phytopathology 76, 306-400.

Daub, M.E. 1986. Tissue culture and the selection of resistance to pathogens. Annual Review of Phytopathology 24, 159-86.

Daub, M.E., and Briggs, S.P. 1983. Changes in tobacco cell membrane composition and structure caused by cercosporin. Plant Physiology 71, 763-66.

Edwards, D.I., and Wehunt, E.J. 1971. Host range of *Radopholus similis* from banana areas of central America with indications of additional races. Plant Disease Reporter 55, 415-18.

Evans, D.A., Sharp, W.R., and Medina-Filho, H.P. 1984. Somaclonal and gametoclonal variation. American Journal of Botany 76, 759-74.

Foure, E. 1985. Les Cercosporioses du bananier et leurs traitements. Fruits 40, 393-99.

FAO. 1971. Crop loss assessment methods. Rome, FAO.

Garber, R.C., and Yoder, O.C. 1984. Mitochondrial DNA of the filamentous ascomycete *Cochliobolus heterostrophus*. Current Genetics 8, 621-25.

Gilchrist, D.G., and Yoder, O.C. 1984. Genetics of host-parasite systems: a prospectus for molecular biology. In: Kosuge, T., and Nester, E.W. ed., Plant-microbe interactions, Vol.1. New York, Macmillan Publishing Co.

Glynn, A.N., and Reid, J. 1969. Electrophoretic patterns of soluble fungal proteins and their possible use as taxonomic criteria in the genus *Fusarium*. Canadian Journal of Botany, 47, 1823-31.

Hansen, M., Busch, L., Burkhardt, J., Lacy, W.B., and Lacy, L.R. 1986. Plant breeding and biotechnology. BioScience 36, 29-39.

Jones, J.P., and Woltz, S.S. 1981. *Fusarium*-incited diseases of tomato and potato and their control. In: Nelson, P.E., Tousson, T.A., and Cook, R.J. ed., Fusarium: Diseases, Biology and Taxonomy. University Park, Pennsylvania State University Press, 157-68.

Kistler, H.C., and Leong, S.A. 1986. Linear plasmidlike DNA in the plant pathogenic fungus *Fusarium oxysporum* f. sp. *conglutinans*. Journal of Bacteriology 167, 587-93.

Kraft, J.M., Burke, D.W., and Haglund, W.A. 1981. Fusarium Diseases of Beans, Peas, and Lentils. In: Nelson, P.E., Tousson, T.A., and Cook, R.J. ed., Fusarium: Diseases, Biology and Taxonomy. University Park, Pennsylvania State University Press, 142-56.

Larter, L.N.H. 1947. Report on banana breeding. Department of Agriculture Jamaica, Bulletin 34, 24 p.

Meredith, D.S. 1970. Banana leaf spot disease (Sigatoka) caused by *Mycosphaerella musicola* Leach. Kew, Commonwealth Mycological Institute, Phyto-pathological Papers, No.11, 1-147.

Meredith, D.S., and Firman, I.K. 1970. Banana leaf spot diseases in Fiji. Tropical Agriculture (Trinidad) 47, 127-30.

Nelson, P.E., Tousson, T.A., and Cook, R.J. ed. 1981. Fusarium: Diseases, Biology and Taxonomy. University Park, Pennsylvania State University Press, 457 p.

Pearson, M.H., Bull, P.B., and Shepherd, K. 1983. Possible sources of resistance to black Sigatoka in the Papua New Guinea Biological Foundation banana collection. Tropical Pest Management 29(4), 303-8.

Persley, G.J. 1986. Bacterial wilt disease in Asia and the South Pacific. ACIAR Proceedings No. 13. Australian Centre for International Agricultural Research, Canberra.

Pinochet, J. 1979. Comparison of four isolates of *Radopholus similis* from Central America on Valery bananas. Nematropica 9, 40-43.

Pinochet, J., and Rowe, P.R. 1978. Reaction of two banana cultivars to three different nematodes. Plant Disease Reporter 62, 727-29.

1979. Progress in breeding for resistance to *Radopholus similis* on bananas. Nematropica 9, 76-78.

Puhalla, J.E. 1985. Classification of strains of *Fusarium oxysporum* on the basis of vegetative compatibility. Canadian Journal of Botany 63, 179-83.

1985. A new race of *Fusarium oxysporum* f. sp. *conglutinans* that attacks cabbage with Type A resistance. Plant Disease 69, 612–13.

Reddy, M.N., and Stahmann, M.A. 1972. Isozyme patterns of *Fusarium* species and their significance in taxonomy. Phytopathologische Zeitschrift 74, 115–25.

Rowe, P. 1984. Breeding bananas and plantains. In Janick, J. ed., Plant Breeding Reviews, Vol.2. Westport, Connecticut, AVI Publishing Co. Inc., 135–55.

Rowe, P.R., and Richardson, D.L. 1975. Breeding bananas for disease resistance, fruit quality, and yield. Bulletin 2. Tropical Agriculture Research Services (SIATSA), La Lima, Honduras, C.A.

Shepherd, K., and Lacy, D.S. 1968. Resistance of mature banana plants and seedlings to Fusarial wilt in Jamaica. London, First International Congress of Plant Pathology, Abstracts, p.181.

Smith, S.N., Ebbels, D.L., Garber, R.H., and Kappelman, Jr., A.J. 1981. Fusarium wilt of cotton. In: Nelson, P.E., Tousson, T.A., and Cook, R.J. ed. Fusarium: Diseases, Biology and Taxonomy. University Park, Pennsylvania State University Press, 457 p.

Stover, R.H. 1962. Fusarial wilt (Panama disease) of bananas and other *Musa* species. Kew, Commonwealth Mycological Institute, Phytopathological Papers No. 4, 122 p.

1963. Sexuality and heterothallism in *Mycosphaerella musicola*. Canadian Journal of Botany 41, 1531–34.

1972. Banana, plantain and abaca diseases. Kew, Commonwealth Mycological Institute.

1976. Distribution and cultural characteristics of the pathogens causing banana leaf spot. Tropical Agriculture (Trinidad) 53, 111–14.

Stover, R.H., and Buddenhagen, I.W. 1986. Banana breeding: polyploidy, disease resistance and productivity. Fruits 41, 175–91.

Su, H., Hwang, S.C., and Ko, W.H. 1986. Fusarial wilt of Cavendish bananas in Taiwan. Plant Disease 70, 814–18.

Sun, E.J., and Su, H.J. 1984. Rapid method for determining differential pathogenicity of *Fusarium oxysporum* f. sp. *cubense* using banana plantlets. Tropical Agriculture (Trinidad) 61, 7–8.

Tarte, R., and Pinochet, J. 1981. Problemas nematologicos del banano, contribuciones recientes a su conocimiento y combate. Panama, Union de Paises Exportadores de Banano. 32 p.

Tarte, R., Pinochet, J., Gabrielle, C., and Ventura, O. 1981. Differences in population increase, host preferences and frequency of morphological variants among isolates of the banana race of *Radopholus similis*. Nematropica 11, 43–52.

Timberlake, W.E. ed. 1985. Molecular genetics of filamentous fungi. New York, Alan R. Liss, Inc.

Vakili, N.G. 1965. *Fusarium* wilt resistance in seedlings and mature plants of *Musa* species. Phytopathology 55, 135–40.

1967. The experimental formation of polyploidy and its effect in the genus *Musa*. American Journal of Botany 54, 24–36.

1968. Responses of *Musa acuminata* species and edible cultivars to infection by *Mycosphaerella musicola*. Tropical Agriculture (Trinidad) 45, 13–22.

Waite, B.H. 1977. Inoculation studies and natural infection of banana varieties with races 1 and 2 of *Fusarium oxysporum* f. *cubense*. Plant Disease Reporter 61, 15–19.

Wehunt, E.J., Hutchison, D.J., and Edwards, D.I. 1978. Reaction of banana cultivars to the burrowing nematode (*Radopholus similis*). Journal of Nematology 10, 368–70.

Yoder, O.C., Valent, B., and Chumley, F. 1986. Genetic nomenclature and practice for plant pathogenic fungi. Phytopathology 76, 383–85.