New Insights into Mango Malformation Disease Epidemiology Lead to a New Integrated Management Strategy for Subtropical Environments

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Mango Malformation Disease: A Major Threat to the Production of Mango

Mango (*Mangifera indica* L.) is regarded as the king of fruits in India, where it has been cultivated for at least 4,000 years and has great cultural and religious significance (61,63). Many Indian mango cultivars originated in the fifteenth century when the best selections of mango seedlings were propagated by grafting and planted in large orchards, in some cases numbering 100,000 trees. With the arrival of voyagers to India from Europe, mango was soon established throughout the tropics and subtropics.

Mango is an important commercial crop (63) that currently ranks fifth among the major fruits cultivated worldwide (14). It has flourished in recent years due to: (i) rapid expansion into growing areas of the Old World such as China and parts of Africa; (ii) cultivation of high quality clonal selections; (iii) adoption of modern agricultural practices, including irrigation management; (iv) integrated disease and pest management; and (v) the use of pesticides and other agrochemicals (38).

Mango is cultivated commercially throughout the tropics and in many subtropical regions (45). The flowering response of mango differs greatly in tropical as opposed to subtropical environments. In the tropics, flowering can be induced chemically, while in the subtropics stimulation is ineffective and is primarily governed by chilling temperatures (28). Synchronization of vegetative growth to ensure that branch terminals are of the same physiological maturity, is a prerequisite in flowering management programs (11). A synchronized reproductive flush flowers once per year in the tropics after tip pruning and chemical treatments. In the subtropics, however, flowering occurs after 5 weeks of night temperatures below 15°C and day temperatures below 20°C (11).

Mango malformation disease (MMD) is one of the most important and destructive diseases of this crop (30,58). It affects inflorescences and vegetative portions of the plant (Fig. 1). Although

trees are not killed, the vegetative phase of the disease impedes canopy development and the floral phase reduces fruit yield dramatically; substantial economic losses can occur since malformed inflorescences do not bear fruit. In 1998, an estimated US\$15 million of fruit was lost to the disease in Egypt (58), and losses in more important producing countries, e.g., India, are undoubtedly much greater (58,59).

MMD was first reported in India in 1891 (29), and has subsequently been observed in Australia, Brazil, China, Egypt, El Salvador, India, Israel, Malaysia, Mexico, Myanmar, Nicaragua, Oman, Pakistan, Senegal, South Africa, Spain, Sri Lanka, Sudan, Swaziland, Uganda, and the United States (2,3,8,9,15,24,29,31,34,37,42,51,55,64,67,70,77). Since the pathogen is easily disseminated in infected germplasm and there are conspicuous gaps in its geographic distribution, malformation may be even more widely spread (58).

The Disease and Pathogens

Disease symptoms. MMD affects vegetative and floral meristematic tissues (6,58). Vegetative malformation is most serious on seedlings and young trees in nurseries, especially where seedlings are grown beneath the canopies of affected trees (60,75), but it also occurs on mature trees. Symptoms of vegetative malformation include hypertrophied, tightly bunched young shoots, with swollen apical and lateral buds. These buds produce misshapen terminals with shortened internodes and dwarfed leaves that curve from the tip back toward the adaxial portion of the petiole (Fig. 2A). Shoot growth is arrested and shoots arising from the same bud produce the “bunchy-top” symptom of the disease (Fig. 2B).

Floral malformation is most important economically since affected inflorescences usually do not set fruit (30,53,58,59,75). Primary or secondary axes on affected panicles are shortened, thickened, and highly branched. Malformed panicles produce up to three times the normal number of flowers, which range from one-half to two times the normal size, and have an increased proportion of male to perfect flowers that are either sterile or eventually abort. Malformed panicles may also produce dwarfed and distorted leaves (phyllody). There are various forms of malformed inflorescences, which include (Fig. 3):

(i) a compact form with thick, green fleshy panicles that resemble cauliflowers (Fig. 3A);

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(ii) a loose form with open larger-than-normal inflorescences, but with thick secondary branches (witches-broom) (Fig. 3B); and
(iii) a combination of various compact and loose forms of vegetative and floral symptoms (Fig. 3C and D). Malformed panicles, which can persist on the tree until the following season as dry, black masses, serve as sources of inoculum for as long as they remain on the tree (Fig. 3E) (22).

MMD etiology. Summanwar et al. (73) first demonstrated that a fungus in the *Gibberella fujikuroi* (Sawada) Wollenweb. species complex, identified at the time as *Fusarium moniliforme* Sheld., was responsible for the floral phase of MMD. Later, Varma et al. (74) showed that *F. moniliforme* also caused vegetative symptoms of the disease. Although the pathogen has had several synonyms in the literature, including *F. subglutinans* (Wollenweb. & Reinking) Nelson, Toussoun & Marasas, it was redescribed as a new species, *F. mangiferae* Britz, Wingfield & Marasas, in 2002 with isolates from Egypt, Florida (USA), Israel, Malaysia, and South Africa (4,42,71). *F. mangiferae* was subsequently identified in India (54,78), Oman (31), Spain (8), Sri Lanka (70), and China (77), and appears to be the most common causal agent of MMD worldwide (Fig. 4).

A second MMD causal agent, *F. sterilihyphosum* Britz, Wingfield & Marasas, was described originally for isolates from a limited area in South Africa (4). It was subsequently detected in Brazil (35), where it was shown to cause malformation after artificial

inoculation (37). Another MMD causal agent, *F. mexicanum* sp. nov., was described from Mexico (55). With multilocus sequencing, *F. mexicanum* was shown to differ significantly from other MMD taxa in the *G. fujikuroi* species complex (55,65) (Fig. 4). A fourth recently described species, *F. tupaense* sp. nov., resembles *F. sterilihyphosum* morphologically, is phylogenetically distinct from both *F. mangiferae* and *F. sterilihyphosum*, and produces a unique teleomorph in the *G. fujikuroi* complex. *F. tupaense* causes malformation in Brazil (36) and Senegal (67) (Fig. 4). An additional two species, *F. proliferatum* (Matsushima) Nirenberg (40,76) and *F. pseudocircinatum* (17), have also been reported as causal agents of MMD.

Although *F. mangiferae* predominates as the MMD pathogen in the Eastern Hemisphere, it has only been confirmed as a pathogen causing MMD in the Western Hemisphere in Florida (4,78). However, descriptions of the MMD agents in the Western Hemisphere have only been made in Florida, Brazil (36), and Mexico (55,65). Additional work is needed to determine whether *F. mangiferae* occurs elsewhere in the West.

PCR primer pairs have been used to identify some of the above taxa. Zheng and Ploetz (78) developed a pair, 1-3F/R, that amplify a 608-bp fragment for *F. mangiferae*, and have been used for diagnostic purposes (75). Another pair, 61-2F/R, developed to diagnose *F. verticillioides* (published as *F. moniliforme* in 46), failed to amplify *F. mangiferae* DNA, but when amplification protocols were modified, the pair amplified a 445-bp fragment for strains of *F. sterilihyphosum* and *F. mexicanum* (65,78). Additional primer pairs have been developed for *F. mangiferae* (50).

Does the mango bud mite play a role in the development of MMD? The mango bud mite, *Aceria mangiferae* Sayed (Eriophyidae), is often observed in high numbers on malformed trees. This association and the reported ability of the mite to cause hypertrophied buds on mango led some to consider it to be the cause of MMD (47–49). However, this hypothesis was not supported by the following evidence:

- (i) the mite was present in Australia, although rare outbreaks of MMD were reported only recently in that country (2);
- (ii) in other mango-producing areas, high populations of the mite can occur in trees that do not develop symptoms (72); and
- (iii) acaricides did not minimize MMD, although they dramatically reduced populations of the mite (41).

Nonetheless, the mite may play a role in the epidemiology of MMD. The pathogen was previously recovered from the mite on culture media (9), and was recently shown to adhere to its body (18) (Fig. 5). Gamliel-Atinsky et al. (20) reported that the mite could not ingest the pathogen, due to its small mouth, but experimentally dispersed conidia of *F. mangiferae* to infection courts

Fig. 1. Mango tree severely infected with malformation disease.

Fig. 2. Vegetative symptoms of mango malformation disease. A, Healthy (right) and diseased (left) young shoots. B, Diseased vegetative growth in orchard-cultivated tree.

within mango buds, probably as a body adherent. Wounds caused by the mites' feeding could facilitate infection of buds by the pathogen (9,18).

In Israel, *A. mangiferae* did not appear to play a significant role in disseminating the pathogen among trees. Mites were not found in traps that were designed to monitor their movement in an MMD-affected orchard, although high numbers of *F. mangiferae* conidia were trapped (19). Whether, and under what circumstances, the mite plays a role in spreading MMD among trees and orchards in other mango-production areas should be determined due to the potential impact these factors would have on MMD management strategies. Other arthropods that frequent infected panicles may serve as dispersal agents even though no conidia were detected on windborne mango bud mites originating from infected panicles (18,20). Aerial dissemination of this pathogen by arthropoda and other means should be re-examined.

Susceptibility of different mango cultivars to malformation. Wide ranges in susceptibility have been reported for different mango cultivars (29). Unfortunately, there are inconsistencies among some of these reports, and none are based on experimental evidence (58). 'Ewais', a popular cultivar in Egypt, was reported to be moderately susceptible to MMD by Bastawros (3), but of low and high susceptibility in two other references (6,59). According to

Bastawros (3), two newly introduced cultivars in Egypt, 'Kent' and 'Keitt', were immune (0% disease), even though they are susceptible to natural and artificial inoculation elsewhere (15; R. C. Ploetz, *unpublished data*). These and other anecdotal observations were made in naturally affected germplasm collections or commercial production blocks in which there was no assurance that evaluated trees were infected. Cultivars listed as "resistant" may have been established from pathogen-free nursery stock or may have escaped infection once planted in the field (58). To date, experimental evidence for the resistance or susceptibility of commercial cultivars to MMD has not been published.

Previous Approaches for Managing MMD

Diverse strategies have been tested for managing MMD, including the use of sanitation and the application of fungicides and other chemicals. However, none of these measures have provided adequate suppression of the disease.

Sanitation. Sanitation, whereby symptomatic tissues are removed from trees and destroyed, is the most common approach to MMD management once the disease is established in an orchard (32,41,48,53,68). Prompt removal of symptomatic panicles and the subtending three nodes has been recommended (41). Regardless of the extent to which sanitation is imposed, it reduces MMD, pre-

Fig. 3. Types of malformed inflorescences. **A**, a compact form whereby the peduncle becomes thick, remains green and fleshy, and branches profusely resembling that of a cauliflower in shape and size. **B**, a loose form whereby the panicle is open in shape and larger than healthy inflorescences in size while the peduncle and main secondary branches are thick resembling that of a witches'-broom. **C**, combined vegetative and floral malformation. **D**, an intermediate form that includes various malformed shapes with varying degrees of compact and loose forms. **E**, malformed panicles persisting on the tree as a dry, black mass.

sumably by reducing inoculum in an orchard. Although it is difficult to impose this treatment on large trees with panicles that are difficult to access, growers may be unwilling to devote the effort that is required to ensure that this approach succeeds. Nonetheless, we regard sanitation as an important component of any integrated strategy to manage this disease.

Chemicals. Singh et al. (69) obtained moderate control with sulfates of cobalt (Co), cadmium (Cd), and nickel (Ni) in India, but it

is unlikely that these compounds could be used safely on a food crop. Darvas (10) reduced the percentage of malformed inflorescences from 96 to 48% by injecting Keitt tree trunks with fosetyl-Al, which is used primarily against diseases caused by oomycetes. This reduction was significant ($P < 0.05$), but the increase in fruit yield, 46 to 95 kg tree⁻¹, was not.

In general, conflicting reports exist on the efficacy of fungicides for MMD management. For example, benzimidazoles have been

Fig. 4. Distribution of the different *Fusarium* species pathogens causing mango malformation disease worldwide.

Fig. 5. Association of *Fusarium mangiferae* with the mango bud mite, *Aceria mangiferae*. A to C, confocal microscope images of the mite surrounded by gfp-marked mycelium and germinating conidia of the fungus within artificially inoculated apical mango buds.

tested frequently against MMD, but their impact has been equivocal, even when positive results have been reported. Muhammad et al. (44) reported that a single spray of Topsin-M applied at the bud differentiation stage reduced malformation to 19.8%, relative to 35.4% in the nontreated control, but their data were not evaluated statistically. In contrast, Ibrahim et al. (26) indicated that benomyl inhibited the pathogen in vitro, but did not affect MMD development when sprayed on mango trees. Similarly, Chadha et al. (5) and Diekman et al. (13) observed no impact of benomyl on MMD in the field.

Combinations of measures have also been tested (7,56). Although statistical significance was not demonstrated, Iqbal et al. (27) reported that pruning the terminal 45 cm of affected shoots in combination with a benomyl treatment resulted in a 70.4% decrease in malformation incidence compared to the nontreated control. Noriega-Cantú et al. (53) reported that mango yields were significantly increased by an integrated management scheme that included removal of the terminal 80 cm of symptomatic shoots, sprays of different fungicides, and five applications of a sulfur acaricide.

Although clear consensus does not exist regarding the efficacy of fungicides for MMD management, they should still be considered as potential management tools. To that end, effective fungicides would need to be identified, as well as effective application intervals that would optimize their use. In most studies in which fungicides were used against MMD, spray applications were applied soon before or during bloom until fruit set but not thereafter (27,53). Although the underlying assumption on which this application window was based and the rationale for the timing of spray treatments was not mentioned in these studies, two possibilities exist. One might presume that buds are not infected and that fungicides would protect them from inocula that are dispersed from malformed panicles while they remain on trees. An alternative assumption, that buds are already infected and that fungicides would eliminate the pathogen therein, seems unwarranted given the absence of thoroughly effective and systemic fungicides. In general, specific fungicides have not been used routinely to manage MMD worldwide. For example, in Israel no fungicide was registered for MMD control in mango until 2013, even though the disease had been known in the country since 1975. Since then, MMD has stopped the establishment of new mango plantings in these affected areas and caused the removal of affected orchards in the south and central regions of the country.

Fig. 6. *Fusarium mangiferae* conidia were isolated at three developmental stages of malformed inflorescences: **A**, bud-break, **B**, mid-stage, and **C**, mature inflorescences.

New Insights into MMD Epidemiology and Disease Management

Inoculum availability and dissemination, and host organs that are infected by the pathogen. As outlined above, significant advances have been made in understanding the etiology of MMD. However, until recently little progress had been made on the epidemiology of this disease (21,30,58,59). The results that are discussed below are only for MMD caused by *F. mangiferae*.

In general, malformed and healthy inflorescences develop simultaneously in an orchard. Numbers of conidia that were recovered from the surface of malformed panicles increased significantly from bud break to bud maturation (Fig. 6). Only low numbers of conidia were trapped in affected orchards when panicles were young (20), but these numbers increased significantly as panicles matured to the cauliflower stage (21).

Microconidia are probably the infective propagules of *F. mangiferae*. They are the primary spores produced by the fungus, form profusely on live and dead malformed tissues (macroconidia are formed less commonly), and chlamydospores and ascospores are absent for this species (33). The rapid mortality of conidia when exposed to direct sunlight may explain the slow spread of MMD in orchards (41). Indeed, recent work showed that 100% of the conidia of *F. mangiferae* died within 2 to 4 h of exposure to sunlight (Fig. 7).

The distribution of *F. mangiferae* in affected trees suggests that apical buds are the primary sites of infection and that systemic colonization of branch tissues does not occur (21,22,57). In Florida, *F. mangiferae* was restricted almost entirely to malformed floral and vegetative tissues (57). Infection was greatest in malformed flowers and vegetative shoots, lower or nonexistent in asymptomatic tissues, and rare in branches even when they supported malformed panicles or shoots. Remnant infections of *F. mangiferae* in scaffold branches and trunks were restricted almost exclusively to dormant lateral buds (32,75). Freeman et al. (15) confirmed that apical buds of mango were primary infection sites by artificially inoculating mango with isolates of *F. mangiferae* that were transformed with the GUS reporter gene (β -glucuronidase). Although wounds may provide entry points for the pathogen, wounding was not a prerequisite for infection in that study (15).

Youssef et al. (75) reported that conidium survival declined rapidly in soil during the summer (after 10 days only 50% of the original population was recovered). However, the pathogen may survive for longer periods in soil within infected plant tissues. For example, when naturally infected panicles were buried 30 cm below the soil surface, survival was approximately 20% after 6 months (22,75).

Reports that infection of mango roots by *F. oxysporum* (29) or *F. mangiferae* (1,29) could result in the development of malformation in seedlings have not been corroborated. Although Youssef et al.

Fig. 7. Survival of conidia of *Fusarium mangiferae* after varying exposures to UV sunlight. Vertical bars represent the standard error of the mean.

(75) infected mango roots with *F. mangiferae*, these infections were not systemic and did not result in symptom development. Movement of the pathogen via seeds seems unlikely since seed infection has not been demonstrated (66,75).

F. mangiferae can be moved great distances in infected budwood and plants (62). Latently infected materials, which would not be evident to production managers or quarantine personnel, could move inconspicuously within and between countries. Surprisingly, symptomatic plants that should be easily identified are still known in international commerce (R. Campbell, *personal communication*).

In summary, the available evidence indicates that *F. mangiferae* is generally restricted to apical, aboveground meristematic tissues of its mango host and that localized, but not systemic, infections of these buds take place. Outside of these pockets of susceptibility, the pathogen is not present or survives poorly. This limited geographic distribution has significant implications for the management of MMD.

Disease cycle. A disease cycle that incorporated *A. mangiferae* was proposed by Ploetz (58), and was refined with data for *F. mangiferae* from Israel (Fig. 8; 22).

Malformed inflorescences and vegetative growth serve as sources of pathogen conidia, which reach infection sites by at least three different routes:

- (i) aerial dissemination via wind;
- (ii) via contaminated bud mites (*A. mangiferae*); or
- (iii) via infected host materials that fall into the funnel-like structure of the apical buds.

Moisture-assisted dissemination of conidia might also occur (e.g., transport in dew droplets or by rain or irrigation splash dispersal), although this is probably uncommon in arid production

areas. Conidial germination and infection can occur with at least 2 h of wetness and temperatures between 5 and 41°C, but is accelerated between 15 and 30°C and wetness >3 h (22). The presence of *A. mangiferae* in apical buds increases the frequency and severity of infection (21). After host penetration, the pathogen colonizes buds but not subtending tissue. Whether infected buds subsequently develop symptoms of MMD apparently depends on the extent to which they are colonized. It appears that a minimum threshold of infection by colonizing mycelia is required for vegetative and floral symptoms to appear; shoots that were only slightly colonized were never symptomatic (58).

Development of a new integrated management strategy.

Based on the above disease cycle, a conceptual model was created for the development of MMD in time and space (Fig. 9). The model indicates that MMD-affected tissues, developed from either natural (see below) or unnatural (e.g., use of infected scion material) infection (58), are sources of inoculum for the infection of new, pathogen-free infection sites. Microconidia, which are assumed to be the primary infective propagule of the pathogen, are produced on infected panicles and are dispersed aerially within and among orchards. Thus, inoculum dissemination and infection by the pathogen coincides with the presence of malformed tissues in the orchard. In Israel, the first malformed panicles and vegetative growth usually begin to appear in April and continue to emerge until late August. The exact dates fluctuate slightly from year to year depending on weather conditions and flowering dates for different cultivars. Conidia are deposited on dormant buds on the same tree or different trees in the same or other orchards. Occasionally, new plantations are located adjacent to heavily infected orchards, and the former serve as a source of inoculum for the newly planted trees (Fig. 10; 21).

Fig. 8. Cycle of mango malformation disease caused by the pathogen *Fusarium mangiferae* (*A. mangiferae* = *Aceria mangiferae*). Modified from Gamliel-Atinsky et al. (22).

Fig. 9. Conceptual model of mango malformation disease epidemiology over a 3-year season in Israel. I = January to March; II = April to June; III = July to September; and IV = October to December.

Fig. 10. Regional spread of mango malformation disease (MMD). The mango orchard on the left was planted in 1995; it is severely infected with MMD and serves as the source of inoculum for the mango orchard on the right which was planted in 2006.

The practical implication of the model is that the window of infection is also the window of protection during which fungicides or other protectants should be applied to manage the disease. The window of protection will vary in different areas, depending on the corresponding environmental and production conditions and when windows of infection occur, but the need to protect trees during the later periods is a constant. Another component of the management strategy is the reduction of primary inoculum. To do so, malformed tissues are removed as soon as they appear, until flowering stops (identified hereafter as “strict sanitation”). Infected buds can remain dormant for several months, after which they may differentiate into malformed or nonmalformed inflorescences, or vegetative

shoots. A portion of the buds in mature sections of the tree may also remain dormant for extended periods of time, and should severe pruning be conducted, these latently infected buds located in mature parts of the tree, such as the trunks, may develop later into malformed tissues (Fig. 9).

Evaluation of a new integrated management strategy. A prerequisite for implementing a new integrated management strategy was the identification of fungicides that could be used to protect buds from conidia of *F. mangiferae*. Thus, fungicide efficacy was evaluated in in vitro tests, in which prochloraz most effectively inhibited *F. mangiferae* (16). The ED_{50} value (i.e., the concentration inhibiting 50% of the pathogen’s radial growth) for prochloraz-Zn was 0.01 $\mu\text{g a.i./ml}$, for carbendazim was 0.7 $\mu\text{g a.i./ml}$, for pyraclostrobin and boscalid was 1.5 $\mu\text{g a.i./ml}$, and for famoxadone and mancozeb, and for azoxystrobin was 10 $\mu\text{g a.i./ml}$. Additional fungicides that did not inhibit in vitro fungal growth of the pathogen compared to those listed above included bupirimate, flutolanil, mancozeb, tebuconazole, thiophanate methyl, triadimenol, and triforine (16).

Next, the impact of prochloraz on bud infection was evaluated. In greenhouse trials, the fungicide reduced infection when applied at a concentration as low as 0.1 mg/ml. In another set of experiments, it was found that infection was reduced by 90% when the fungicide was applied up to 14 days before inoculation (protective activity) and up to 14 days following inoculation (curative activity), theoretically spanning a 4-week period (Fig. 11).

Based on the above findings, a set of six experiments was initiated in 2005 in naturally infected commercial orchards in Israel. In these experiments, it was shown that timely application(s) of prochloraz was required to suppress MMD (Table 1; 16). When prochloraz was applied during an inappropriate timing event, either preflowering when inocula were not present in the orchard or postflowering, after inocula had already reached unprotected buds, MMD incidence was not reduced (16). However, when prochloraz sprays coincided with the production of inocula on infected panicles, MMD incidence declined significantly in treated compared to control plots (Table 1).

As indicated above, prompt removal of primary inocula via strict sanitation is a vital component of the new integrated MMD management strategy. In another experiment, it was shown that over a 3-year period, a significant yield increase (39.3%) was attained when strict sanitation was combined with prochloraz treatment, compared with the control unsprayed treatment, in which symptomatic tissue was removed only at harvest. Strict sanitation or application of the fungicide alone resulted in insignificant increases in yields (17.8 and 14.4%, respectively). Thus, timely sprays reduced MMD incidence and, when combined with strict sanitation, increased yield. Based on these studies, strict sanitation, combined with prochloraz sprays during the window of infection (the window of protection), is currently recommended in Israel. Prochloraz was registered for emergency use to manage MMD in Israel in 2013. Long-term implementation of this new integrated management strategy is expected to provide cumulative reductions in disease annually, eventually achieving negligible levels of malformation in treated orchards over time.

Treatment with prochloraz may serve an additional purpose, as it also reduces powdery mildew of mango blossoms (39). Thus, both diseases could be managed as they occur at the same phenological stage of panicle development. Alternatives for prochloraz should be

sought, as *F. mangiferae* could develop resistance to this fungicide (23,25,43). Reduced sensitivity and field resistance to prochloraz has been reported for other fungal pathogens (23,25,43). Thus, prochloraz alternatives would be needed if it is introduced on a large scale for management of MMD.

Comparison of the new integrated strategy with MMD management practices worldwide. MMD management practices vary in different mango-production countries. In South Africa, the Agricultural Research Council (ARC) recommends removing the terminal three nodes of affected terminals and disposal in plastic bags which are then burned (12). The ARC publication states that if sanitation “is done every year the disease soon assumes insignificant proportions.” No chemical treatments are mentioned by the ARC. Nevertheless, disease proportions have recently reached epidemic levels in that country (S. Freeman, *personal communication*), indicating that sanitation alone may need to be integrated with alternative measures as a successful management strategy.

In Mexico, MMD is managed with copper sulfate pentahydrate and tribasic copper sulfate, which are applied after the terminal 80 to 100 cm of malformed shoots are removed during the vegetative stage, or before spraying potassium nitrate to promote flowering (52). However, the numbers of applications that are required for effective management of MMD in Mexico are not mentioned. Thus, a combination of sanitation and sprays is suggested for MMD management in Mexico, but the timing and frequency of these treatments are not specified.

In Brazil, there are no published MMD management protocols. However, commonly used measures include: (i) removal of lateral twigs or inflorescences with symptoms of malformation, 50 cm beyond infected panicles, and burning; (ii) general inspection of trees after harvest, aimed at elimination of all material with symptoms after the production cycle; (iii) sanitation of tools used during pruning by immersion in hypochlorite solution; (iv) treatment of pruned parts of the plants with a copper paste; and (v) application of registered fungicides and acaricides during the period of dormancy (Moacir Brito Oliveira, *personal communication*).

In South Africa, Mexico, and Brazil, the extent of the recommended sanitation measures has not been explained. Other than the report of Noriega-Cantú et al. (53), there are no data that demonstrate the efficacy of these treatments. Statistically significant support could facilitate the adoption of these recommendations by producers which, in turn, would reduce the impact of this important disease.

It is expected that long-term implementation of the new management strategy will result in cumulative disease reductions. Presumably, the components of the new integrated management strategy presented above, that is, sanitation and application of

Fig. 11. Protective and curative properties of prochloraz-Zn against *Fusarium mangiferae*. Fungicide was applied at a rate of 1 mg/ml in two sets of experiments. In the first set, the protective property of the fungicide was evaluated and sprays were applied up to 28 days prior to inoculation (red line). In the second set, the curative property of the fungicide was evaluated and sprays were applied up to 28 days after inoculation (blue line). Inoculation date is indicated by dashed gray line, modified from Freeman et al. (16).

Table 1. The efficacy of prochloraz sprays in the suppression of MMD in six field experiments carried out in Israel in the years 2006 to 2013^u

Exp No.	Year ^v	Cultivar	Disease intensity in untreated plots ^w	Treatment ^x	Control efficacy (%) in treated plots ^y
1	2006	Tommy	23.3 (I)	Untimely prochloraz-Mn sprays	–6.4 to 30.5
2	2006	Keitt	16.7 (I)	Untimely prochloraz-Mn sprays	28.7 to 35.3
3	2007, 2008, 2009	Tommy	17.4, 26.3, 34.7 (I)	Timely prochloraz-Mn sprays	44.3*, 26.6*, 34.3*
4	2007, 2008, 2009	Keitt	16.0, 24.6, 24.6 (I)	Timely prochloraz-Mn sprays	18.8, 28.0*, 28.0*
5 ^z	2009, 2011	Maya	9.6, 16.5 (I)	Timely prochloraz-Zn sprays	57.7*, 80.0*
6 ^z	2012, 2013	Keitt	37.5, 4.5 (S)	Timely prochloraz-Mn sprays	47.6, 48.9*

^u Results published by Freeman et al. (16).

^v Assessments were performed in the years that follow the application of fungicides in the orchards. For example, in experiments no. 1 and 2 the sprays were initially applied in 2005.

^w MMD incidence (I): the percentage of malformed panicles out of the total number of panicles that developed on the sampled trees; MMD severity (S): the number of malformed panicles that developed on the sampled trees.

^x Untimely sprays: prochloraz sprays (6 to 12 in each experiment) were applied at periods that eventually were found to be beyond the window of protection; Timely sprays: prochloraz sprays (6 to 8 in each experiment) that were applied during the window of protection.

^y Control efficacy in treated plots (CE_t ; in %) was calculated for each experiment, in each year, using the formula: $CE_t = 100 - BC_t / BC_u \times 100$; where BC_t and BC_u are disease incidence or severity in the fungicide and the control treatments, respectively. Numbers followed by an asterisk denote a significant difference from untreated plots, as determined by the Tukey-Kramer HSD test at $P = 0.05$.

^z Strict sanitation, removal of malformed tissues as soon as they appear until termination of flowering, was performed in these experiments.

fungicides, were included in management practices described above from other countries. Nevertheless, the underlying rational concepts and the timing of implementation in the new integrated management strategy differ from the other strategies. Sanitation in the new integrated management strategy is aimed at removing the infected plant organs producing inocula (conidia) that infect healthy buds **externally**. Thus, sanitation has to be initiated as soon as malformed tissues are observed and continue as long as they are present. In most of the other countries, sanitation is aimed at removing the systemically infected branches bearing infected tissues, assuming that these branches are colonized and produce inocula (mycelia) that infect healthy buds **internally**. Thus, under this assumption, sanitation can be performed at any time, preferably after all malformed tissues are detected. Fungicide spraying in the new integrated management strategy is aimed at protecting the susceptible host tissues (buds) from infections by airborne conidia. Thus, spraying to protect noninfected buds from inoculum produced from malformed panicles has to be initiated as soon as they are observed, and until symptomatic tissues are no longer produced. In some of the other countries, spraying was recommended at other times and the rational for that timing was not explicitly defined.

Summary and Future Studies

Progress in several different areas of research may depend on identifying which fungi cause MMD in different regions. The ability to identify different MMD agents would be needed to develop reliable disease diagnostics, as well as understand the diseases' etiology and epidemiology in different environments and improve MMD management strategies.

The growing list of *Fusarium* spp. that cause this disease indicates that there is much to learn about MMD. How MMD pathogens other than *F. mangiferae* behave on mango is not known. For example, whether other species affect different mango cultivars to the same extent as *F. mangiferae* should be determined as it would inform management recommendations for MMD. For the same reason, it should be determined whether the other species interact with the mango bud mite, *Aceria mangiferae*, as does *F. mangiferae*. And why/how several distinct species cause the same symptoms on this host should be examined, as it could reveal much about the host/pathogen interaction and physiology of the disease.

Infection by *F. mangiferae* is not systemic (20,58,75), and the most susceptible organ of infection has been shown to be the apical meristem (22). These critical data have helped develop the new integrated strategy to manage MMD, but the timing of fungicide applications has not yet been optimized. Additional data that are required for optimization of the spray regime (within the window of protection) should address weather conditions that impact dissemination and germination of conidia and their infection of the host. In the tropics, where flowering is erratic and often synchronized chemically, the new integrated MMD management could be adapted.

In Israel, implementation of strict sanitation, by continuously removing infected panicles, is aimed at reducing the primary inoculum in the orchard for minimizing the occurrence of new infections. Although malformed panicles are burned in some areas, this may not be possible where the practice is strictly regulated due to the fire hazard and concerns about air pollution. Thus, alternative treatments, such as solarization, should be considered. In addition, since latent infection of budwood is common, a treatment that would eliminate the pathogen from budwood would be invaluable. In this regard, prochloraz may be useful, although additional fungicides should be examined given the potential for resistance to this fungicide to develop.

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Dr. Ploetz joined the faculty at the Tropical Research and Education Center in Homestead in 1986. He is an authority on diseases of tropical fruit crops, and has written and edited numerous books, book chapters, and other publications on these topics. He has worked in more than 60 countries on these problems, speaking at conferences, advising students, and interacting and consulting on research projects. He has worked on mango malformation for more than 20 years, and began international work on the problem with a USAID-funded project in Egypt in the mid-1990s. A proposal he wrote at that time with Egyptian cooperators morphed into long-term USAID funding that Dr. Freeman has received on this disease.

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