Epidemiology of Mango Malformation in Guerrero, México, with Traditional and Integrated Management

D. H. Noriega-Cantú, Inst. Nal. Inv. Agrícola, Pecuaria y Forestal, Campo Exptal. Iguala, a.p. # 5 Iguala, México; **D. Téliz** and **G. Mora-Aguilera**, Professors of Inst. Fitosanidad; **J. Rodríguez-Alcazar**, Professor of Inst. Recursos Genéticos, Colegio de Postgraduados, 56230 Montecillos, México; **E. Zavaleta-Mejía** and **G. Otero-Colinas**, Professors of Inst. Fitosanidad; and **C. Lee Campbell**, Professor, Department of Plant Pathology, North Carolina State University, Raleigh 27695-7616

ABSTRACT

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The temporal progress of malformation (MM) of mango (*Mangifera indica*) was studied from 1993 to 1995 with three management technologies applied to commercial plantations in North Guerrero, Mexico. Management influenced shoot production and thus determined the dynamics of epidemics. Environmental factors also affected disease incidence, particularly through an apparent effect on inoculum dispersal. In general, integrated management (IM), consisting of pruning, acaricide, and fungicide sprays, resulted in slower rates of epidemic development, lower levels of initial and final disease, and lesser areas under the disease progress curves. In the first cycle, IM increased yield per tree by 51% in relation to high technology (HT) and 74% in relation to lower traditional technology (LT), representing a benefit-cost rate of 2.8 and 3.3, respectively. Change of malformation incidence was correlated positively with the number of macroconidia of *Fusarium* sp. trapped in the canopy ($r = 0.90$, $P = 0.0001$) and wind speed ($r = 0.83$, $P = 0.0001$); both variables lagged over a 4-month period. The greatest change in malformation occurred during the main vegetative flush, which occurred 3 to 6 months after picking the fruit (May). The accumulated proportion of diseased shoots was correlated with the following variables measured over a 1-week period: average maximum daily temperature ($r = -0.68$, $P = 0.01$), average temperature per hour ($r = -0.59$, $P = 0.04$), average number of hours with relative humidity $\geq 60\%$ ($r = -0.82$, $P = 0.001$), and wind speed ($r = 0.94$, $P = 0.0001$). In general, the greatest spore density was found during the rainy season, with a morning periodicity showing the highest correlation with wind speed ($r = 0.812$, $P = 0.0001$). *F. subglutinans* was isolated consistently from diseased (86%) and asymptomatic (5%) vegetative and flowering shoots.

Malformation is an important disease of mango (*Mangifera indica*). It has been reported from the Indian subcontinent, Africa, Southeast Asia, and the Middle East, and in certain areas of North, Central, and South America (7,8,11,14,22). Malformation was found for the first time in Mexico in 1958 in mango plantations of the states of Morelos, Guerrero, and Veracruz (14). Currently, the disease has spread to most states where mangos are grown and causes the most severe damage in the states of Guerrero and Michoacan (17,35).

Symptoms of the disease include loss of the apical dominance and swelling of vegetative buds, proliferation of leaves and flowers, phyllody and hypertrophy of panicle axes. Lack of fruit set or extensive

abortion of fruit is observed in severely diseased panicles (11,22). The vegetative deformation may also affect immature trees and nursery stock, which can lead to the spread of infected plants. According to mango producers in Mexico, yield reduction in some regions of the tropical, dry land growing region may be up to 30 to 40%.

The disease has been associated with physiologic disorders and hormonal imbalances (28,29) and attacks of an eriophyid mite, *Aceria (Eriophyes) mangifera* (Sayed) (7). However, Koch's postulates have only been completed for *Fusarium subglutinans* (3,4,10,19,23,32) and *F. oxysporum* (1,4,6) as the causal agents of malformation. Yet some controversy remains regarding species identification (22) and the inoculation methods used (11).

Attempts to control malformation have met with little success. Pruning of infected buds, symptomatic tissues, and several subtending nodes (5,11,21) and applications of systemic fungicides like benomyl (27) as well as insecticides-acaricides such as monocrotophos, sulfur, and gusathion have shown some efficacy (5,7,17,30). The

combination of some of these measures has provided better control of malformation (5,21) than individual measures alone.

Epidemiological studies on the malformation of mango are limited; however, temperature apparently has a key role in disease development. In India, the disease is present in all mango-producing areas (34), with a lower incidence in the southern and eastern than in the northern region. Temperatures in those regions are warmer than in the north, where cold conditions precede flowering. Earlier emerging floral buds are the most severely damaged, whereas later ones escape the disease (11). Escape was attributed to the occurrence of relatively high temperature during panicle development. Coincidentally, a study of seasonal variation of the population density of *F. moniliforme* on mango shoots indicated that spore density reached a maximum in February, when temperature ranged from 8 to 27°C and the humidity was 85%, and that a decline of spore density coincided with hot, dry conditions (11).

Because neither data on the temporal characterization of malformation epidemics nor information on the biotic and abiotic factors involved in the progress of epidemics are available, this study was performed with the objectives of characterizing the temporal progress of malformation in mango under three agricultural management systems. We also examined the relationship of spore density of *F. subglutinans* and/or *F. oxysporum* and climatic variables to changes in disease incidence.

MATERIALS AND METHODS

Experimental orchard. The experiment was conducted during the 1993-94 and 1994-95 growing cycles in a 10-year-old commercial orchard of the mango cv. Haden in the state of Guerrero. The soil was a clayey-sand, lightly compacted, with 1% organic matter and pH 7.9. Trees averaged 5 m high, with a mean trunk diameter of 0.9 m and a spacing of 10 m between tree rows. The trees had an average of 50 floral and vegetative deformations at the beginning of the experiment. A randomized block design was used for the three treatments: (i) high technology (HT), (ii) low traditional technology (LT), and (iii) integrated management (IM), with five blocks

Corresponding author: D. Téliz
E-mail: dteliz@colpos.colpos.mx

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(double-tree plots) per treatment. The experimental unit was a tree, and the same trees were used in both cycles.

The HT treatment included removal of symptomatic shoots from the previous cycle (30 cm below the lowest diseased shoot in a branch); nine fungicide sprays (copper oxychloride at 2.6 g a.i./liter [C], benomyl at 0.25 g a.i./liter [B], and mancozeb at 4 g a.i./liter [M]) in the sequence C(6)-B(2)-M applied singly in succession at fortnightly intervals from before flowering until fruit set; control of ants (Hymenoptera: Formicidae) (methyl parathion at 8 g a.i./ha); and addition of chicken manure (2.5 kg/tree once a year). The LT consisted of sanitary pruning similar to the previous treatment and two alternated fungicide sprays (benomyl at 0.25 g a.i./liter and mancozeb at 4 g a.i./liter) applied at fortnightly intervals during panicle development. The IM consisted of removing of diseased shoots (80 cm below the lowest diseased shoot); four

fungicide sprays (copper oxychloride at 2.6 g a.i./liter) applied at monthly intervals during the vegetative period and three sprays (captan at 1.5 g a.i./liter, benomyl at 0.25 g a.i./liter, and mancozeb at 4 g a.i./liter) in succession applied at fortnightly intervals from before flowering until fruit set; five applications of an acaricide (sulfur 3.6 g a.i./liter) applied at monthly intervals during the vegetative period; and control of ants and addition of chicken manure as before. For both, HT and IM, 3% potassium nitrate (KNO_3) (18) was applied in water to the whole canopy to promote uniformity in flowering. Fruit picking, done in April and May, and standard cultural practices such as irrigation, fertilization, one general insecticide spray (malathion at 1.5 ml a.i./liter), and weed elimination were the same for all treatments.

Disease assessment. For the purpose of disease assessment, 5 of 10 trees per treatment were selected in 1993-94. In 1994-95,

one additional tree was evaluated. Four branches at the four cardinal points were labeled per tree canopy. During each assessment, the total number of healthy and diseased shoots (vegetative and floral) was counted on each branch and averaged over the four branches per tree. The disease progress was determined as the accumulated proportion of diseased shoots per tree (Y_{ic}) corrected for host growth. At each time, i , Y_{ic} was calculated as: $Y_{ic} = Y_i/N$, in which, Y_i is the accumulated number of diseased shoots at time i ; N is the total number of shoots produced in the growing cycle. Evaluations were carried out monthly in the vegetative stage (June to December) of mango growth and weekly during panicle development (January to February).

Temporal analyses. Disease progress data were corrected initially for maximum disease incidence (Y_{max}) (2.15), with arbitrary values of 0.1, 0.5, and 0.7. Only one Y_{max} value, 0.7, was selected to correct all epidemic curves for purposes of comparison. The apparent infection rate (r) was estimated with the slope parameter of the linearized forms of the monomolecular, Gompertz, and logistic models fitted with the least square method of the GLM procedure of SAS (Release 6.03, SAS Institute, Cary, NC). The best model was selected by examining the proportion of variance explained (r^2) and by plotting standardized residual versus predicted values (2). After the best model for each epidemic was selected, the values of the slope parameter from the various models were transformed onto a standardized scale (r_s) through the use of the weighted mean absolute rate of disease incidence as the p -parameter of the Richard's model (25) for the overall linear model that was selected most frequently.

The epidemics also were characterized with the Weibull distribution function modified as a two-parameter model (20,26,33). The b parameter is related inversely to apparent infection rate (r), and the c parameter is related to the shape and slope of the density function (dy/dt per unit) for disease progress curve. The estimation of b and c was done empirically by means of the interactive process of nonlinear regression with the DUD method (SAS, Release 6.03, SAS Institute).

Epidemics were also characterized by the area under the disease progress curve (AUDPC) of malformation estimated with the trapezoidal integration method. AUDPC was standardized for differential epidemic duration as $AUDPC_s = AUDPC/T_i$ in the 1994-95 growth cycle, where T_i was length of duration of the epidemic (2). Other curve parameters included initial (Y_0) and final disease incidence (Y_f).

Effect of treatments. Analysis of variance was performed on the r , Y_0 , c , b^{-1} , Y_f , AUDPC, $AUDPC_s$, and yield of mango for each growing cycle. The Student-Newman-

Fig. 1. Disease progress curves of malformation of mango cv. Haden in North Guerrero, México, during 1993-94 and 1994-95. HT = treatment with high technology; LT = treatment with low traditional technology; and IM = integrated management. 1Jan to 4Jan = first to fourth week of January.

Keuls (SNK) was used to separate treatment means, provided that the F value of the ANOVA was significant (31).

Climatic data. Wind speed, air temperature, and relative humidity were measured at the canopy level using an anemometer and hygrothermograph (both by Weather Measure Corporation, Sacramento, CA), respectively. Measurements were recorded daily during 1 week per month during the vegetative stage and daily during panicle development.

Fungus isolation and trapping of spores. Fungus isolations were made from 15 malformed shoots and 10 asymptomatic shoots from the experimental orchard. These shoots were kept in plastic bags at 10°C until used, which usually was within 48 h after shoot removal. Five to seven pieces (4 to 6 mm) of tissue were taken from each of the malformed and asymptomatic shoots. The tissue was surface-disinfested with sodium hypochlorite (0.5%) for 5 min, rinsed three times in sterile distilled water, and dried with paper toweling. The tissue was aseptically transferred to 3.9% potato dextrose agar (PDA) (Difco Laboratories, Detroit, MI) in petri dishes and placed under natural illumination at room temperature. Single-conidium transfers were made to PDA from developing *Fusarium* colonies for identification purposes (16).

A volumetric spore trap with a 7-day record (9) was used to estimate conidial abundance in the air. The spore trap was placed at 60 m from the east side of the orchard border and 15 m to the south of the experimental plots, at 2-m height in the canopy. A clear tape on which spores were deposited was cut into 39.5-mm sections corresponding to each 24-h period and mounted on microscope slides. Slides were examined at $\times 600$ magnification in three transects across each slide for counting of spores. The mean number observed was calculated. If fewer than five macroconidia of *Fusarium* spp. were observed, an additional three transects were counted and the mean was calculated. Traps were operated daily in 1 week per month during the vegetative stage and daily during flowering.

Correlative studies. The variables number of hours with relative humidity (RH) higher or equal to 60%, number of hours with $RH \leq 40\%$, maximum (T_{max}), and minimum (T_{min}) daily temperature, average temperature per hour (T_h), wind speed (m/s), and numbers of spores trapped were regressed against the change of disease incidence. Variables were recorded during June to January in 1993-94 and June to February in 1994-95 growing cycle.

RESULTS

Disease assessment. The malformation disease progress curves in 1993-94 and 1994-95 varied greatly in shape among treatments, particularly in the first growth

cycle; the final disease incidence (Y_f) was also greater in 1993-94 (Fig. 1). The first visible symptoms on the vegetative shoots (Y_o) and the greatest incremental increase of disease incidence occurred after fruit picking in October and November (Fig. 2). A second, small incremental change occurred during full-bloom in the first (1993-94) and last (1994-95) 2 weeks of January. A third peak in disease change on vegetative shoots was observed in March only in the 1994-95 cycle. Correction of disease incidence for host growth was needed in 12 of 14 and in 16 of 16 disease progress curves in first and second growth cycles, respectively.

Temporal analyses. Ten out of 14 epidemics of 1993-94 were best described by the Gompertz model, three by the monomolecular, and one by the logistic. Overall, six epidemics were fitted by the best model with an $r^2 \geq 0.90$; the remaining epidemics had an $r^2 \geq 0.80$ (Table 1). For comparison purposes, estimates of the rate of disease

progress for epidemics described by the monomolecular and logistic models were transformed with the Richard's procedure (25) to provide values of the rate parameters equivalent to those for the Gompertz model.

All 16 epidemics of 1994-95 were best described by the monomolecular model. Nine epidemics were fitted with an $r^2 \geq 0.73$, and seven had an $r^2 \geq 0.90$ (Table 1). The r_m was used for comparative purposes. Two IM repetitions that remained healthy were included with an r_m of zero.

The Weibull model adequately described all the epidemics in both growing cycles (Table 1). In 1993-94, 10 out of 14 epidemics were fitted with $r^2 \geq 0.90$. The remaining epidemics had r^2 values of 0.83 to 0.86. These epidemics in general had the lowest Y_o (0.021 to 0.030) and Y_f (0.051 to 0.247) values. In 1994-95, 10 out of 16 epidemics were fitted with $r^2 \geq 0.90$. The remaining epidemics had r^2 values of 0.84 to 0.89. These epidemics had also the low-

Fig. 2. Change of disease incidence of malformation of mango (MM) in vegetative and floral shoots of mango cv. Haden, number of macroconidia of *Fusarium* spp., maximum temperature (T_{max}) and average temperature per hour (T_h), number of hours with relative humidity (RH) higher than or equal to 60%, and wind speed. 1Jan to 4Jan = first to fourth week of January. North Guerrero, México, during 1993-94 and 1994-95.

est Y_o (0.010 to 0.069) and Y_f (0.026 to 0.155) values. The c and b parameter estimates were negatively correlated in both growth cycles ($r > -0.89$, $P = 0.05$).

Effect of treatments. In the 1993-94 cycle, the HT treatment had the highest values of r_s , Y_o , b^{-1} , Y_f , and AUDPC ($P = 0.05$) followed by IM and LT. IM and LT were not significantly different with respect to r_s and Y_o but differed in values of b^{-1} , Y_f , and AUDPC ($P = 0.05$) (Table 2). Values of the c parameter were not significantly different among treatments. The highest average yield (97 kg/tree) was recorded in IM ($P = 0.05$) (Table 2). This

yield was 74 and 51% more than that obtained with the LT and HT treatments, respectively (Table 3). The significance of these yield differences was evident in the financial analysis (Table 3).

In the 1994-95 cycle, the disease incidence was generally lower than in the previous cycle (Figs. 1 and 2). HT had the greatest values of r_m , Y_f , AUDPC, and AUDPC_s followed by LT and IM. Values for parameters associated with HT were statistically different from those of LT and IM only for AUDPC and AUDPC_s ($P = 0.05$). LT and IM were different with respect to values for r_m , Y_o , and AUDPC_s (P

$= 0.05$). Yield was lower in this cycle (6 to 14 kg/tree) than in 1993-94, and no statistical differences were found among treatments (Tables 2 and 3).

Fungus isolation. *Fusarium* sp. was isolated consistently from diseased (86%) and asymptomatic (5%) vegetative and flowering mango shoots. These isolates were identified as *F. subglutinans* (Jean Juba, Fusarium Research Center, Penn State University). Other fungal genera (*Pestalotia*, *Lasiodiplodia* [= *Botryodiplodia*], and *Aspergillus*) were also recovered at lower frequencies.

Correlative studies. The correlation among change in malformation incidence in treatment HT, number of macroconidia, and climatic factors (Fig. 2) were examined using a Pearson's correlation matrix (Table 4). Change in incidence of malformation was correlated with values for the number of macroconidia of *Fusarium* spp. ($r = 0.90$, $P = 0.0001$) and the wind speed ($r = 0.83$, $P = 0.0001$) obtained 4 months prior to the specific observation of disease incidence (=lag 4 months).

Conidia were trapped most frequently during July in the cycles 1993-94 and 1994-95. Another peak in conidial number was reached in November (1993-94) and October (1994-95). A third peak, found only in 1994-95, was recorded in February. The number of conidia was correlated positively with wind ($r = 0.812$, $P = 0.0001$) (Table 4). The largest number of conidia was caught on the average between

Table 1. Summary of the analysis of regression used to evaluate four models for the progress of malformation of mango, cycles 1993-94 and 1994-95

Model	Epidemics ^w	r^2	MSE ^x	Growth rate ^y
Cycle 1993-94				
Monomolecular	3	0.82-0.94	0.0001-0.0002	0.003-0.004
Logistic	1	0.91	0.1865	0.151
Gompertz	10	0.80-0.94	0.013-0.182	0.035-0.185
Weibull	14	0.83-0.95	0.00002-0.007	1/22-1/4,052
Cycle 1994-95 ^z				
Monomolecular	16	0.73-0.94	0.0001-0.0023	0.003-0.013
Logistic	0	...	...	...
Gompertz	0	...	...	...
Weibull	16	0.78-0.96	0.00001-0.0004	1/67.2-1/1,382.2

^w Number of epidemics best described by a particular model of 14 and 16 epidemics analyzed for cycles 1993-94 and 1994-95, respectively.

^x Mean square error of the estimate of the rate of apparent infection and r^2 = coefficient of determination.

^y Growth rate of apparent infection estimated as the slope of the monomolecular, logistic, and Gompertz linearized model forms and with the inverse of the Weibull scale parameter (b).

^z In this cycle, two epidemics in the integrated management treatment did not develop.

Table 2. Effect of treatments on parameters^y of the curve of progress of the "malformation" and yield of the mango, cv. Haden, in the state of Guerrero for growth cycles in 1993-94 and 1994-95

Treatments ^z	r	Y_o	c	b^{-1}	Y_f	AUDPC	AUDPC _s	Yield kg/tree
Cycle 1993-94								
HT	0.136 a	0.058 a	1.480 a	0.036 a	0.538 a	7.595 a	...	47 b
LT	0.020 b	0.026 b	1.300 a	0.005 c	0.070 c	0.904 c	...	25 b
IM	0.069 b	0.029 b	2.348 a	0.022 b	0.269 b	2.922 b	...	97 a
Cycle 1994-95								
HT	0.0093 a	0.039 a	...	...	0.161 a	3.306 a	0.129 a	14 a
LT	0.0082 a	0.041 a	...	...	0.105 ab	1.449 b	0.073 b	6 a
IM	0.0033 b	0.013 b	...	...	0.051 b	0.797 b	0.030 c	10 a

^y r is the apparent infection rate (per unit week⁻¹) standardized by Richard's method to the Gompertz model (1993-94) and estimated directly with the monomolecular model (1994-95) (estimated by the slope of the line fitted to each epidemic); c and b are, respectively, the curve-shape and scale parameters estimated by the Weibull model; Y_o and Y_f are the initial and final disease incidences; AUDPC = area under disease progress curve (proportion-week), and AUDPC_s = the AUDPC standardized by dividing AUDPC by time total duration of an epidemic in week.

^z Treatments: HT = high technology, LT = low traditional technology, and IM = integrated management. Multiple comparison of means by Student-Newman-Keuls test ($P = 0.05$).

Table 3. Financial analysis of production of a 10-year orchard of mango cv. Haden under three technological management systems in Guerrero, México

Treatments ^x	Trees/ha	Fruit kg/ha	Fruit value (\$) ^y	Cost/ha (\$)	Net benefit/ha	B/C	B/C total ^z
Cycle 1993-94							
HT	100	4,700	2,087	863	1,226	1.4	0.72
LT	100	2,500	1,110	591	520	0.9	0.27
IM	100	9,700	4,307	827	3,484	4.2	2.15
Cycle 1994-95							
HT	100	1,400	617	714	-96	-0.13	
LT	100	600	265	489	-224	-0.46	
IM	100	1,000	441	684	-243	-0.36	

^x Treatments: HT = high technology, LT = low traditional technology, and IM = integrated management.

^y Using \$0.444 (1993-94) and \$0.441/kg (1994-95) of fruit in average per cycle. The U.S. dollar exchange rates were 4.50 and 6.80 Mexican pesos in May 1994 and May 1995, respectively.

^z Benefit/cost per two cycles (1993-94 and 1994-95).

0700 and 1100 h. The accumulated proportion of shoots with malformation was correlated negatively with $T_{\max}$ ($r = -0.681$, $P = 0.01$), T_h ($r = -0.586$, $P = 0.04$), and $RH \geq 60$ ($r = -0.82$, $P = 0.001$), and correlated positively ($r = 0.935$, $P = 0.0001$) with the wind speed (Table 4).

DISCUSSION

Our goal in this research was to quantify and examine the progress of malformation of mango and to compare the dynamics of epidemic development in orchards with different management tactics. Change in the traditional technology of Mexican mango growers, particularly pruning and the use of flowering promoters (KNO_3) (IM and HT), resulted in greater numbers of shoots produced and greater yield. However, increased shoot production also requires good management of MM to prevent high incidence levels due to the increased relative abundance of infection sites. IM, a new and alternative management strategy proposed to control MM, in general resulted in slower rates of epidemic development, lower levels of initial and final disease incidence, and a lower AUDPC in comparison with the other two management strategies. Yield was clearly higher with IM than with the other strategies in the first cycle (1993-94). In the second production cycle, yield was lower for all treatments due to alternate bearing, a common phenomenon in mango. The benefit-cost ratio of 4.2 for IM was almost three and four times higher than that obtained with HT and LT, respectively, in the higher production year. Combining both cycles, IM still had the higher benefit-cost ratio, i.e., 2.15 (Table 3). These results show the efficacy of combining practices, such as pruning and burning of diseased shoots, in reducing inoculum and allowing production of healthy vegetative shoots. In addition, the protection of these shoots with systemic and contact fungicides and the control of mites and ants, which apparently are factors for spore dissemination and tissue wounding (6,24; D. H. Noriega-Cantú, unpublished data), contributed to

the reduction of MM. Trees in the LT treatment did have low levels of disease in the first and second years; however, this was attributed primarily to a lack of vegetative and floral shoots. After the 2 years of this study, the management of MM using our IM approach appears promising.

In addition to examining the effects of the various management strategies, we also considered several factors related to epidemic analysis in the mango malformation system and to the effects of environment and inoculum availability on disease development. Several challenges, such as a need for incorporating $Y_{\max}$ into the disease progress models and for correcting for host growth, suggested comparison among treatments with several alterations to curve parameters in order to generate consistent conclusions. Incidence of MM never reached 100% in any of the treatments; thus a more appropriate value for $Y_{\max}$ was selected to fit disease progress curves to linear models (13). Only one $Y_{\max}$ was used (70%) to allow comparison among treatments (15). Also, the AUDPC was not used for the 1994-95 cycle due to different times of epidemic duration. Rather, AUDPC standardized (AUDPC_S) was used instead. This standardized parameter proved to be most appropriate for both cycles. Another correction for disease dilution due to host growth, i.e., the division of all values of number of diseased shoots by the final number of shoots produced, was applied to all disease progress curves. These corrections may be needed for many pathosystems in tropical perennial crops (2,12).

The causal agent of MM is controversial; however, some studies support the involvement of an airborne pathogen (3,4,6,11,19,23). In this study, we isolated *F. subglutinans* from diseased shoots, and the inoculation tests have been positive for this pathogen (D. H. Noriega-Cantú, unpublished data), which agrees with previous findings in Mexico and in many other mango-producing countries (3,4,19,23,32). However, *F. oxysporum* has also been

found in some regions where mangos are grown in México (4,6).

The association between disease incidence and climatic variables reflected a strong dependence of disease development on microclimatic factors measured at the canopy level. The cumulative disease incidence did not increase when the maximum daily temperatures and the average temperature per hour increased and prevailed at levels greater than 33 and 25°C, respectively, usually from March to May. In India, early-emerging flower buds were severely infected; whereas later buds escaped the disease; this difference was empirically attributed to the relatively high temperature during panicle development (11).

Even though *F. subglutinans* was consistently isolated from diseased shoots, it cannot be stated that the spores trapped in the canopy were exclusively attributed to this species. In general, the highest spore density was found during the rainy season, when wind speed (1.5 m/s) and relative humidity (92 to 94%) were high and the temperature was moderate (16 to 17.5°C). Similar results were reported in India (11). These researchers found high spore density of *F. subglutinans* with min/max temperatures of 8/27°C and with humidity of 85%. In our study, the greatest number of trapped airborne macroconidia of *Fusarium* spp. was characterized by morning periodicity (0700 to 1100 h), when wind speed (2.8 m/s) and temperature (29°C) were high and humidity was relatively low (55%). Because conidial density was strongly correlated with wind speed, wind may play a major role in the dispersal of the causal organism of MM. Wind could also be more important in the liberation of spores from dying or dry panicles than from live, infected panicles.

Integration of our results suggests the following sequence of events for malformation in a typical commercial orchard in the North Guerrero region: (i) Vegetative shoots emerge from the first (in mid-June) to the sixth (November) month after picking (MAP) the fruit. In this stage, the apical meristems are colonized extensively by

Table 4. Correlation coefficient, levels of significance, and number of observations in order to relate the incidence of malformation of mango with the dispersal of conidial and climatic variables. Growth cycles 1993-94 and 1994-95

Variables ^y	Fsp ^z	$T_{\max}$	T_h	$RH \geq 60$	$RH \leq 40$	Wind
CMM	0.9043	-0.403	-0.389	-0.064	-0.254	0.8336
	0.0001	0.137	0.152	0.821	0.361	0.0001
	15	15	15	15	15	15
MM		-0.681	-0.586	-0.820	-0.164	0.9345
		0.010	0.035	0.001	0.593	0.0001
		13	13	13	13	13
Fsp		-0.252	-0.243	0.029	-0.301	0.8120
		0.346	0.365	0.916	0.258	0.0001
		16	16	16	16	16

^y CMM is the change of the MM for the high technology treatment; MM is the accumulative proportion of disease; Fsp is the number of macroconidia of *Fusarium* spp.; $T_{\max}$ is the maximum daily temperature; $T_{\min}$ is the minimum daily temperature; T_h is the average temperature per hour; $RH \geq 60$ is the number of hours with relative humidity greater than or equal to 60%; $RH \leq 40$ is the number of hours with relative humidity less than or equal to 40%; and wind speed is in m/s.

^z First number is the actual value for the correlation; second number is the significance level of the correlation or the probability of obtaining a greater value; and the third number is n, the number of observations in the correlation analysis.

F. subglutinans (first to third MAP). (ii) The first visible symptoms appear during the fifth (October) and sixth (November) MAP on emerging vegetative shoots (Fig. 2). In this vegetative stage, disease incidence has the highest rate of incremental increase, with an incubation period from 2 to 5 months. Usually these vegetative shoots develop into diseased panicles, which are unproductive. (iii) Full bloom occurs in the seventh (December) to eighth (January) MAP. In this period, a second incremental increase in disease incidence is observed (January) (Fig. 2). Panicles that become diseased at this stage are also unproductive. (iv) A second vegetative flush occurs during the eighth (January) to the ninth (February) MAP, and a third incremental increase in disease incidence occurs (in mid-February to mid-March (Fig. 2). (v) Deformed vegetative and floral shoots remain in the tree until conditions of high humidity, appropriate temperatures, and strong winds promote dispersion of conidia and extensive colonization of the new apical meristems after fruit harvest. The emerging young shoots are susceptible to infection, and the development of vegetative and floral shoots is important to complete the inoculum production. However, it is unlikely that this accounts for all the variation observed, and other sources of seasonal variation are likely to be of importance. For example, changes in host susceptibility to infection between production cycles due to the influence of environmental and physiologic conditions of the host may be important. Because shoot development appears to play a significant role in the progress of MM, specific measures of host development and its incorporation into an epidemic model may be needed in future studies. Management influences shoot development and thus determines dynamics of the epidemic. Environmental factors, however, also influence changes of disease incidence, particularly through the inoculum dispersal. Studies are underway to take into account important environmental parameters and to consider host development more extensively so that more efficient MM management strategies can be evaluated, especially the IM strategy and other, even better, strategies that may be proposed for the North Guerrero region.

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