

Evaluation of Mango Cultivars for Resistance to Infection by *Ceratocystis manginecans*

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Abstract

Ceratocystis manginecans has been reported to cause a serious wilt disease of mango in Oman and Pakistan. To identify plants resistant to this disease, 30 mango cultivars were artificially inoculated with isolates of *C. manginecans* in three trials. Statistical analysis revealed significant differences ($P < 0.0001$) in lesion lengths among mango cultivars. Similarly, there were significant differences in the aggressiveness of the isolates used for inoculations. However, in trials where more than one isolate was used, there was no significant isolate x cultivar interaction suggesting that isolates do not affect the ranking of cultivars as susceptible or resistant. Cultivar 'Pairi' and local mango cultivars had the longest lesions and were ranked as highly susceptible. In contrast, cultivars 'Hindi Besennara', 'Sherokerzam', 'Mulgoa', 'Baneshan', 'Rose' and 'Alumpur Baneshan', had the smallest lesions and are considered as relatively resistant against *C. manginecans*. The inoculation results are concurrent with the incidence of wilt of these cultivars under field conditions.

INTRODUCTION

Mango (*Mangifera indica* L.) is one of the most important perennial fruit crops in the Sultanate of Oman (MoA, 2009a, 2009b). Mango wilt disease, caused by *Ceratocystis manginecans* M. van Wyk, A. Al-Adawi, & M.J. Wingf (Al Adawi et al., 2006; Van Wyk et al., 2007), is the most serious threat to the production of mango in the country. Since it was first reported in 1998, the disease has led to the death of many thousands of productive mango trees in the country and has been accompanied by a loss of valuable germplasm (Al Adawi et al., 2006).

Mango wilt disease in Oman is closely associated with the wood-boring beetle, *Hypocryphalus mangiferae* (Coleoptera: Scolytinae) that infests healthy trees. The bark beetle carries *C. manginecans* and as it bores into the wood, it creates an open wound for infection by the pathogen. Trees infected with the pathogen exude gum from infected stems/branches and the wood displays brown to black vascular discoloration. As the disease advances, tree parts or entire trees wilt and subsequently die. The disease is most serious in trees propagated from local seed sources and on exotic cultivars that are grafted on rootstock of local Omani cultivars. Where trees are grafted onto susceptible rootstocks, that rootstock is preferentially infested by wood boring insects and *C. manginecans* and tree death is more rapid than in the case of trees propagated from local seed sources (Al Adawi et al., 2006).

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A mango wilt disease very similar to that found in Oman has been reported from Pakistan. *Ceratocystis manginecans* and *H. mangiferae* have also been recovered from dying trees in that country (Malik et al., 2005; Van Wyk et al., 2007). Recent genetic analysis of *C. manginecans* populations from Oman and Pakistan using microsatellite markers has shown that the isolates from both countries collected from dying trees and from *H. mangiferae* are identical (A.O. Al Adawi, unpublished results). The population of *C. manginecans* collected from Oman and Pakistan represents a single genotype that was most probably introduced into these countries via mango propagation materials from a single source (Van Wyk et al., 2007; A.O. Al Adawi, unpublished results).

In 2001, the Ministry of Agriculture in Oman initiated a mango wilt disease management programme. This programme involved eradication of severely infected and dead mango trees, pruning of dead branches and using systemic insecticides and fungicides applied to infected trees through spraying or soil drenching. Following these measures, more than 13% of the mango trees in the Al Batinah region were eradicated (Al Adawi et al., 2006). Despite this aggressive programme, mango wilt disease continues to progress and occurs in all areas where mango trees are propagated. Since its first appearance in Oman, more than 60% of mango trees have been affected by the disease in certain regions (Al Adawi et al., 2003).

Control of the mango wilt disease using fungicides and insecticides has not been effective. This is in part because *C. manginecans* infects trees internally and the bark beetle larvae penetrate the wood. Consequently, development of genetic material resistant to infection by *C. manginecans* is considered as the most appropriate means to reduce the impact of the disease (Ploetz and Freeman, 2009). The aim of this study was to evaluate available local and exotic monoembryonic mango germplasm for resistance to infection by *C. manginecans*.

MATERIALS AND METHODS

Field Evaluation of Mango Cultivars

The incidence and severity of mango wilt disease was assessed in a farm in Barka (20 km west of Muscat) during April 2001. The mean incidence of mango wilt disease in Barka area was estimated at 13.6% during 2001 (Al Adawi et al., 2006). A plot size of 1.4 ha with trees of over six years of age and a multiplicity of mango cultivars was selected for incidence and severity assessments. All 13 mango cultivars were grafted on local rootstocks with the exception of local, seed propagated cultivars. The incidence of mango wilt disease was assessed as the number of wilted trees of each cultivars present in 2001 and was reassessed after three years. A linear scale from 0 to 5 was developed to evaluate disease severity, where 0 = no symptoms, 1 = slight gummosis, 2 = moderate gummosis with obvious vascular discoloration, 3 = extensive gummosis and severe vascular discoloration, 4 = severely infected with partial wilt and 5 = total tree wilt.

Disease incidence was also assessed on mango genebank material at Waqaibah (24°20'42.79"N, 56°43'25.93"E) and Wadi Haibi (24°19'10.37"N, 56°43'00.62"E) close to Sohar in North East Oman during 2002 and 2003. The genebank consisted of 32 local mango accessions collected from various mango cultivation areas in Oman, in addition to the 23 exotic cultivars imported as part of a cultivar performance assessment programme. The age of all local mango accessions and six of the exotic cultivars ('Arumanis', 'Hamlet', 'Golek', 'Oaies', 'Taimour' and 'Zabidia') at time of the first disease incidence assessment was five-years-old with 3 trees per accession. Sixteen of the exotic mango cultivars grown at Waqaibah were 12 twelve year old trees, with 16 trees per cultivar. At Wadi Haibi, five exotic mango cultivars ('Baramasi', 'Dasheheri', 'Neelum', 'Rose' and 'Tenneru') of three-year old trees were also included in the wilt incidence evaluation. The number of wilted trees of each cultivar was recorded monthly. An incidence disease scale was used where 0-20% indicated tolerant cultivars, 21-40% moderately tolerant cultivars, 41-60% moderately susceptible cultivars, 61-80% susceptible cultivars and 81-100% highly susceptible cultivars.

Inoculum and Inoculations

Thirty mango cultivars from diverse sources were evaluated for resistance to *C. manginecans* (Table 1). Due to variable availability of plants, three separate inoculation trials were conducted in April 2005, February 2007 and November 2009. Each of the mango cultivars was grafted onto root-stocks of undefined local origin and grown in 13 cm diameter pots containing loamy soil mixed with peat moss (1:1 v:v) and kept under shade-house conditions.

Inoculum of *C. manginecans* was grown on malt extract agar (MEA) for two weeks at 25°C. Mango plants were inoculated by making a 1 cm, I-shaped incision 20 cm above the graft union using a sterile scalpel and following the methods described by Al Adawi et al. (2006).

Inoculation Trials

In the first trial, six monoembryonic mango cultivars including ‘Baramasi’, ‘Dasheheri’, ‘Pari’, ‘Rose’, ‘Tenneru’ and ‘Zafran’ were artificially inoculated with two isolates (CMW15351 and CMW13854) of *C. manginecans* shown to be pathogenic in previous pathogenicity tests (Al Adawi et al., 2006). Twenty, six-month-old seedlings with an average scion diameter of 14.2 mm (at inoculation position) were used. Seven plants of each cultivar were inoculated with each isolate by inserting mycelial discs (2 mm diameter) into the wounds on the stems. Five plants of each mango cultivar were also inoculated with sterile MEA as controls. All wounds were covered with moistened, sterile cotton pads and sealed with Parafilm to maintain a humid environment. After 60 days of incubation, the bark was removed to expose the discolored xylem and the lesion length was measured.

The second trial included thirteen mango cultivars, eight Indian monoembryonic cultivars (‘Baramasi’, ‘Dasheheri’, ‘Imampasand’, ‘Langra’, ‘Pari’, ‘Rose’, ‘Tenneru’ and ‘Zafran’), one Egyptian polyembryonic cultivar (‘Hinidi Besennara’), and four locally developed cultivars (‘Ishbiah’, ‘Mantkah Al thour’, ‘Muscati’ and ‘Sohar 2005’). Nine, eighteen-month-old mango plants, with an average scion diameter of 11.3 mm, were inoculated as above using *C. manginecans* isolate CMW13854 and a control of sterile MEA. Lesion length was evaluated 60 days after inoculation.

In the third trial, twenty eight mango cultivars were used including local and exotic monoembryonic and polyembryonic mango cultivars (Table 5). Twenty, two-month-old seedlings with an average scion diameter of 12.7 mm, were inoculated as above using two isolates (CMW13851 and CMW13854) of *C. manginecans*. Each isolate was inoculated on four plants of each cultivar and an equal number of control plants were inoculated with sterile MEA. Lesion lengths were measured after 36 days.

Data Analysis

Statistical analyses were performed using SAS (SAS Institute, Cary, NC, USA). Analysis of variance according to the general linear model procedure (GLM) was used to compare lesion lengths between isolates and mango cultivars. Where there were significant differences, Duncan’s multiple range tests was used to separate means. The interaction between isolate and cultivar in the first and last trials were examined and the data of the three trials were combined after determining that there was no significant interaction. Where cultivars had not been evaluated in one or two trials, they were treated as missing data in the combined data set. The differences in lesion lengths for combined data and among isolates, years and cultivars were tested using analysis of variance and significant differences between means were separated by Duncan’s multiple range test. Because isolate CMW13854 was used in all three trials, the data were reanalyzed using inoculation data for only this isolate to evaluate possible differences between trials.

RESULTS

Field Evaluation of Mango Cultivars

The assessment of wilt incidence in Barka during the period 2001-2004 revealed that almost half of the mango trees present in 2001 ($101/208 = 48.6\%$) had been killed by 2004. The number of dead trees was higher in local germplasm ($43/69 = 62.3\%$). The proportion of killed trees was also high in 'Tenneru' ($3/4 = 75\%$), 'Alphonso' ($11/19 = 57.9\%$), 'Langra' ($3/6 = 50\%$), 'Imampasand' ($1/2 = 50\%$), 'Neelum' ($9/19 = 47.4\%$) and 'Pairi' ($9/20 = 45\%$). The cultivars 'Rose' ($2/8 = 25\%$), 'Baramasi' ($1/4 = 25\%$), 'Bangalora' ($0/4 = 0\%$) and 'Baneshan' ($0/1 = 0\%$) were least affected.

Disease severity assessments during 2001 indicated the lowest mean disease severities in cultivars 'Bangalora', 'Neelum', and 'Dasheheri', and the highest in local germplasm and the cultivars 'Langra' and 'Pairi' (Fig. 1). Seed-propagated mango trees were pooled as local types, or non-grafted material whereas cultivars propagated by grafting were grouped as "grafted" in order to determine the effect of propagation method on disease severity. There was a significant difference ($P < 0.0001$) in mean disease severity for local (2.94) and grafted (1.45) material (Fig. 2). Infection and disease development in grafted trees was invariably within the rootstock, i.e., originating from infection of local material. Although xylem discoloration was rarely observed in the scion of grafted trees, rapid tree death was recorded due to death of the rootstock.

During 2002-2003, the overall incidence of tree death due to mango wilt was 53.2% ($194/365$) in the mango germplasm trial at Sohar. Local mango germplasm showed high incidence, with 26 of the 32 accessions (81.3%) showing tree losses between 66.7 and 100%. Ten of the 23 (43.5%) exotic cultivars had tree losses between 50 and 100%. Local germplasm 'Al Arash' and 'Oad Al Roob' both showed 100% wilt incidence in the germplasm trial. The highest incidence among exotic cultivars was recorded in 'Bangalora' (81.3%), 'Neelum' (66.7%), 'Golek' (68.8%), 'Alphonso' (62.5%), 'Pairi' (56.3%), 'Imampasand' (56.3%) and 'Sherokerzam' (50.0%). Lowest disease incidence among exotic cultivars was recorded in 'Langra' (37.5%), 'Baramasi' (37.5%), 'Baneshan' (31.3%), 'Zafran' (31.3%), 'Rose' (30.8%), 'Hindi Besennara' (25.0%), 'Mulgoa' (25.0%), 'Tenneru' (24.0%) and 'Dasheheri' (18.8%) (Table 6).

Artificial Inoculation of Mango Cultivars

Statistical analyses of the results of the inoculation trial conducted in 2005 revealed significant differences ($P < 0.0001$) in lesion length between mango cultivars (Table 2). Significantly longer lesions ($P = 0.005$) were produced by isolates CMW13854 ($X = 26.12$ cm) and CMW15351 ($X = 19.20$ cm) compared to control inoculations ($X = 1.12$ cm). Mean lesion length in cultivar Pairi (40.3 cm) was significantly greater than that on cultivar Rose, which had the smallest ($X = 11.1$ cm) lesions of all cultivars tested (Table 3). (These should be \bar{x} with a hyphen above to denote means).

For the trial conducted in 2007, there were significant differences ($P < 0.0001$) in the response of different cultivars to inoculation with *C. manginecans* (Table 2). Results of this trial also showed that cultivar 'Pairi' and the four local mango cultivars tested were highly susceptible to infection, having the most extensive lesion development ($X = 42.11$ cm). Cultivar 'Rose' ($X = 18.1$ cm), 'Hindi Besennara' ($X = 22.4$ cm) and 'Baramasi' ($X = 22.7$ cm) had the smallest lesions. Insignificant lesions ($X = 1.3$ cm) were associated with the control inoculations (Table 4).

In the trial conducted in 2009, there were significant ($P < 0.0001$) differences in lesion length among cultivars (Table 5). Cultivar 'Pairi' was highly susceptible to infection, having the most extensive lesion development ($X = 60.8$ cm). The six local mango accessions 'Al Batikah', 'Al Arash', 'Ishbiah', 'Oad Al Roob' and 'Sohar 2005' also had extensive lesion development. Some of this material died during the trial (Table 5). Cultivars with the most limited lesion development included 'Baneshan', 'Rose' and 'Alumpur Baneshan'. Among the Egyptian polyembryonic cultivars tested in this trial, only 'Golek' was highly susceptible to infection. In contrast, the 'Taimor' and 'Hindi

Besennara' were less susceptible to infection and their mean lesion lengths (22.8 cm and 18.9 cm respectively) were similar to those of the least susceptible Indian cultivars. The lesions associated with control inoculations were small and significantly different from those where *C. manginecans* was used as inoculum ($P < 0.0001$).

Analysis of variance for lesion length where the data for three trails were combined revealed significant differences ($P < 0.0001$) among cultivars and between isolates ($P = 0.0046$) tested in different trails. There was no significant difference between trial years ($P = 0.3893$), the isolate x cultivar interaction was not significant ($P = 0.7073$) but there was a significant year x cultivar interaction ($P < 0.0001$). Inoculations using isolate CMW13854 were repeated in three trails and this isolate also consistently produced the most extensive lesion development compared with other isolates (CMW13851 and CMW15351) tested in 2005 and 2009.

Analysis of variance of lesion length for pooled data for the three trails but including only data for isolate CMW13854 revealed significant differences between cultivars ($P < 0.0001$), no significant difference ($P = 0.4466$) between trial years, and a significant ($P = 0.0006$) year x cultivar interaction. 'Pairi' and local mango cultivars were consistently, highly susceptible to infection by isolate CMW13854. Likewise, the cultivars 'Rose', 'Baneshan' and 'Alumpur Baneshan' consistently displayed the lowest levels of infection by this isolate (Table 6).

DISCUSSION

Thirty mango cultivars were evaluated for resistance to infection by *C. manginecans* in three consecutive inoculation trails. Although the isolates did not have the same level of aggressiveness, there was no significant isolate x cultivar interaction. Cultivars did, however, differ in their susceptibility to infection and results were consistent with field observations where local germplasm was generally more susceptible than that from outside Oman.

Inoculation trials showed that none of the cultivars tested were immune, or showed high levels of resistance to infection by *C. manginecans*. Ranking of mango cultivars based on lesion length following artificial inoculation generally correlated with field assessments of the disease. Cultivar 'Pairi' was highly susceptible having the longest lesions after inoculation. Similarly, most of the local mango accessions included in the third inoculation such as 'Al Batikah', 'Ishbiah' and 'Al Arash' had the longest lesions and the highest levels of mortality at 88, 37 and 25% respectively. This is consistent with mango wilt disease incidence evaluation in the mango genebank at Sohar where 'Pairi' and local cultivars such as 'Al Batikah', 'Al Arash', and 'Oad Al Roob' were severely affected by the disease with mortality incidences of 56.3, 33.3, 100 and 100% respectively. In the cases where local mango accessions were used as the scion, the disease symptoms were first expressed on the scion part of infected trees which represents the more susceptible portion of the trees. Furthermore, higher levels of disease were found in local mango cultivars compared to exotic cultivars. Disease severity assessments at a farm in the Barka area showed consistently higher levels of disease in local mango cultivars other than a limited number of exotic cultivars such as 'Pairi' (Al Adawi, 2002; Al Adawi et al., 2006). Therefore, high incidence of wilt in some exotic cultivars might be attributed to the death of local rootstock material due to *C. manginecans* infection, rather than susceptibility of exotic cultivars.

Results of this study showed that there are some reasonable levels of resistance to infection by *C. manginecans* in some mango cultivars. In the largest inoculation trial conducted in 2009 and taking into consideration inoculations with isolate CMW13854 in all three trials, cultivars 'Hindi Besennara', 'Mulgoa', 'Baneshan', 'Rose' and 'Alumpur Baneshan' are the most resistant. Some of these cultivars have been assessed for disease incidence and severity at the mango genebank at Sohar and at Barka and they have also been shown to be relatively resistant to infection under natural conditions (Al Adawi, 2002). Thus, the mango wilt management program should consider excluding the highly

susceptible mango cultivars and to rather use these cultivars that performed much better for future propagation.

In all trials, control plants were randomly placed between those that were inoculated. A small number of control plants in the 2009 trial developed vascular discoloration similar to that in the inoculated plants, probably due to insects or mites carrying inoculum to wounds made on the control plants. *Ceratocystis manginecans* produces a very strong banana odour and inoculum would have been attractive to insects that could move between plants (Kile, 1993; Van Wyk et al., 2007). While the control plants were not required for comparisons of response between different mango cultivars, they were included to provide a basis of comparison in the inoculations.

There was a significant year x cultivar interaction in the inoculation trials. Scion diameter could have contributed to this interaction. Scion diameter reflects tissue age and this varied significantly ($P < 0.0001$) among inoculated cultivars within and between trials. Mango trees undergo phenological changes including annual vegetative and reproductive growth and consequently, physiological changes (Davie and van Vuuren, 1998; Davenport, 2009) that are influenced by the environment. Host physiological status such as tissue age and phenological change are well-known factors that can affect pathogen progress within inoculated tissue (Eversmeyer et al., 1980; Tomerlin et al., 1983; Turechek and Stevenson, 1998; Prell and Day, 2001; Pariaud et al., 2009). The possible effect of physiological status during cultivar evaluation for resistance against *C. manginecans* needs further investigation.

In the 2005 and 2009 trials, isolates of *C. manginecans* showed significant differences in aggressiveness to mango cultivars. However, all of these isolates of *C. manginecans* were pathogenic and produced vascular discoloration in all the tested mango cultivars. The isolate CMW13854 was shown to be most aggressive in all inoculation trials but none of the isolates interacted differentially with mango cultivars in the 2005 and 2009 trials. Van der Plank (1984) suggested that a non-significant isolate x cultivar interaction would indicate horizontal resistance. Horizontal resistance is known to be durable since it is determined polygenically by the combined action of many minor genes. Most of the mango cultivars grown in Oman are derived from Indian monoembryonic cultivars that have a single zygotic embryo and produce hybrid seedlings (Iyer and Schnell, 2009). Hence, seed propagation from those cultivars will produce plants with substantial genotypic variation and give rise to diverse genotypes. Therefore, the levels of resistance in mango cultivars to infection by *C. manginecans* (and other desirable characteristics) could be enhanced through an extensive breeding program (Prell and Day, 2001).

Breeding for resistance to *C. manginecans* would probably need to include hybridization between resistant cultivars in order to produce highly resistant cultivars. Furthermore, hybridization between resistant mango cultivars and those with lower levels of resistance would potentially produce resistant cultivars with more suitable horticultural criteria. Remnant mango trees left after the serious mango wilt epidemic represent a potential source of horizontal resistance and should be selected and evaluated for their resistance to infection. For example, breeding programs in Brazil have produced mango cultivars resistant to infection by *Ceratocystis fimbriata* s.l. such as 'IAC 101 Coquinho', 'IAC 102 Touro', 'IAC 104 Dura' and 'IAC 106 Jasmin' that are descendants of susceptible cultivars including 'Coquinho' and 'Jasmin' (Ribeiro et al., 1995; Rossetto et al., 1996).

In order to replace current susceptible local monoembryonic rootstock cultivars, resistant polyembryonic cultivars as potential future rootstocks represent a high priority in the breeding program. Currently, 'Taimour' and 'Hindi Besennara' represent a suitable option to serve as rootstocks until further highly resistant cultivars can be found. Therefore, the current breeding program in Oman includes a selection of remnant mango trees and introduction of monoembryonic and polyembryonic mango germplasm from various part of the world for evaluation of resistance against *C. manginecans*.

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Tables

Table 1. Embryo type and origin of 30 mango cultivars used in resistance evaluations against *Ceratocystis manginecans* during three inoculation trials.

No.	Cultivar	Embryo type ¹	Origin	No.	Cultivar	Embryo type	Origin
1	Amrapali ²	M	India	16	Langra	M	India
2	Alumpur Baneshan	M	India	17	Mantkah Al thour	M	LA
3	Al Arash	M	LA ³	18	Mulgoa	M	India
4	Al Batikah	M	LA	19	Muscatti	M	LA
5	Alphonso	M	India	20	Neelum	M	India
6	Bangalora	M	India	21	Oad Al Roob	M	LA
7	Baneshan	M	India	22	Pairi	M	India
8	Baramasi	M	India	23	Rose	M	India
9	Dasheheri	M	India	24	Sherokerzam	M	India
10	Golek	P	Egypt	25	Sindhu ⁴	M	India
11	Hajeb	M	LA	26	Sohar 2005	M	LA
12	Hindi Besennara	P	Egypt	27	Sournka	M	India
13	Imampasand	M	India	28	Taimour	P	Egypt
14	Ishbiah	M	LA	29	Tenneru	M	India
15	Khoh	M	LA	30	Zafran	M	India

¹ M = Monoembryonic, P = Polyembryonic.

² Amrapali = Dasheheri × Neelum.

³ Local accession.

⁴ Sindhu = Ratna (Neelum × Alphonso) × Alphonso.

Table 2. Analysis of variance of lesion length caused by isolates of *Ceratocystis manginecans* inoculated on different mango cultivars during infection trials.

Year	Source of variation	df	Sum square	Mean square	F value	P > F
2005	Isolate	1	1004.65	1004.65	8.41	0.0050
	Cultivar	5	6644.46	1328.89	11.12	0.0001
	Isolate * Cultivar	5	608.76	121.75	1.02	0.4130
2007	Cultivar	12	4186.55	348.88	5.24	0.0001
2009	Isolate	1	643.84	643.84	4.25	0.0407
	Cultivar	27	28931.78	1071.55	7.08	0.0001
	Isolate * Cultivar	27	2701.29	100.05	0.66	0.8971
All years	Isolate	2	1345.36	672.68	5.49	0.0046
	Cultivar	29	30574.84	1054.30	8.60	0.0001
	Year	2	232.09	116.05	0.95	0.3893
	Isolate * Cultivar	31	3210.73	103.57	0.84	0.7073
	Year * Cultivar	14	5577.23	398.37	3.25	0.0001
All years with isolate CMW13854 only	Cultivar	29	26639	918.6	6.9	0.0001
	Year	2	215.4	107.7	0.81	0.4466
	Year * Cultivar	15	5599	373.3	2.8	0.0006

Table 3. Mean lesion length in six mango cultivars inoculated with two isolates of *Ceratocystis manginecans* in evaluation trial conducted during April 2005.

Cultivar	Scion diameter (mm)	Lesion length (cm)			
		CMW13854	CMW15351	Mean	Control
Pairi	16 ± 2.4	48.9 ± 15.1 a ¹	31.6 ± 8.9 a	40.3a	0.5 ± 0.6
Baramasi	16.3 ± 4.3	28.4 ± 19.3 b	20.4 ± 5.4 abc	24.4b	1.1 ± 0.9
Zafran	13.3 ± 2.4	22.5 ± 14.5 b	22.6 ± 11.8 ab	22.5bc	0.3 ± 0.5
Tenneru	13.4 ± 2.3	21.8 ± 8.1 b	16.2 ± 10.2 bc	19bc	1.5 ± 1.6
Dasheheri	15 ± 2.3	20.1 ± 6.9 b	17.1 ± 10.7 abc	18.6bc	2.3 ± 1.9
Rose	11.1 ± 4.1	15.1 ± 2.9 b	7.2 ± 6.3 c	11.1c	0.8 ± 0.4
Mean ²	14.2 ± 3.5	26.1a	19.2b		1.1c

¹ Values within same column followed by the same letter are not significantly different at $P < 0.05$ using Duncan's multiple range test.

² Values within mean row followed by the same letter are not significantly different at $P < 0.05$ using Duncan's multiple range test.

Table 4. Mean lesion length on 13 mango cultivars inoculated with one isolate of *Ceratocystis manginecans* in evaluation trial conducted during February 2007.

Cultivar	Scion diameter (mm)	Lesion length (cm)		Mortality (%)
		CMW13854	Control	
Pairi	9.4 ± 1.2	42.1 ± 8.2 a ¹	1	10
Tenneru	10.9 ± 1.5	35.4 ± 13 ab	1	0
Imampasand	12.3 ± 1.2	33.7 ± 8.8 abc	2	0
Mantkah Al thour	10 ± 1.8	33.5 ± 6.7 abc	4	0
Sohar 2005	16.8 ± 1.4	32.7 ± 12.7 abc	2	0
Dasheheri	13.3 ± 1.2	32.7 ± 7.5 abc	1	0
Muscati	13.2 ± 1.9	32 ± 9.1 bcd	2	0
Ishbiah	7.5 ± 1.1	31.6 ± 9.8 bcde	1	100
Zafran	10.9 ± 0.7	27.6 ± 4 bcde	1	0
Langra	13.1 ± 1.3	24.6 ± 6.1 cdef	1	0
Baramasi	8.7 ± 1.8	22.7 ± 3.5 def	1	11.1
Hindi Besennara	12.9 ± 2.4	22.4 ± 5.2 ef	1	0
Rose	8.7 ± 0.9	18.1 ± 5.4 f	1	0
Mean ²	11.3 ± 2.9	30a	1.3b	

¹ Values within same column followed by the same letter are not significantly different at $P < 0.05$ using Duncan's multiple range test.

² Values within mean row followed by the same letter are not significantly different at $P < 0.05$ using Duncan's multiple range test.

Table 5. Mean lesion length of 28 mango cultivars inoculated with two isolates of *Ceratocystis manginecans* in infection trials conducted during December 2009.

Cultivar	Scion diameter (mm)	Lesion length (cm)			Mortality (%)	
		CMW13851	CMW13854	Mean		
Pairi	17.5 ± 4.6	46.5 ± 29.6 ab ¹	75 ± 44.9 a	60.8a	21.3 ± 24.2	0
Golek	14.9 ± 5.7	53 ± 7.5 a	50.8 ± 9.7 b	51.9ab	25.8 ± 14.3	12
Al Batikah	10.6 ± 1.8	38.5 ± 2.4 abc	51 ± 10.6 b	44.8bc	1 ± 0	88
Ishbiah	13.2 ± 2.4	39.3 ± 9.8 abc	45 ± 16.3 bc	42.1bc	17.1 ± 11.1	37
Alphonso	13 ± 4.1	36 ± 12.3 bcde	36.3 ± 2.6 bcd	36.1cd	2.5 ± 2.4	0
Imampasand	14.3 ± 4.5	39.3 ± 10.9 abc	30.3 ± 14.4 bcd	34.8cde	0.5 ± 0	0
Al Arash	12.3 ± 3	31.5 ± 7.9 bcdef	37.8 ± 7.4 bcd	34.6cde	6.5 ± 5.7	25
Oad Al Roob	12.7 ± 2.3	28.5 ± 11.2 cdefg	35.3 ± 11.1 bcd	31.9cdef	6.1 ± 7.9	0
Khoh	12 ± 5.2	29.8 ± 14.7 bcdefg	33.5 ± 29.4 bcd	31.6cdef	32 ± 26.9	0
Sohar 2005	13.4 ± 2.1	25 ± 4.3 cdef	35.8 ± 11 bcd	30.4cdef	2.3 ± 2.5	0
Bangalora	17.5 ± 3.3	24 ± 12 cdefg	29.3 ± 19.9 bcd	26.6defg	1 ± 0	0
Langra	11.2 ± 3.4	28.3 ± 16.7 cdefg	18 ± 0 d	25.8defg	1 ± 0.6	0
Neelum	15.6 ± 5.1	21.8 ± 12.3 cdefg	29.3 ± 8.6 bcd	25.5defg	7.9 ± 8.3	0
Sournka	13 ± 7	19 ± 7.7 efg	28.3 ± 11 bcd	23.6defg	15.5 ± 20.5	0
Baramasi	6.9 ± 1	22.7 ± 5.5 cdefg	23.1 ± 10.8 cd	22.9defg	3.4 ± 5.5	0
Amrapali	12.8 ± 6.5	19 ± 9.4 efg	26.8 ± 13.7 bcd	22.9defg	6 ± 7.1	0
Taimour	13 ± 5.5	25.8 ± 14.9 cdef	19.8 ± 7.1 cd	22.8defg	3.4 ± 5.5	0
Sindhu	11.4 ± 4.6	19.5 ± 9.6 efg	24 ± 3.9 cd	21.8defg	3.4 ± 2.8	0
Dasheheri	12.7 ± 2.2	20.5 ± 7.2 defg	20.8 ± 8.8 cd	20.6efg	1 ± 0	0
Sherokerzam	12.1 ± 2.7	19.8 ± 3.4 efg	19.9 ± 6.9 cd	19.8fg	1 ± 0	0
Mulgoa	14.3 ± 3	18.5 ± 7.6 efg	20 ± 5.2 cd	19.3fg	1 ± 0	0
Hindi Besennara	14.8 ± 3	17.1 ± 4.3 fg	20.8 ± 5.3 cd	18.9fg	4.8 ± 5.7	0
Hajeb	10.9 ± 5.1	14.8 ± 5.4 fg	22.5 ± 3.4 cd	18.6fg	11.5 ± 0.7	0
Tenneru	14 ± 4.6	17.8 ± 9 efg	19 ± 3.7 d	18.4fg	1 ± 0	0
Zafran	10 ± 3.4	19.8 ± 2.5 efg	15.3 ± 4.9 d	17.5fg	1 ± 0	0
Baneshan	12.5 ± 4.5	11.9 ± 3.1 g	16.3 ± 9.1 d	14.1g	1 ± 0.3	0
Rose	6.7 ± 0.6	12.8 ± 6.4 g	15.3 ± 3.3 d	14g	6 ± 7.1	0
Alumpur Beneshan	11.6 ± 4.3	14 ± 3 fg	13.5 ± 7.8 d	13.8g	1.4 ± 0.5	0
Mean ²	12.7 ± 4.6	25.5b	29.3a		6.3c	

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Table 6. Combined data analysis of mean lesion length of 30 mango cultivars inoculated with isolates of *Ceratocystis manginecans* in three infection trials.

Cultivar	Trials	Mean lesion length in three trials	Mean lesion length in three trials using isolate CMW13854	Death incidence in Barka farm 2001-2004	Death incidence in Sohar genebank 2002-2003	
					Waqibah	Wadi hebi
Golek	2009	51.9a ¹	50.8a	-	2/3 (66.7%)	-
Pairi	2005, 2007, 2009	46.1ab	51.1a	9/20 (45%)	9/16 (56.3%)	-
Al Batikah	2007, 2009	44.8abc	51a	-	1/3 (33.3%)	-
Ishbiah	2007, 2009	36.5bcd	35.7abc	-	-	-
Alphonso	2009	36.1bcd	36.3abc	11/19 (57.9%)	10/16 (62.5%)	-
Al Arash	2009	34.6cde	37.8ab	-	3/3 (100%)	-
Imampasand	2007, 2009	34.3cdef	32.5bcd	1/2 (50%)	9/16 (56.3%)	-
Mantkah Al thour	2007	33.5defg	33.5bc	-	-	-
Muscati	2007	32defgh	32bcd	0/1 (0%)	-	-
Oad Al Roob	2007, 2009	31.9defgh	35.3bc	-	3/3 (100%)	-
Khoh	2009	31.6defgh	33.5bc	-	-	-
Sohar 2005	2007, 2009	31.5defghi	33.8bc	-	-	-
Bangalora	2007, 2009	26.6defghij	29.3bcde	0/4 (0%)	13/16 (81.3%)	-
Neelum	2007, 2009	25.5defghijk	29.3bcde	9/19 (46.4%)	11/16 (68.8%)	9/22 (41%)
Langra	2007, 2009	25.defghijk	23.8bcde	3/6 (50%)	6/16 (37.5%)	-
Sournka	2009	23.6efghijk	28.3bcde	-	-	-
Baramasi	2005, 2007, 2009	23.6efghijk	25bcde	1/4 (25%)	6/16 (37.5%)	3/24(13%)
Amrapali	2009	22.9fghijk	26.8bcde	0/1 (0%)	-	-
Tenneru	2005, 2007, 2009	22.8fghijk	26.5bcde	3/4 (75%)	-	6/25(24%)
Taimour	2009	22.8fghijk	19.8cde	-	1/3 (33.3%)	-
Dasheheri	2005, 2007, 2009	22.6fghijk	25.2bcde	4/10 (40%)	3/16 (18.8%)	1/22(5%)
Zafran	2005, 2007, 2009	22.3ghijk	22.9bcde	15/39 (38.5%)	5/16 (31.3%)	-
Sindhu	2009	21.8ghijk	24bcde	-	-	-
Hindi Besennara	2007, 2009	20.6hijk	21.8bcde	-	4/16 (25%)	-
Sherokerzam	2009	19.8ijk	19.9cde	-	8/16 (50%)	-
Mulgoa	2009	19.3jk	20cde	-	4/16 (25%)	-
Hajeb	2009	18.6jk	22.5bcde	-	-	-
Baneshan	2009	14.1k	16.3de	0/1 (0%)	5/16 (31.3%)	-
Rose	2005, 2007, 2009	13.9k	16.5de	2/8 (25%)	-	8/26(31%)
Alumpur Beneshan	2009	13.8k	13.5e	-	5/16 (31.3%)	-

¹ Values within same column followed by the same letter are not significantly different at $P < 0.05$ using Duncan's multiple range test.

Figures

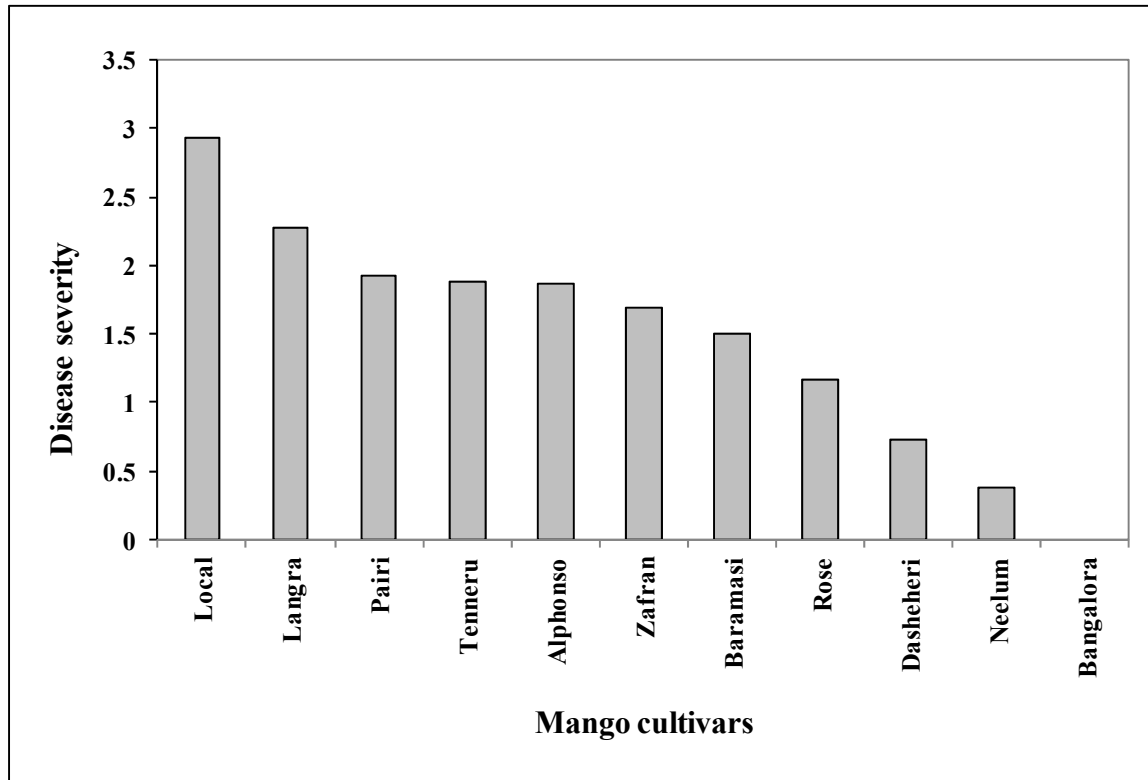


Fig. 1. Severity assessments for mango wilt disease on eleven mango cultivars in mango farm in Barka area during 2001.

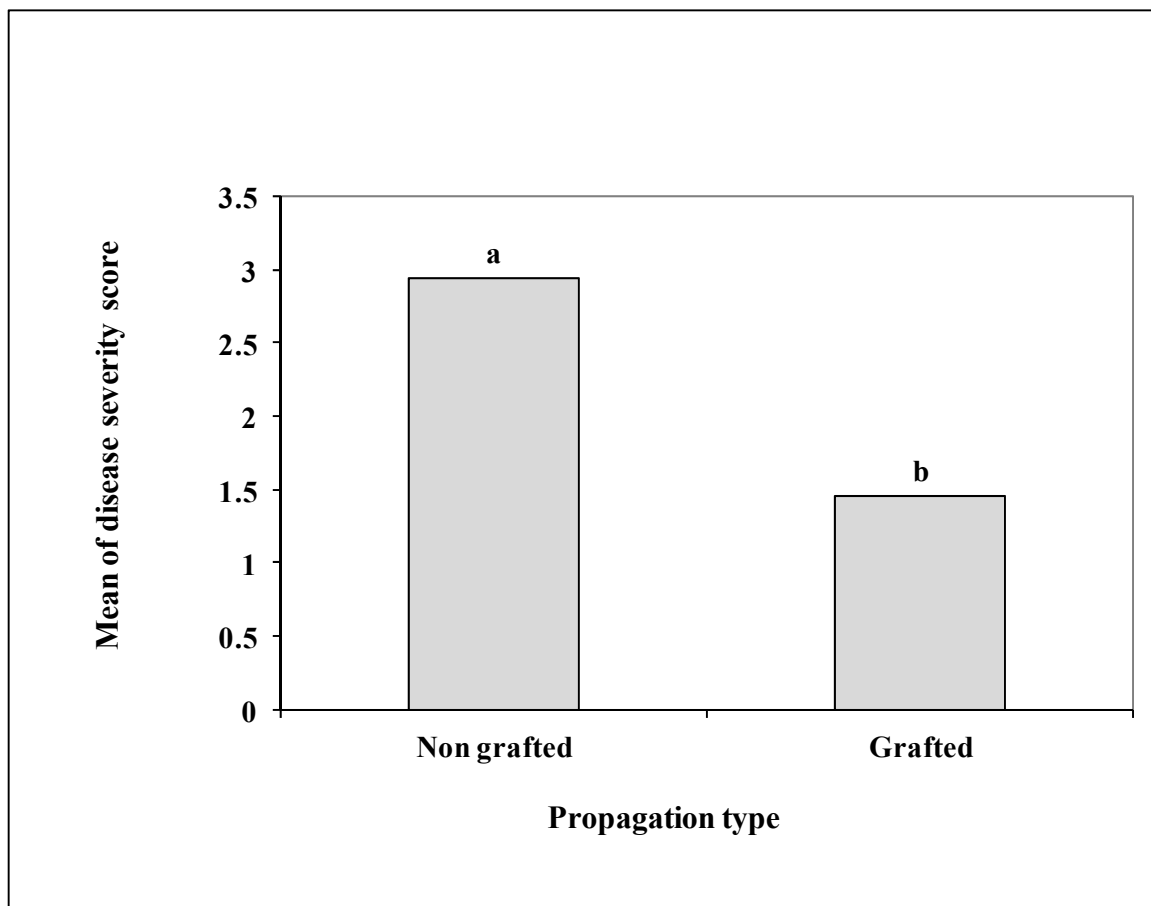


Fig. 2. Disease severity assessments of mango wilt disease for grafted and non grafted mango trees in a farm in Barka area during 2001.