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BACTERIAL APICAL NECROSIS OF MANGO CAUSED BY *PSEUDOMONAS SYRINGAE* PV. *SYRINGAE* IN EGYPT.

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ABSTRACT: A necrotic bacterial disease of mango trees (*Mangifera indica*) affecting buds, leaves, and stems were observed on commercial mango trees in Egypt since 2004 - 2005.

Twenty bacterial strains representing 20 different localities isolated from symptomatic tissues were identified as *P. syringae* pv. *syringae* on the basis of its physiological and biochemical characteristics, ice nucleation activity and host range. Pathogenicity tests on mango plants showed that the all bacterial strains incited the typical symptoms of apical necrosis.

Bacterial apical necrosis of mango caused by *Pseudomonas syringae* pv. *syringae* is described for the first time in Egypt.

INTRODUCTION

Mango, *Mangifera indica* L., is grown throughout the tropics and subtropics and is one of the most important fruit crops in the world. India produces 70% of the world's mangoes, although Mexico is the largest exporter of fresh fruits (Ploetz et al, 1994). In Egypt, mango crop is consider as one of the most economic fruit crops after citrus and grapevine, and its planting is concentrated in Al-Esmaeilia (33904 Feddans), Al-Sharkia (17004 Feddans), Al-Giza (7665 Feddans), Al-Fayoum (4342 Feddans) and Al-Nobaria region (11221 Feddans), that produce approximately 287317 tons (Ministry of agriculture 2004).

Some bacterial diseases of mango trees caused by *Agrobacterium tumefaciens*, *Bacillus subtilis*, *Erwinia carotovora* pv. *carotovora* (Bradbury, 1986), and *Xanthomonas campestris* pv. *Mangiferaeindicae* (the most widely known and producer of bacterial black spot of mango fruits) have been previously described (Ploetz et al, 1994). Apical necrosis disease results in significant economic losses and is one of the primary factors limiting mango fruit production in Spain and Portugal (Cazorla et al, 1992 & 1998).

The disease is caused by *P. syringae* pv. *syringae*. This pathogen usually produces blossom blast and leaf necrosis from autumn to spring. but blast of dormant buds is the most destructive phase of the disease (Cazorla et al, 1998). The environmental conditions favoring disease development are relatively low temperature and rainy wet weather (Cazorla et al, 1998). Similarly, apical necrosis in mango trees is frequently observed from late October to March and reaches its maximum development during January which is the cool wet season in Egypt.

Disease symptoms include necrosis of vegetative and flower buds and bud failure before bud break. Necrotic lesions in buds sometimes extend to the leaf petiole through the stem. Generally, a white milky gum exudes from necrotic lesions on buds, stems, and less frequently on petioles. Necrotic symptoms also affect flower panicles, causing the most severe economic losses because of the decrease in fruit set (Cazorla et al, 1998).

Apical bud necrosis and leaf spotting have been observed in mango trees grown in different localities in Egypt since 2004 - 2005. Mango buds, leaves, and stems are all susceptible to infection, but fruit lesions have not been observed.

The main object of this work was to identify and study the role of *P. syringae* pv. *syringae* as a causal agent of the apical necrosis in mango trees in Egypt.

MATERIALS AND METHODS

Causal agent isolation :

Mango trees in orchards at 20 localities in Egyptian Governorates of Al-Ismailia, Al-Sharkia, Al-Behira (Al-Nobaria region), Al-Giza and Al-Fayoum were sampled for the presence of bacterial apical necroses disease during November 2005 to April 2006. Affected leaves, buds, and other tissues showed symptoms of apical necroses disease were collected, placed in plastic bags and transported to the laboratory, where they kept at 5°C until processed within 24-48 h. To isolate the causal agent of apical necrosis, small pieces of affected leaves, buds, and other tissues were cut from the edge of a recent necrotic lesion. The samples were processed by two methods (Cazorla et al, 1998). (i) A portion was surface-disinfected by immersion in a sterile aqueous solution of 0.1%

(w/v) HgCl2, rinsed in sterile distilled water, macerated in few droplets of sterile distilled water and plated on King's medium B (KB). (ii) The remainder was homogenized in a lab blender for 3 min with 10 ml of sterile phosphate buffer (0.1 M, pH 7.2) per gram of fresh plant material, and the supernatant was serially diluted in the same buffer, and 0.01-ml portions from each dilution were plated on KB medium supplemented with 100 µg of cycloheximide per milliliter to inhibit fungal growth. Inoculated plates were incubated at 24°C for up 3 days. Plates were examined under long wave (366 nm) ultraviolet light to determine production of fluorescent pigments (King et al 1954). Colonies associated with such pigments were transferred further purified on KB with a series of single-colony transfers and kept on nutrient agar slants at 3C°.

Identification and characterization

Biochemical and physiological tests:

Blue-green fluorescent pigmented strains on KB medium under UV at 366 nm were tested for Gram's stain (Schaad, 1988) and cytochrome oxidase activity (Kovacs, 1956) and the strains gives a negative result for both tests were provisionally identified as *P. syringae* pv. *syringae*.

Selected strains were further characterized by two determinative schemes: (i) LOPAT tests including: levan production, oxidase reaction, potato soft rot, arginine dihydrolase, and tobacco hypersensitivity (Klement, 1963; Lelliott et al, 1966 and Lelliott & Stead 1987) and GATTa tests including: gelatin liquefaction, aesculin hydrolysis, tyrosinase activity and tartrate utilization (Latorre and Jones 1979).

The bacterial isolates were also tested to other biochemical and physiological tests including nitrate reduction; growth on yeast extract-dextrose-calcium carbonate agar; and utilization of glucose, mannitol, inositol, adonitol, sorbitol, gluconate, propionate, , L-lactate, and L-histidine. (Bradbury 1986, Hildebrand et al., 1988, and Lelliott & Stead 1987).

All inoculated media were incubated at 22°C for 3 to 5 days. Duplicate tubes or plates were run for each test, and tests were repeated at least twice.

Ice nucleation activity (INA):

Measuring the ice nucleation activity was determined by the method of Lindow et al. (1978). Cells were suspended to a concentration of approximately 10^8 CFU/ml (OD590 = 0.3) in sterile distilled water. Ten drops (10µ each) of cell suspension were pipetted onto a paraffin coated sheet of aluminum foil floating on a 70% ethanol solution maintained at –5°C in a circulating water-ethanol bath. The number of drops that froze within 10 min was recorded (eight or more frozen drops were considered INA positive). The bacteria were tested three times

Pathogenicity tests :

Pathogenicity on other hosts :

Pathogenicity tests in tomato plants, immature lemon and pear fruits, and bean pods were performed to confirm the diagnosis of the bacterial strains (Lelliott & Stead 1987).These assays were repeated three times for each studied strain, and each assay was carried out on fifteen different leaves, fruits, or pods. The fruits were swabbed with 70% ethyl alcohol and washed in sterile water. Five drops of inoculum (10^6 CFU/ml) were placed on the fruit surface (bean pods, lemon, and pear), which was punctured by pricking through the drop with a sterile needle. The inoculated fruits were incubated at 25°C in closed boxes lined with damp blotting paper. Tomato plants with fully expanded leaves were inoculated with a 20-µl drop of the same bacterial suspension (10^6 CFU/ml) that was deposited on a fresh wound made on the midrib of the leaf, each isolate was inoculated on three leaves on separate twigs. After inoculation, the plants were placed in a growth chamber at 22°C with a 16 h (light)/8 h (dark) photoperiod and covered by transparent polyethylene bags to maintain a high relative humidity. The bags were removed after 48 h (Lelliott et al 1987). The appearance of infected leaves was recorded after a 7-day incubation period. Sterile phosphate buffer were used as control.

Pathogenicity on mango plants:

To test the pathogenicity of bacterial isolates on mango, the test were carried out beginning in 1st December 2005 to 21 March 2006. Buds of 2-year-old mango plants (cv. zebda) growing in pots were inoculated with 10 µl of a 10^9-CFU/ml bacterial suspension in phosphate buffer, and the inoculum was forced into the bud with a microsyringe

(Cazorla et al 1998). Sterile phosphate buffer was used as a control. The trees were maintained under field conditions and during the test, the average temperature was 17°C with a minimum temperature of 8°C, and rainfall totaled 50 mm.

To confirm the presence of *P. syringae* in inoculated tissues, reisolation was carried out from plant buds showing necrotic symptoms after 3 or 6 weeks. The identity of bacterial isolates was verified by Gram staining. fluorescent pigment production, LOPAT, and INA tests, as described above.

RESULTS

Causal agent isolation :

Isolations from diseased mango tissues yielded almost pure cultures of translucent bacterial colonies that were fluorescent on KB under UV light after 72 h of incubation at 24°C. Among both isolation methods, the first one yielded lowest non target colonies with some fungal growth in sometimes. However, in the second method, the number of non target colonies was relatively high but no fungal growth was observed. Twenty isolates representing different localities were chosen to pursue subsequent studies.

Identification, and characterization :

On the basis of diagnostic tests results. all of the 20 selected isolates were identified as *P. syringae* pv. *syringae*. The strains showed similar biochemical and physiological characteristics that were typical of *P. syringae* pv. *syringae* (Table 1). In LOPAT tests, the bacteria were positive for levan and negative for oxidase, arginine dihydrolase and pectolytic activity and produced a rapid hypersensitive reaction on tobacco leaves. In GATTa tests, the bacterial isolates were positive for gelatin liquefaction and aesculin hydrolysis and negative for tyrosinase activity and tartrate utilization. In other biochemical and physiological tests the bacterial isolates were negative for nitrate reduction and gives white colonies on yeast extract-dextrose-calcium carbonate agar (YDC). Thy utilize glucose, mannitol, inositol, adonitol, sorbitol, gluconate, propionate, L-lactate, and L-histidine.

Ice nucleation activity (INA):

All the isolates had ice nucleation activity at –5°C.

Table 1. Physiological and biochemical characteristics identifying the bacterial strains isolated from necrotic lesions in buds and leaves of mango trees growing in 20 different localities of Egypt , from November 2005 to April 2006.

Test	Reaction
LOPAT tests:	
Levan	+
Oxidase	-
Potato soft rot	-
Arginine dihydrolase	-
Tobacco hypersensitivity	+
GATTa tests :	
Gelatin liquefaction	+
Aesculin Hydrolysis	+
Tyrosinase activity	–
Utilization of :L-tartrate	–
Utilization of :	
Glucose	+
Mannitol	+
Inositol	+
Adonitol	+
Sorbitol	+
Gluconate	+
Propionate	+
L-lactate	+
L-histidine	+
Ice nucleation activity	+
Pathogenicity on :	
Tomato	+
Pear fruits	+
Lemon fruits	+
Bean pods	–

+ = positive reaction – = negative

All the strains were rod-shaped Gram-negative bacteria.

Pathogenicity to other hosts:

Bacterial strains isolated from diseased mango tissues induced typical symptoms of *P. syringae* pv. *syringae* on inoculated immature

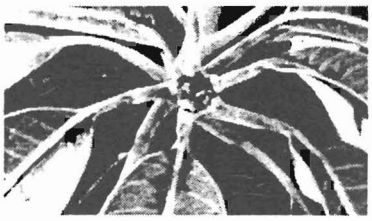
lemon and pear fruits. appearing as deep, black necrotic pits on lemons and dark necrotic spots on pears. Many strains produced water-soaked lesions with a reddish margin on dwarf bean pods, although they did not produce disease symptoms. In leaves of tomato inoculated by pricking. necrotic areas were seen 4 days after inoculation.

Pathogenicity on mango plants.

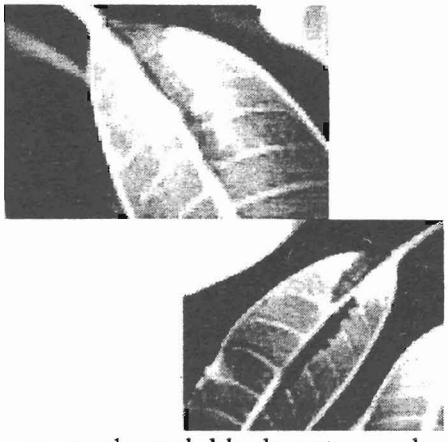
Twenty strains of *P. syringae* pv. *syringae* isolated from diseased mango trees in different locations were individually inoculated onto healthy buds and shoots of mango plants to test for pathogenicity. All strains produced typical symptoms of the disease on inoculated buds and shoots (Fig 1). on inoculated buds caused symptoms of apical necrosis (A). On shoots, the first symptoms appeared 6 to 7 days after inoculation. Lesions on leaves started as interveinal, angular, water-soaked spots (1 to 3 mm in diameter) that coalesce, become black and slightly raised (B). Necrotic lesions frequently extend to the leaf petioles and stem (C). Necrotic symptom development was never observed when buds were inoculated by phosphate buffer. No differences in symptom development were observed between different *P. syringae* pv. *syringae* strains. Bacterial strains were readily reisolated from the inoculated buds that developed typical symptoms. Reisolation yielded fluorescent bacteria with characteristics similar to the inoculated isolates.

DISCUSSION

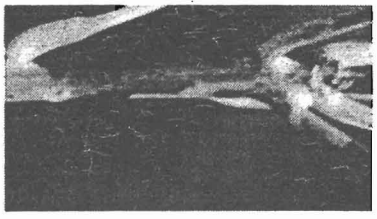
Bacterial apical necrosis is a new disease of mango which characterized by a rapid expansion of necrotic lesions in buds and leaves Symptoms similar to those observed in mango have been reported on other species of fruit trees infected by *P. syringae* pv. *syringae* (Hattingh et al. 1989) such as peach (Endert, and Ritchie, 1984), citrus (Lopez, 1989), cherry (Sundin et al, 1989), almond (Lindow and Connell, 1984), and pear (Montesinos and Vilardell, 1991). A disease of mango showing similar symptoms has been reported in Israel (bacterial black blight), but the causal agent has not been conclusively identified (Pinkas et al. 1996). The disease has been previously reported on mango for the first time in southern Spain(Cazorla et al, 1998). Apical bud necrosis and leaf spotting have been observed in mango trees grown in different localities in Egypt since 2004 - 2005.



A: Necrotic symptom on apical bud



B : coalesced, black water-soaked Lesions on leaves.



C : Necrotic lesions extended to the leaf petioles and stem.

Fig. 1, typical symptoms of bacterial apical necrosis disease on inoculated mango buds and shoots.

Preliminary isolation from diseased tissues of mango with apical necrosis symptoms have shown the presence of bacterial strains resembling fluorescent pseudomonads isolated from diseased mango tissues were conclusively identified as *P. syringae* pv. *syringae* on the

basis of their nutritional and biochemical profiles.

The traditional method for diagnosing diseases incited by *P. syringae* pv. *syringae* involves isolation of the suspected pathogen in pure culture and characterization by a combination of physiological and biochemical tests. The Levan production, Oxidase reaction, Potato soft rot, Arginine dihydrolase, and Tobacco hypersensitive response (LOPAT) tests are used to discriminate *P. syringae* from other species of fluorescent pseudomnads (Lelliott et al, 1966 and Lelliott & Stead 1987). The Gelatin liquefaction, Aesculin hydrolysis, Tyrosinase activity and Tartrate utilization (GATTa) tests are used to separate pathovar *syringae* from other pathovars of *P. syringae* (Latorre and Jones 1979). Characteristics of tested isolates were identical to other reports of *P. syringae* pv. *syringae* (Gardan, et al, 1991; Hildebrand et al, 1988; Palleroni 1984 and Young & Triggs 1994). More rapid identification of *P. syringae* pv. *syringae* has been attempted by measuring the ice nucleation activity (INA) of strains (Lindow et al, 1978; Gross et al, 1984; Baca et al, 1987 and Schaad 1988).

There is no standard method for evaluating pathogenicity in *P.s.* pv. *syringae*. Inoculation of immature pear and sweet cherry fruit (Endert & Richie 1984, Gross et al., 1984 and Quigley & Gross 1994), leaves and twigs of apricot, peach, nectarine and plum, etiolated pear, apple and peach hypocotyls (Endert & Ritchie, 1984), immature yellow tomato fruit (Cameron, 1970 and Canfield et al., 1986), bean pods (Legard & Hunter, 1990), lemon fruit and pea stems (Mazarei & Kerr, 1990), lilac petioles (Young,1991), red maple stems (Malvick & Moore, 1988 a, b) and pear blossoms (Whitesides & Spotts, 1991 and Yassad et al., 1992) have all been tested. The results of all these methods were variability in symptoms expression and low correlation of results from bioassays with physiological or biochemical tests (Heather et al., 1997). The tested 20 bacterial isolates had ice nucleation activity and a host range typical of the pathovar *syringae* (Lelliott & Stead, 1987).

The role of *P. syringae* pv. *syringae* strains in the induction of bacterial apical necrosis of mangoes was demonstrated by the production of typical symptoms following inoculation of the isolates into buds of young mango plants and the subsequent reisolation of *P. syringae* pv. *syringae* from the affected buds . The role of *P. syringae* pv. *syringae* as a causal agent of the apical necrosis in mango trees in Egypt is confirmed for the first time .

Pseudomonas syringae pv. *syringae*, which is a common inhabitant of a large number of plant species, where it may be present in epiphytic or pathogenic association (Hirano and Upper, 1983). The population size and epiphytic growth of mango pathogenic *Pseudomonas syringae* pv. *syringae* remains to be investigated.

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مرض موت القمة البكتيرى المتسب عن

Pseudomonas syringae pv. *syringae*

فى مصر

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الملخص العربى

شوهدت أعراض مرض موت القمة البكتيرى علي أشجار المانجو في موسم ٢٠٠٤ -٢٠٠٥ وبدأت دراسة المرض في موسم ٢٠٠٥-٢٠٠٦ و جمعت العينات المصابة من المناطق الرئيسية لزراعة المانجو في مصر وهي محافظة الإسماعيلية و الشرقية والبحيرة(منطقة النوبارية) و الجيزة و الفيوم.

و كانت الدراسة و نتائجها كما يأتى:-

١- بإجراء العزل من الأنسجة المصابة وذلك على بيئة كينج (Kings medium B) كانت معظم المستعمرات النامية مشابهة من النواحى المورفولوجية لصفات الجنس *Pseudomonas* على هذه البيئة و خاصة إفرازه لصبغة الفلوروسين الذائبة.

2- على أساس إفراز صبغة الفلوروسين و التفاعل السالب لكل من صبغة جرام واختبار oxidase، تم اختيار عشرون عزلة بكتيرية ممثلة لعشرون منطقة من المناطق التى جمعت منها العينات.

3- بدراسة الصفات الفسيولوجية و البيوكيماوية و المدى العوائلى لهذه العزلات وجدت أنها متطابقة فى هذه الصفات مع صفات البكتريوم *Pseudomonas syringae* pv. *syringae* المذكورة فى المراجع المختلفة.

٤- بإجراء اختبار القدرة المرضية لهذه العزلات على أشجار مانجو صغيرة (عمر سنتين) من الصنف زبدة، أعطت أعراضا مشابهة لأعراض المرض الناتجة عن الإصابة الطبيعية.

طبقا للمراجع المتاحة يعتبر هذا هو أول وصف لمرض موت القمة البكتيرى فى المانجو ومسببه فى مصر.