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in

Myrta A. (ed.), Di Terlizzi B. (ed.), Savino V. (ed.).
Production and exchange of virus-free plant propagating material in the Mediterranean region

Bari : CIHEAM

Options Méditerranéennes : Série B. Etudes et Recherches; n. 35

2001

pages 37-42

Article available on line / Article disponible en ligne à l'adresse :

<http://om.ciheam.org/article.php?IDPDF=2002216>

To cite this article / Pour citer cet article

Myrta A., Boscia D. **Plum Pox Virus: a Risk for the Mediterranean Fruit Tree Industry.** In : Myrta A. (ed.), Di Terlizzi B. (ed.), Savino V. (ed.). *Production and exchange of virus-free plant propagating material in the Mediterranean region.* Bari : CIHEAM, 2001. p. 37-42 (Options Méditerranéennes : Série B. Etudes et Recherches; n. 35)



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Plum pox virus: a risk for the Mediterranean fruit tree industry

A. Myrta¹ and D. Boscia²

Introduction

Plum pox virus (PPV), causal agent of Sharka, is one of the most severe pathogens of stone fruit trees in Europe (Dunez and Sutic, 1988) with recent introduction to the Mediterranean area (Roy and Smith, 1994).

Geographic distribution

Sharka was first observed in plums in Bulgaria between 1915 and 1918. Between 1932 and 1960 the disease moved north and east from Bulgaria into Yugoslavia, Hungary, Romania, Albania, Czechoslovakia, Germany and Russia. Following World War II Sharka progressed into western Europe reaching Germany and Austria by the late 1950's. By the mid-60's, Sharka had reached The Netherlands, Switzerland, Greece, England and Turkey; France, Italy, and Belgium by the early 70's; Spain and Portugal by the early 80's; Egypt, Syria, and Cyprus by the late 80's (Table 1). In the 90's it started to invade also other continents (Chile in 1992; India in 1994; USA and Jordan in 1999 and Canada in 2000).

The situation in the Mediterranean is heterogeneous: in the Balkans the disease was distributed early and the infection level is high, in other Northern Mediterranean countries PPV is distributed but heterogeneously, whereas in many South Mediter-

1 Istituto Agronomico Mediterraneo di Bari (Italy)

2 Centro Studi del CNR sui Virus e le Virosi delle Colture Mediterranee, Bari (Italy)

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anean countries PPV is not reported (Roy and Smith, 1994).

Table 2 reports the recent status of PPV in the Mediterranean elaborated by Levy *et al.* (1999).

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Table 1. Reports of PPV in the Mediterranean

First occurrence	Country	First report
1917	Bulgaria	Atanassoff (1932)
1935	Ex-Yugoslavia	Josifovic (1937)
1947	Albania	Papingji (1965)
1967	Greece	Demetriades and Catsimbas (1968)
1968	Turkey	Sahtiyanci (1969)
1970	France	Devignes and Morvan (1970)
1973	Italy	Canova <i>et al.</i> (1977)
1984	Spain	Llácer <i>et al.</i> (1985)
1984	Portugal	Louro and Corvo (1985)
1987	Egypt	Dunez (1988)
1987	Syria	Dunez (1987)
1987	Cyprus	Dunez (1987)
1999	Jordan	Al Rwahnih <i>et al.</i> (2000)

Table 2. PPV status in the Mediterranean

Disease Status	Country
Widespread (high level)	Croatia, Greece, Former Yugoslavia;
Restricted distribution (heterogeneous levels)	Slovenia, Albania, Cyprus, France, Italy, Portugal, Spain, Syria, Turkey;
Introduced, Established	Egypt, Jordan;
Not present	Israel, Lebanon, Malta, Morocco, Palestine, Tunisia;
Unknown status	Libya

Virus detection and strain identification

To date, PPV falls in four groups, differentiated molecularly and serologically as PPV-Marcus (PPV-

M), PPV-Dideron (PPV-D), PPV-Cherry (PPV-C) and PPV-El Amar (PPV-EA). Individual isolates within each strain/serogroup may vary biologically.

PPV-D, originally isolated from apricot in south-eastern France, is the most common strain of the virus in western Europe. Apricot, peach, and plum are the natural *Prunus* hosts of the D strain. This strain is less efficiently aphid-vectored, and is the non-epidemic strain of PPV.

PPV-M, originally isolated from peach in northern Greece, is the most common strain of the virus in southern, eastern, and central Europe. Peach is the main natural *Prunus* host, however, apricot and plum are susceptible. PPV-M is spread rapidly by aphids and is considered to be the epidemic strain of the virus.

PPV-EA, originally isolated from apricot in Egypt, where it is still restricted. Although little information is available for the EA strain, some characteristics of PPV-EA are similar to the M strain.

PPV-C, is originally isolated from sour cherry from Moldova. The natural *Prunus* host range of PPV-C is limited to sweet and sour cherry. PPV-C is transmitted efficiently by aphids, and has a wider experimental host range than other PPV strains. This strain has been reported in eastern and central European countries, and Italy. However, after the eradication of the Italian outbreak, it seems not to be present in the Mediterranean any longer.

It is important to have of simple diagnostic tools suitable for both the timely discovery of PPV foci and reliable strain identification. RFLP analyses of the PCR-amplified cDNA fragment (Wetzel *et al.*, 1991) RT-PCR assay based on strain-specific primers (Candresse *et al.*, 1998) and the differences in electrophoretic mobility of dissociated coat protein (CP) are used to type PPV isolates. Monoclonal antibodies (MAbs) with serotype-specific reactivity are already available for this purpose (Myrta *et al.*, 1998).

Epidemiology and control

PPV is mainly transmitted by planting infected material. As PPV is transmitted by several aphid species common in all fruit growing areas, in a not persistent manner, natural infections can occur. Chemical insect control has a limited efficiency in controlling the disease spread. Nevertheless, it can contribute to slowing down disease spread where present and therefore should not be neglected.

Preventing the entry of PPV and successive spread to a new area or country is the best way for its control. Severe controls regarding the importation and movement of propagating materials, indexing of imported germplasm in quarantine, production of virus-free propagating material in the frame of a certification program are of main importance for the virus control.

When the disease is still limited in its distribution, eradication of infected trees and/or orchards is an effective control strategy.

Breeding for resistance seems to be the only way to control Sharka efficiently where it is already widespread. Unfortunately, natural sources of resistance are rare, and breeding programmes need interspecific crosses to introduce natural resistant genes. Recently, pathogen-derived resistance has been experimented for PPV in transgenic plum and apricot lines expressing PPV genome sequences. Some show a high degree of resistance or immunity after artificial inoculation and now are being evaluated under field conditions. However, it is not still a practical way to control the disease.

Conclusions

In the Mediterranean basin PPV spread is relatively recent and there is a high risk of further spread. As several countries in the region are implementing certification schemes for stone fruits as important Mediterranean crops, the spread of disease can be a real risk for the virus-free production of propagating material.

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