PAPAYA RINGSPOT VIRUS

Introduction

Papaya ringspot virus (PRSV) disease occurs in nearly every region where papaya (*Carica papaya)* is grown except for Africa (Gonsalves, 1993). PRSV was first reported in *Carica papaya* by Jensen, 1949. It was in the 1970s and 1980s when PRSV caused disasters to papaya production in much of the South and South-East Asia. Since then the disease has continued to spread steadily, causing large yield losses.

Identity

Classification

Genome	: (+) ssRNA
Family	: Potyviridae
Genus	: *Potyvirus*
Synonyms	: Papaya distortion mosaic virus, Papaya leaf distortion virus, Papaw distortion ringspot virus, Papaw mosaic virus, Watermelon mosaic virus 1
Role	: Pest
Strains	: Type p, Guadeloupe Papaya ringspot, Papaya ringspot-type W has been shown to be the Water melon mosaic virus 1 potyvirus.

Signs & Symptoms

The earliest symptoms on papaya are a yellowing and vein-clearing of the young leaves. This is followed by a very conspicuous yellow mottling of the leaves and sometimes severe blistering and leaf distortion. Dark green streaks and rings also appear on the leafstalks and stems (Heu et al.2002) (**Figure 1).**

Symptoms induced by different isolates of PRSV–p may vary in intensity, but are dark green and the rings, often slightly sunken in the fruit, are diagnostic. Fruits often show uneven bumps, especially those fruits that develop after a tree is infected. The disease derives its name from the striking symptoms that develop on fruit. The number of rings in fruits can be variable, and the rings become less distinct as the fruit matures and yellows. The symptoms on the fruit consist of concentric rings and spots or C-shaped markings, a darker green than the background-green fruit colour. **(Figure 2**).

Vigour of trees and fruit set are usually reduced depending on the age of the plant when infected. Fruit quality, particularly flavour, is also adversely affected. Other key symptoms of PRSV (P-strain) are intense yellow mosaic on leaf laminae and numerous "oily" streaks on petioles (Persley, 1999). The leaf canopy becomes smaller as the disease progresses due to the development of smaller leaves and stunting of the plant. Leaf and fruit symptoms are most intense during the cool season. Leaves often develop a shoe-string appearance caused by the extreme reduction of leaf laminae similar to that caused by broad mites (Heu et al. 2002)**.**

Papaya trees of all ages are susceptible and generally will show symptoms two to three weeks after inoculation. Trees infected at a very young stage never produce fruits but rarely die because of the disease. There are, however, some isolates in Taiwan which cause wilting and sometimes death of young trees.

Morphology

Brunt et al.(1996)

Papaya ringspot potyvirus, the genome of which contains single stranded RNA (ssRNA), is a long flexuous rod about 760-800 nm long x 12nm wide. The Genome is unipartite. The particles (virions) contain 5.5% nucleic acid and 94.5% protein.

Biology & Epidemiology

Bateson et al. (1994)

Papaya ringspot is grouped into two types - PRSV-type p infects papaya and cucurbits, and PRSV-w infects cucurbits but not papaya. Analysis of the coat protein genes of isolates from Australia, Thailand and the USA indicates that in each country Papaya ringspot – p isolates have evolved independently from the Watermelon mosaic virus 1 isolates.

Dispersal / Vectors

Goncalves (1993)

The PRSV can be rapidly spread by several aphid species e.g.Myzus *persicae*, *Aphis gossypii* in a non persistent manner. Therefore, the spread of the virus (PRSV-p) within an orchard is primarily from tree to tree. The virus can also spread by the movement of infected papaya plants and cucurbit seedlings.

Though many cucurbits are susceptible to PRSV-p, they do not serve as important alternate hosts. Instead, the dominant strain in cucurbits is PRSV-w. An aphid is capable of transmitting the virus and it only takes a few seconds of feeding time to acquire the virus into its mouthparts. It is then able to transmit the virus to other plants during brief feeding probes. Papaya ringspot virus is not spread by other insects and it does not survive in soil or dead plant material. Once infected, plants cannot be cured by spraying with pesticides or removing plant parts showing symptoms. There is no evidence that PRSV can be transmitted through seeds from infected papaya or cucurbits.

The development of the disease in an orchard follows the general pattern of viruses that are spread by aphids in a non-persistent manner. The amount of primary infection increases as the distance from infected papaya trees decreases. Secondary infection spreads rapidly and an orchard can become totally infected in three to four months. This situation occurs in young orchards located close to infected plants and during periods when populations of winged aphid flights are high.

Management

Efforts to control papaya ringspot in papaya have included roguing, breeding for tolerance to PPV-p, cultural practices and cross protection. None of these methods individually provides ideal control of the disease. An Integrated Pest Management System within an overall Integrated Crop Management programme would more likely achieve not only effective and sustained disease management but also allow the deployment of environmentally compatible measures. Most parts of the Caribbean that have not yet reported PRSV should strictly apply exclusion tactics through Plant Quarantine Laws and Regulations of these virgin countries to prevent and /or at least delay spread of this destructive virus on papaya. For example in Hawaii PRSV-p has been kept out of the Puna district where nearly 98% of the state's commercial papaya is produced. Their success is due to the combined practices of regular monitoring and roguing of infected papaya by the State Department of Agriculture in the surrounding areas, and discouraging the movement of seedlings into the Puna District.

Other important cultural practices in reducing crop losses are the establishment of orchards with seedlings that are not infected with PRSV-p. Newly cultivated orchards should be situated as far as feasible from infected orchards. Orchards should not be established by interplanting seedlings among trees that are infected with PRSV-p. The practice of cultivating non-host crops, such as corn, around the orchard is commonly used and even between rows. The rationale for this is that aphids flying into the papaya orchard would first land and feed on the alternate crop and lose their ability to transmit the virus to papaya due to the non-persistent mode of transmission by aphids.

The use of tolerant varieties to PRSV-p developed in both programmes in Florida and Taiwan has been successful. Tolerance is obtained from a dioecious papaya variety i.e. having male and female flowers on separate plants. The tolerant character is polygenic and is inherited quantitatively. These tolerant varieties are susceptible to PRSV-p but the fruit and leaf symptoms are milder and infected trees produce reasonable quantities of fruit. Hawaiian solo type papaya is not tolerant to PRSV-p.

Cross protection has been used to manage the PRSV disease with varying degrees of success. The underlying principle includes inoculating seedlings with a mild strain prior to planting the trees out into orchards. A nitrous acid mutant of a PRSV strain from Hawaii has been used extensively. It seems the success of cross protection depends on environmental factors. For example, cross protection is highly successful in Hawaii, moderately successful in Taiwan,and not successful in Thailand. In Taiwan, the practice of cross protection has been applied to thousands of trees every year since 1984. Cross protection is most effective when protected orchards are isolated from severely infected trees and when roguing is done prior to the flowering stage.

Breeding for resistance in *Carica papaya* is done by conventional breeding techniques using interspecific hybridization with resistant *Carica spp*. and then followed by embryo rescue. Papaya plants resistant to PRSV have been produced by incorporating the protein gene of the virus, using genetic engineering techniques.
However this generation of genetically transformed papaya plants was found only resistant to the Hawaii strain. This technique still holds much hope for Jamaica and the

rest of the Caribbean, in the development of resistant papaya varieties to this singly and most destructive virus of papaya, worldwide.

Pest Significance and Phytosanitary Risk

PRSV is of major concern in South East Asia and so far is of minor significance in the Caribbean region.

Host Notes

Brunt et al. (1996)

Diagnostically susceptible host species and symptoms:

- *Carica papaya* – symptoms variable; vein clearing then mottled and malformed leaves with smaller lobes:
- *Cucurbita pepo* – some isolates give mosaic and leaf malformation but others induce mild mottle (Gonsalves and Ishii, 1980).
- *Cucumis metuliferus* cv.accession 2459)-systemic mottling or mosaic

Susceptible host species

- *Carica papaya*
- *Chenopodium amaranticolor*
- *Chenopodium quinoa*
- *Cucumis sativus*
- *Cucurbita maxima*
- *Cucurbita moschata*
- *Cucurbita pepo*

Insusceptible host species

- *Carica papaya*
- *Cucumis metuliferus*
- *Myosotis sylvatica*
- *Nicotiana bethamiana*

Distribution

South and South East Asia, Australia, India, Sri Lanka, Hawaii, Texas, Florida (USA), South and Central America, Trinidad & Tobago, Jamaica in two parishes only St. Mary and St. Catherine.

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Figure 1: Dark green streaks on leaf stalks and leaves

(Picture taken from D Persley Queensland Horticulture Institute, Australia)

3

Figure 2: Concentric rings spots and C' markings on fruit infected with PRSV
(Picture taken from D Persley Queensland Horticulture Institute, Australia)