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REVIEW ON BIOLOGICAL AND GENOMIC DIVERSITY OF *PAPAYA RING SPOT VIRUS*

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Abstract: *Papaya is an important fruit crop grown in tropical and subtropical low land regions. One factor significantly reducing the production of papaya is the damage or loss of crops to disease. Cultivated papaya (Carica papaya L.) has a narrow gene pool that may be partially responsible for the susceptibility of the papaya to many diseases. The range of pathogens causing diseases in papaya is diverse and includes viruses, bacteria, fungi and fungi-like pathogens, phytoplasma and nematodes. About a dozen different viruses are reported to limit papaya production in different parts of the world. Of these, Papaya mosaic and Papaya ringspot viruses appear to be most widely distributed whereas other viruses have a restricted distribution. In India also, Papaya ringspot virus (PRSV) is the most commonly occurring virus followed by Papaya leaf curl virus, which has a limited distribution.*

Key words: *Papaya, papaya ring spot virus, genetic divercity.*

Introduction

The papaya ring spot virus was first discovered in 1945 (Lindner *et al*., 1945) and the term "*Papaya ringspot virus*" (PRSV) was coined by Jensen in 1949 (Jensen, 1949). The name of the disease has been derived from the ringed spot symptoms on the fruit.

Distribution of PRSV is widespread throughout the world, including United States, South America, the Carribbean countries, India, Taiwan, Africa, Japan, Australia, Srilanka, Mexico and other countries (Silva-Rosales *et al*., 2000; Parameswari, 2009). PRSV pathotype W occurs wherever cucurbits are grown (Purcifull *et al*., 1984); Pathotype P had devastating effects on the papaya industry around the world. In Hawaii, Pathotype P became widespread on Oahu in the early 1950's and has since spread to all commercial papaya growing regions (Gonsalves, 1998). The virus has drastically affected papaya production in severely infected regions, resulting in loss of income for farmers. Pathotype P also destroyed most of the commercial plantations on the west coast of Southern Taiwan within four years after it was first recorded in 1975 and production dropped by 60% in this period (Yeh *et al*., 1988).

Like other parts of the world, PRSV in India is the most destructive virus on papaya. The disease has been described differently by different workers, such as mosaic (Marathe and Samanavar, 1984); leaf reduction (Singh, 1969) and distortion ringspot (Khurana, 1974). In India, natural infection of PRSV was first reported by Capoor and Varma (1958) from Maharashtra. Subsequently, it has been reported from Rajasthan (Surekha *et al*., 1977) and Uttar Pradesh (Khurana, 1974). Now the virus seems to be widespread and occurs wherever papaya is grown (Bag *et al.,* 2007). More than 95% incidence of PRSV has been recorded or even up to 100% in some states. The disease could affect over 90% of the grown up plants, reducing latex and sugar contents. Although there are no reliable loss estimates caused by PRSV, the disease has appeared in epidemic proportions in some regions and the papaya cultivation has been reduced to an annual crop and to kitchen gardens.

Viral taxonomy

Papaya ringspot virus (PRSV) belongs to the family *Potyviridae* and the genus *Potyvirus*. Virion is non-enveloped, flexous (780×12 nm) consisting of positive sense, single stranded RNA of about 10 Kb

(Purcifull *et al*., 1984; Yeh and Gonsalves, 1985). The genome is surrounded by a single coat protein of 32-36 kD (Gonsalves and Ishii, 1980).

Biological diversity

Like other potyviruses (Shukla *et al*., 1994), PRSV has a narrow host range restricted to three dicotyledonous families. Two pathotypes, which are serologically indistinguishable, have been recognized based on the host range - P and W. Pathotype P infects plant species belonging to three families (Caricaceae, Chenopodiaceae and Cucurbitaceae), while pathotype W infects Chenopodiaceae and Cucurbitaceae. pathotype W is non-infectious to the species of family Caricaceae (Purcifull *et al*., 1984; Brunt *et al*., 1996). Both pathotypes P and W are reported to occur in India. Host range of pathotypes P and W was restricted to Caricaceae and/or Cucurbitaceae (Roy, 2000). Similarly pathotype P from Delhi and Uttar Pradesh, Jharkhand and West Bengal were also restricted to papaya (Roy, 2000). In another sudy, pathotype P from Maharashtra, Uttar Pradesh and Rajasthan exhibited wider host range infecting papaya and cucurbits (Yemewar and Mali, 1980; Roy, 2000). Prasad and Sarkar (1989) have reported that the virus can infect members of Chenopodiaceae. Shaikh (1996) and Lakshminarayana Reddy (2000) also reported natural infection of pathotype P on a wide range of cucurbits *viz*,. *Lagenaria siceraria*, *Citrullus lanatus*, *Luffa acutangula*, *Cucumis sativus*, *Cucumis melo*, *Cucurbita moschata*, *Cucumis anguira*, *Benincasa hispida* and *Cucurbita pepo*. Possibility of mixed infection of pathotypes P and W in plants belonging to family Cucurbitaceae exists under natural condition. Recently mixed infection of pathotypes P and W infecting bottlegourd (*Lagenaria siceraria*) from Maharashtra was recorded (Mantri *et al*., 2005). Similarly, in another study natural infection of pathotype W from Maharashtra was recorded on sponge gourd (*Luffa cylindrica*). Pathotype W produced systemic symptoms on *Cucumis melo*, *Cucumis sativus*, *Cucurbita maxima*, *Cucurbita pepo*, *L. acutangula*, and *L. cylindrica*, and was non- infectious to *Carica papaya* (cvs. CO2 and Red Lady) (Verma *et al*., 2006).

Genome organization and diversity

Potyviruses belong to the picorna-like supergroup of viruses whose RNAs have a protein (VPg) covalently bound to the 5'-end of the genome, a poly(A) tail at the 3' end and whose genomes are expressed as a large polyprotein which is subsequently cleaved co-and/or post translationally by proteases to yield functional proteins (Dougherty and Carrington, 1988). These include conserved set of genes encoding nonstructural proteins that are involved in RNA replication. The order of the proteins within the polyprotein is: first protein (P1), helper component (HC-Pro), third protein (P3), cylindrical inclusion protein (CI), small nuclear inclusion protein (NIa), which includes an N-terminal VPg and C-terminal protease domain, large nuclear inclusion protein (NIb) and coat protein (CP). Small, 6Kd, proteins are located between the CI and NIa and between the P3 and CI in some potyviruses. Both the VPg and the CP are found in virions, whereas the P1, HC-Pro, P3, CI, NIa and NIb proteins are detected in infected plants (Rodriguez-Cerezo and Shaw, 1991). Comparison of potyviral sequences indicate that genes encoding P1, P3 proteins and the N terminal region of CP exhibit the greatest sequence variation. The translated proteins of these genes are speculated to function in specific host-virus interactions (Shukla *et al*., 1994). Other protein sequences such as the CP exhibit intermediate levels of conservation, and sequences can diverge by 12% between isolates (Gonsalves, 1998). Most of these proteins have been shown to be multifunctional while all the proteins are involved in genome amplification (Urcuqui-Inchima *et a*l., 2001).

PRSV: Genome organization

PRSV genome organization is similar to other members of the *Potyvirus* genus (Yeh *et al*., 1992; Wang and Yeh, 1997). The complete genome of fifteen PRSV isolates, one each from China (Lu *et al*., 2008, GenBank Acc. No: EF 183499), Mexico (Noa-Carrazana *et al.,* 2007, GenBank Acc. No: AY231130), South korea (GenBank Acc. No: AB369277), two each from, Brazil (Inoue-Nagata *et*

al., 2007, W isolates, WC - GenBank Acc. No: DQ374152, W1 - DQ374153) and Thailand (one P isolate (AY162218**)** and one W isolate (AY010722), three from Hawaii (Yeh *et al.,* 1992, GenBank Acc. No: X67673, NC001785, EU126128), and five P isolates from Taiwan (four P isolates - X97251, DQ340769, DQ340770, DQ340771 and one W isolate - AY027810) are already available in GenBank. The viral RNA of PRSV (9-10 kb) is relatively large compared to other potyvirus sequences (Shukla *et al*., 1994), although SPFMV is larger (10.8 kb). 5'UTR (85 nt), is shorter compared to many other potyvirus 5'UTRs (Yeh *et al*., 1992). PRSV has a relatively long P1 coding region compared to other potyviruses (Wang and Yeh, 1997). The P1 protein appears to be the most variable potyviral protein and shows a wide variation in size (from 29K-63K) among reported potyviruses (Yeh, 1994). Between the P1 proteins of Taiwanese and Hawaiian PRSV isolates, there is only 70.9% nucleotide identity and 66.7% amino acid identity (Wang and Yeh, 1997). This high level of variability is also seen within isolates from a particular country. Charoensilp *et al*., (2003) reported that PRSV pathotypes P&W from the same geographical location were more divergent at the P1 protein level as compared to the other proteins and full genome polyprotein level. The amino acid sequence divergence inPRSV-Thai pathotypes at the P1 protein was 18%. Next to the P1 protein, the P3 is the least conserved between PRSV and other potyviruses suggesting that the function of these proteins may be more virus specific (Yeh and Gonsalves, 1994). Comparative sequence analysis of PRSV–YK (Taiwan isolate) and PRSV-HA (Hawaii) isolates revealed that except P1 protein which shared only 70.9% and 66.7% identity at nucleotide and amino acid levels respectively, the other proteins showed high degree of identity at both nucleotide (82.5-92.3%) and amino acid levels (91.2%-97.6%), (Y Though sequence analysis of viral genomes of PRSV P and W pathotypes from Thailand suggests that the pathotype P type arose locally from pathotype W, yet exact differences between pathotypes P and W to account for host specificity have not yet been discovered (Charoensilp *et al.,* 2003). Characterization of CP and/or 3'UTR has been reported for several PRSV isolates (Quemada *et al.,* 1990; Wang and Yeh, 1992; Bateson *et al.,* 2002). Studies have shown that differences in the CP sequence among PRSV strains were located mostly in the N terminus and include differences in number of amino acids as has been demonstrated in other potyviruses (Shukla *et al.,* 1994). This variation in the CP of PRSV is related to the difference in geographic origin rather than host specificity (Wang and Yeh, 1997; Bateson *et al.,* 2002). With PRSV, most studies have focused on examining sequence variation in CP region. Initial data from the USA and Australia (Bateson *et al*., 1994; Quemada et al., 1990) suggested that there was little sequence variation within these countries. However, recent sequence data on CP genes from PRSV isolates from India (Bag et *al*., 2007) and Mexico (Silva- Rosales *et al*., 2000) suggest greater sequence variation among local populations. Reported CP sequence diversity at the amino acid (10%) and nucleotide (14%) levels were highest among the Asian populations of PRSV isolates (Jain *et al*., 2004). Interestingly, this variation is considerably less than that found among isolates of other potyviruses, such as *Yam mosaic virus* (YMV), where the nucleotide sequence diversity was reportedly as high as 28%. The PRSV isolates collected within India were as different from each other in CP gene sequence (0–11%) as they were to the CP gene sequences of isolates collected in Bangladesh (9–11%), other Asian countries (4–14%), Australia (5–11%) and the Americas (5–11%). A study by Bateson *et al*. (2002) also described the high levels of diversity in PRSV isolates from the Indian subcontinent and proposed that they probably represented the oldest population of PRSV, and based on their basal position according to phylogenetic analysis, the origin of PRSV might have been South Asia (Yeh *et al.,* 1992; Wang and Yeh, 1997).

References:

Bag, S., Agarwal, S. and Jain, R.K. 2007. Sequence diversity in the coat proteins of Papaya *ringspot virus* isolates originating from different locations in India. *Indian Phytopath* **60:** 244-250.

Bateson, M.F., Henderson,J., Chaleeprom,W., Gibbs, A.G. and Dale, J.L. 1994. *Papaya ringspot potyvirus*: isolate variability and the origin of PRSV type Australia. *J Gen Virol* **75**: 3547-3553.

Bateson, M.F., Lines R.E., Revill, P., Chaleepron, W., Ha, V.C., Gibbs, A.J. and Dale, J.L. 2002. On the evolution and molecular epidemiology of potyvirus *Papaya ringspot virus*. *J Gen Virol* **83:** 2575-2585

Brunt, A.A., Crabtree, K., Dallwitz, M.J., Gibbs, A.J. and Watson, L. 1996. Viruses of plants. Descriptions and lists from the VIDE database. CAB International, Wallingford, UK. pp 1484.

Charoenslip, G., Attasart, P., Juricek, M., Panyim, S. and Kertbundit, S. 2003.Sequencing and characterization of Thai *Papaya ringspot virus* isolate type PRSVthP. *Science Asia* **29:** 89-94.

Gonsalves, D. 1998. Control of papaya ringspot virus in papaya: a case study. *Ann Rev Phytopathol* **36:** 415-437.

Gonsalves, D. and Ishii, I., 1980. Purification and serology of papaya ringspot virus. Inoue-Nagata, A.K., Franco, C.D.M., Martin, D.P., Rezende, J.A.M., Ferreria, G.B., Dutra, L.S. and Nagata, T. 2007. Genome analysis of a severe and a mild isolates of Papaya ringspot virus – type W found in Brazil. *Virus Genes* **35:** 119-127.

Jain, R.K., Sharma, J., Sivakumar, A.S., Sharma, P.K., Byadgi, A.S., Verma, A.K. and Varma, A. 2004. Variability in the coat protein gene of papaya ringspot virus isolates from multiple locations in India. *Arch Virol* **149:** 2435-2442.

Jensen, D.D. 1949. Papaya virus diseases with special refernce to Papaya ringspot. Phytopathology 39: 191-211.

Khurana, S.M.P. 1974. Studies on three virus diseases of papaya in Gorakhpur. India. Abstract Pro XIX Int Hort Cong, Warszawa, Poland. **7:** 260.

Lakshminarayan Reddy, C.N. 2000. Studies on *Papaya ringspot virus* disease on papaya (*Carica papaya L.*). M.Sc (Agri) Thesis, University of Agricultural Sciences, Bangalore.

Lindner, R.C., Jensen, D.D. and Ikeda, W. 1945. Ringspot: New papaya plunderer. Haw Farm and Home 8: 10-14.

Lu, Y.W., Shen, W.T., Zhou, P., Tang, Q.J., Niu, Y.M., Peng, M. and Xiang, Z. 2008. Complete genomic sequence of a *Papaya ringspot virus* isolate from Hainan island, China. *Arch Virol* **153:** 991-993.

Marathe, T.S. and Samanavar, A.A. 1984. Detection of papaya mosaic virus in different plant parts of papaya. *Indian Phytopath* **37:** 704-706.

Noa-Carrazana, J.C., Gonzalez-De-Leon, D., and Silva-Rosales, L. 2007. Molecular characterization of a severe isolate of papaya ringspot virus in Mexico and its relationship with other isolates. *Virus Genes* **35:** 109-117.

Parameswari, B. 2009. Genome organization of *Papaya ringspot virus* isolates from India and their biological characterization. PhD Thesis, IARI, New Delhi.

Prasad, S.M. and Sarkar, D.P. 1989. Some ecological studies on Papaya ringspot virus in Ranchi. *Ind J Virol* **5:** 118-122.

Purcifull, D.E., Edwardson, J.R., Hiebert, E. and Gonsalves, D. 1984. Papaya ringspot virus. CMI/AAB Description of plant viruses. No. 292 Wallingford, UK: CAB International.

Quemada, H., L'Hostis, B., Gonsalves, D., Reardon, I.M., Heinrikson, R., Seiu, L.C. and Slightom, J.L. 1990. The nucleotide sequence of the 3'-terminal regions of papaya ringspot virus strains W and P. *J Gen Virol* **71:** 203-210.

Rodriguez-Cerezo, E. and Shaw, J.G. 1991. Two newly detected non structural viral proteins in potyvirus-infected cells. *Virology* **185:** 572-579.

Roy, G. 2000. Characterisation of papaya ringspot potyvirus and its management through cross protection. Ph.D. Thesis, IARI, New Delhi.

Shukla, D.D., Ward, C.W. and Brunt, A.A. 1994. The potyviridae. CAB International, Wallingford, UK, 516 pp.

Silva-Rosales, L., Becerra-Leor, N., Ruiz-Castro, S., Teliz-Ortiz, D. and Noa CarrazanaJ.C. 2000. Coat protein sequence comparisons of three Mexican isolates of papaya ringspot virus with other geographical isolates reveal a close relationship to American and Australian isolates. *Arch Virol* **145:** 835-843.

Surekha, S.K., Mathur,K. and Shukla, D.D. 1977. Virus diseases of papaya(*Caricapapaya*) in Udaipur. *Indian J Mycol Plant Path* **7:** 115-121.

Urcuqui-Inchima, S., Haenni, A. Bernardi, F. 2001. Potyvirus proteins: a wealth of functions. *Virus Research* **74:** 157-175.

Verma, R., Baranwal, V.K., Prakash, S.P.S, Pant, R.P. and Ahlawat, Y.S. 2006. First report of *Papaya ringspot virus* W in sponge gourd from India. *Plant Disease* **90:** 974.

Wang, C.H. and Yeh, S.D. 1992. Nucleotide sequence comparison of the 3'terminal regions of severe, mild and non-papaya infecting strains of papaya ringspot virus. *Arch Virol* **127:** 345-354.

Wang, C.H. and Yeh,S.D. 1997. Divergence and conservation of the genomic RNAs of Taiwan and Hawaii strains of papaya ringspot virus potyvirus. *Arch virol* **142:** 271-285.

Yeh, S.D. 1994. Comparison of the genetic organisation of papaya ringspot virus with other potyviruses. *Plant Pathology Bulletin* **3:** 54-64.

Yeh, S.D., Gonsalves, D., Wang, H.I., Namba, R. and Chiu, R.J. 1988. Control of *Papaya ringspot virus* by cross protection. *Plant Disease* **72:** 375-380.

Yeh, S.D., Jan, F.J., Chiang, C.H., Doong, T.J., Chen, M.C., Chung, P.H. and Bau, H.J. 1992. Complete nucleotide sequence and genetic organization of papaya ringspot virus RNA. *J Gen Virol* **73:** 2531-2541.

Yemewar, S.I. and Mali, V.R. 1980. On the identity of a sap transmissible virus of papaya in Marathwada. *Indian J Mycol Plant Pathol* **10:** 155-160.