

Effect of *Papaya ringspot virus* watermelon strain on growth, yield and quality of melon

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ABSTRACT

Papaya ringspot virus watermelon strain (PRSV-W) is a member of the genus *Potyvirus* that infects Cucurbitaceae crops. The first occurrence of PRSV-W in Indonesia was reported in Bali in 2022. However, information regarding yield loss caused by PRSV-W infection in melon has not yet been reported. This study aimed to analyze yield loss and changes in fruit quality of melon resulting from PRSV-W infection. The research methods included individual and population level disease observations, assessment of agronomic variables and disease severity, yield loss estimation, and data analysis. Observations were conducted at the Pegok Experimental Farm, Faculty of Agriculture, Udayana University, covering an area of 1000 m². The observed plant ages ranged from 6 to 66 days after planting (dap). Mosaic disease development was observed from 18 dap until before harvest. The highest disease incidence and disease severity recorded at the end of the observation period were 58.4% and 61.8%, respectively. Actual yield loss, calculated by comparing mosaic symptomatic and asymptomatic melon plants, reached 49.45%. The most prominent quality defects were changes in fruit color and shape and fruit malformation, with values of 14.06% and 17.5%, respectively. The greatest economic loss occurred in plots with an AUDPC value of 751, resulting in a total yield loss of IDR 15,721,500. Yield loss showed strong correlations with disease incidence and disease severity, with correlation values of 95.41% and 96.19%, respectively. PRSV-W infection altered fruit skin color from yellow-orange to pale yellow with ringspot symptoms, and PRSV-W was detected in the skin tissues of infected fruits. In addition, PRSV-W infection reduced fruit sweetness to 9–12 °Brix, whereas healthy fruits exhibited sweetness levels of 14–17 °Brix.

Keywords: Bali isolate, cucurbitaceae, *Potyvirus*, PRSV-W, yield loss

INTRODUCTION

Melon is one of the crops whose resistance to pests and diseases is strongly influenced by climate change. Under changing climatic conditions, vulnerable melon crops are infected more rapidly by pathogens, including plant viruses. Moreover, virus-infected crops become highly susceptible to secondary infections by other pathogens (Singh et al., 2023). To date, there have been no reports of melon varieties in Indonesia possessing virus-resistant traits (Daryono & Natsuaki, 2009; Listihani et al., 2018; Selangga et al., 2021).

Several viruses are known to infect melon, including *Cucumber green mottle mosaic virus* (CGMMV), *Cucumber mosaic virus* (CMV), *Kyuri*

green mottle mosaic virus (KGMMV), *Papaya ringspot virus* papaya strain P (PRSV-P), *Papaya ringspot virus* strain W (PRSV-W), *Squash mosaic virus* (SqMV), *Tobacco mosaic virus* (TMV), *Watermelon mosaic virus* (WMV), and *Zucchini yellow mosaic virus* (ZYMV) (Wang et al., 2017; Roques et al., 2021; Maachi et al., 2022; Maina et al., 2017a; Listihani et al., 2018; Maina et al., 2018; Damayanti et al., 2022). According to Maina et al. (2017b), PRSV-W has been detected infecting melon, watermelon, and pumpkin in Timor Leste and Australia.

Reports on the major viruses infecting Cucurbitaceae, particularly melon crops, in Indonesia remain limited. PRSV-P infection was first reported in 2012 in Nangroe Aceh Darussalam on papaya crops (Hidayat et al., 2012) and has since spread rapidly and widely. PRSV-P has been reported infecting papaya crops in Aceh, Medan, West Java, Yogyakarta, Central Java, East Java, and Bali (Hidayat et al., 2012; Harmiyati et al., 2015), as well as cucumber crop (Listihani et al., 2018). Infections by SqMV, CABYV, TMV, ZYMV, and SLCV on several Cucurbitaceae crops have been found more frequently in Java and Bali

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and may potentially spread to other regions (Listihani et al., 2020; Listihani et al., 2021; Pandawani et al., 2022; Selangga & Listihani, 2022). Similarly, WMV infection has been reported in watermelon crops in South Sulawesi (Wakmana et al., 2002).

Symptoms caused by viral infections in Cucurbitaceae crops are diverse. CMV infection induces mosaic patterns, stripes, leaf malformation, stunting, and curling (Shehata et al., 2023). ZYMV infection causes yellow mosaic, fruit and leaf malformation, blistering, reduced leaf lamina size, necrosis, and stunting (Ahsan et al., 2023). SqMV symptoms typically include greenish-yellow to dark green mosaic patterns, mainly around the leaf blade, accompanied by rigid, wrinkled leaves, reduced leaf size, and stunting (Listihani et al., 2022). Cucurbitaceae crops infected by PRSV exhibit symptoms such as stunting, mosaic, and fruit malformation (Listihani et al., 2018). WMV infection results in mosaic patterns, striping, and leaf distortion (López-Martín et al., 2024), while CGMMV infection causes leaf chlorosis, pedicle necrosis, and fruit damage (Xie & Wu, 2022).

Virus infections in Cucurbitaceae crops lead to significant production losses in several countries. CMV infection in chili pepper has caused yield losses of 30–60% in Croatia, Egypt, Greece, Israel, Italy, Japan, Poland, Portugal, and North America. ZYMV infection in France resulted in 64–85% yield losses (Li et al., 2020). CGMMV infection in cucurbits in Israel caused up to a 70% reduction in production (Philosoph et al., 2018).

Research on the presence of PRSV-W in Cucurbitaceae crops in Indonesia has been limited. PRSV-W is classified as a quarantine plant pest organism (A1 category, group 1), whose presence had not been previously recorded in Indonesia (Regulations of the Minister of Agriculture, 2020). PRSV-W has been reported to infect melon crops in Timor Leste (Maina et al., 2017) and was reported for the first time in Indonesia on melon crops in Bali Island (Selangga et al., 2022). Yield losses in Cucurbitaceae crops due to PRSV-W infection have reached up to 70–80% in Australia and Brazil (Rezende & Pacheco, 1998; Tian et al., 2024). Molecular characterization of PRSV-W Bali isolates showed the closest nucleotide and amino acid homology with East Timor isolates (KX655874), at 98.8% and 99.1%, respectively.

To date, there have been no reports estimating yield losses due to mosaic disease in melon crops in several countries. Therefore, information on yield loss caused by PRSV-W isolates from Bali is essential to determine the magnitude of damage caused by this

virus. Data on production loss can serve as a basis for predicting appropriate timing for virus distribution prevention and control strategies to mitigate PRSV-W damage to melon and other Cucurbitaceae crops. Accordingly, this study aimed to analyze yield loss and fruit quality reduction in melon crops in Bali resulting from PRSV-W infection.

MATERIALS AND METHODS

Research Site. The study was conducted at the Pegok Experimental Farm, Faculty of Agriculture, Udayana University. Pathogen identification was carried out in the Plant Disease Laboratory, Faculty of Agriculture, Udayana University.

Melon Cultivation. Melon cultivation consisted of several stages, including land preparation, seed sowing, planting, maintenance, and harvesting. Land preparation began with the construction of planting beds measuring 30 cm in height, 1 m in width, and 4 m in length. The distance between beds was 50 cm. Each bed contained two rows of plants, with a spacing of 60 × 80 cm between plants. The basal fertilizer used was fermented cow manure, applied at a rate of 10 kg per bed. Dolomite lime was applied after the manure application at a rate of 800 g per bed. The beds were then covered with plastic mulch.

Seed sowing was conducted by soaking the seeds in warm water at approximately 40 °C for 30 min, followed by soaking in atonik solution at a concentration of 1 mL/L for 30 min. The seeds were then placed in a damp cloth, covered, and kept at room temperature until germination. Germinated seeds were sown in a seedling medium consisting of cocopeat. Planting was carried out in the afternoon using 12 day old seedlings with four fully developed leaves. Seedlings were carefully removed from the seedbed and planted individually in prepared planting holes, with one seedling per hole. Crop maintenance included staking, watering, replanting, supplementary fertilization, and pruning.

Individual and Population (Plot or Field) Plant Disease Observation. Disease observation was conducted at the Experimental Farm, Faculty of Agriculture, Udayana University, Denpasar, Bali. The farm covers an area of 1000 m² and was used as the observation plot. Geographically, the site is located at 8°27'46.5" S and 115°13'00.0" E. The selection of the observation site in Denpasar, Bali, was based on previous research indicating that the highest incidence

of mosaic disease occurred in this area. A total of 50 symptomatic and asymptomatic melon crop samples were collected using a purposive sampling method from the predetermined plot. Observations were conducted from 6 days after planting (dap) until 66 dap.

Disease Observation with Different Disease Severity.

Observations at different disease severity levels were conducted on seven predetermined melon cultivation plots, each covering an area of 1000 m². The melon cultivar observed in this study was 'Alisa', with plant ages ranging from 30 dap to 86 dap. A total of 50 plants were observed in each plot using a zigzag sampling pattern.

Agronomic Variables and Disease Severity.

Observations of agronomic variables and disease severity were conducted on (1) diseased crops in the cultivation area and (2) crops representing different disease severity levels. Observations were performed every two weeks for a total of five observation periods. The variables measured included plant height, disease severity, and the area under the disease progress curve (AUDPC). Disease severity was determined using the following equation:

$$DS = \frac{\sum_i (n_i v_i)}{N \times Z} \times 100\%$$

DS = Disease severity;

ni = Number of melon crops showing a specific score;

vi = Score assigned to melon crops;

N = Highest score;

Z = Total number of melon crops observed.

AUDPC was calculated based on disease severity observations using the following equation:

$$AUDPC = \sum_{i=1}^n \frac{X_i + 1 + X_1}{2} X(t_i + 1 - t_i)$$

AUDPC = Area under disease progress curve;

X_i = Disease severity at the i-th observation;

t_i = Time of observation;

n = Number of observation until terminal disease.

At the end of the observation period, total harvest production, selling price, and yield loss percentage were calculated for each plot.

Yield Loss Estimation. Yield loss was calculated based on (1) individual crop observation data and (2) observations at different disease severity levels. Harvest yield from symptomatic and asymptomatic

crops was measured and calculated, including the number of fruits produced, individual fruit weight per plant, total fruit weight per cultivation area, fruit quality, and percentage of yield loss at the end of the observation period.

According to Selangga & Listihani (2022), yield loss percentage was calculated using the following parameters:

$$Y = \frac{P - R}{P} \times 100\%$$

Y = Yield loss;

P = Optimum result (the result obtained in a condition without pathogen invasion),

R = Actual result (the result obtained during a pathogen invasion and after control effort has been done).

Data Analysis. Data obtained from field observations were analyzed using the chi-square test to compare the number of fruits, individual fruit weight, and total fruit weight per plant within observation plots. Correlation and regression analyses were conducted to evaluate the relationship between yield loss and varying levels of disease severity.

RESULTS AND DISCUSSION

Mosaic disease strongly affected melon plant height. From the first to the last observation, the average height of melon plants exhibiting mosaic symptoms differed from that of healthy plants, measuring 104.02 cm and 115.8 cm, respectively. These results indicate that mosaic disease inhibits melon plant growth. Growth inhibition disrupts nutrient uptake, which subsequently affects fruit production. Mosaic disease development was observed from 18 days after planting until just before harvest. Observations were conducted when plants were aged 6 to 66 days after planting (dap) (Figure 1). These findings demonstrate that plant growth progression is closely associated with disease development.

Disease incidence and disease severity of mosaic disease influenced melon plant height in the experimental plots. Mosaic disease pathogens infect melon plants systemically, beginning in the leaves and subsequently spreading throughout the entire plant. Such systemic infection adversely affects plant height.

Mosaic disease has become a serious problem in melon cultivation areas in Bali Province. Observations showed that mosaic disease infected melon plants from as early as 6 dap until harvest. Locations at lower elevations, approximately 15 m above sea level,

exhibited the highest disease incidence. Environmental conditions and elevation significantly influence disease progression. According to Hutasoit et al. (2023) and Selangga et al. (2023), factors affecting disease incidence in the field include insect vector populations, climate change, mixed virus infections, host plant susceptibility, and improper cultivation practices.

The causal agent of mosaic disease in melon has been confirmed as Papaya ringspot virus watermelon strain (PRSV-W) by Selangga et al. (2022). This study reported PRSV-W infection in melon crops in Bali, Indonesia, for the first time. PRSV-W isolates from Bali showed high sequence similarity and a close phylogenetic relationship with cucurbit isolates from Timor Leste. These findings suggest that PRSV-W outbreaks and rapid global spread may be associated with host availability, vector abundance, and geographical proximity.

PRSV-W is classified as a quarantine plant pest organism (OPTK) under A1 category group 1, according to Indonesian quarantine regulations (Regulation of the Minister of Agriculture, 2020). The A1 category

indicates that the virus was previously absent from Indonesia. PRSV-W may have entered Indonesia through international trade involving plants, plant products, or vegetative propagules that unknowingly carried the virus. Group 1 OPTK indicates that the pathogen cannot be separated from its carrier media.

Disease incidence was positively correlated with disease severity. Observations revealed that higher disease incidence resulted in increased disease severity. The highest disease severity recorded at the end of the observation period reached 61.8% (Figure 2). Disease intensity was strongly associated with melon yield loss, where increased disease incidence and severity resulted in reduced yield.

Harvest yields from healthy melon plants and mosaic-symptomatic plants showed significant differences in individual fruit weight. This finding indicates that mosaic disease reduces fruit weight. Melon yield is influenced by both fruit quality and quantity; larger fruits contribute to higher yield. Differences in fruit weight between asymptomatic and symptomatic plants significantly reduced total

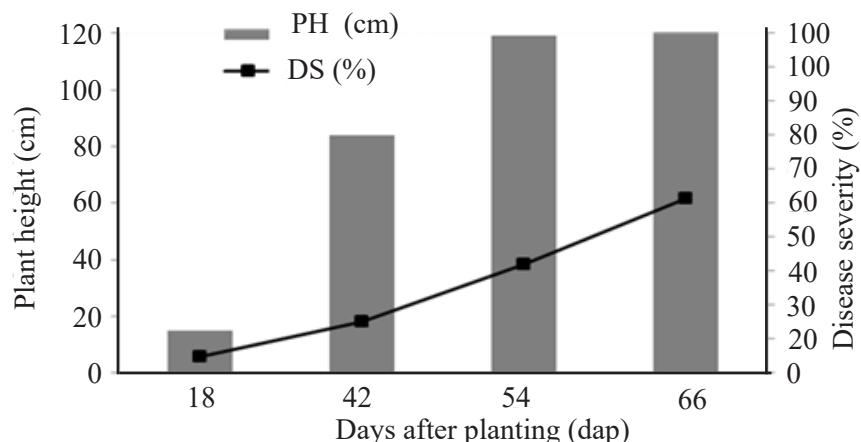


Figure 1. Plant height and disease severity in plants exhibiting mosaic symptoms.

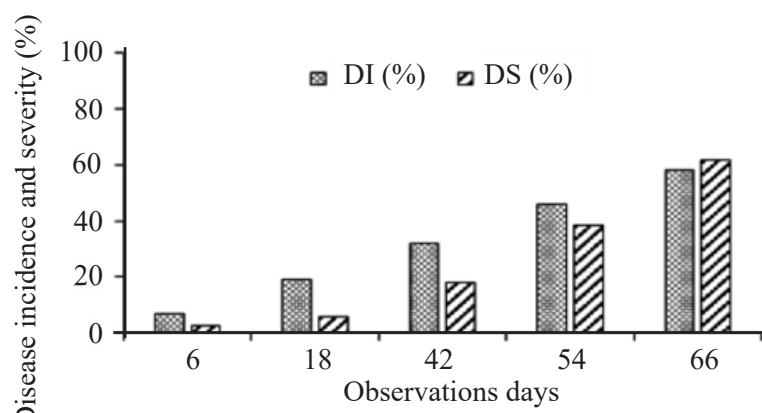


Figure 2. Incidence and severity of melon mosaic disease at different levels of infection.

yield (Table 1). These results are consistent with Luis-Arteaga et al. (1998), who reported that virus infection reduces fruit weight in melon compared to healthy plants. The actual yield loss calculated by comparing asymptomatic and mosaic symptomatic plants reached 49.45% (Table 1).

Yield losses from mosaic disease resulted in both direct and indirect impacts. Direct impacts included economic losses affecting farmers and consumers, while indirect impacts were associated with reduced fruit quality. Lower-quality produce resulted in reduced market prices. Fruits from mosaic-symptomatic plants exhibited quality defects, including shape malformation, discoloration, perforation, rotting, and abnormal shape and color changes (Table 2). The highest incidence of quality defects was observed in shape malformation and color and shape changes, accounting for 17.5% and 14.06%, respectively.

Mosaic disease caused a significant reduction in both yield quality and quantity. Yield loss reaching 49.45% was primarily due to the production of smaller fruits compared to those from healthy plants (Table 1). Reduced fruit weight due to viral infection has been widely reported in melon crops. Early-season

virus infection has been reported to cause yield losses ranging from 30% to 60% (Premchand et al., 2025).

Melon productivity was significantly influenced by disease severity levels. Higher disease severity resulted in lower melon production. Production loss reached 30.15% in plots with a disease severity level of 61.8% (Table 3). The lowest yield was observed in plots with an AUDPC value of 751, producing only 1126.6 kg per 1000 m². This reduction corresponded with visible fruit symptoms, particularly reduced fruit size. Fruit size and quality directly influence market price. High-quality melons without physical defects command higher prices. Market prices for melon average approximately IDR 25,000 per kg, while farm-gate prices average IDR 18,000 per kg. Mosaic disease reduced market value, resulting in substantial income loss for farmers. The greatest economic loss occurred in plots with an AUDPC of 751, reaching IDR 15,721,500 (Table 3).

Linear regression analysis showed high coefficients of determination (R^2) between disease incidence and yield loss (0.9541) and between disease severity and yield loss (0.9619) (Figures 3 and 4). These results indicate strong correlations, explaining 95.41%

Table 1. Harvest results of healthy melons and those with mosaic symptoms

Plants	Agronomic variables	
	Weight per fruits (g)	Harvest (g)
Symptomless mosaic	2441 a	649,401.99 a
Mosaic symptom	1293 b	328,292.53 b
Yield losses (%)	47.03	49.45

Numbers in one column followed by different letters indicate the difference with the chi-square test.

Table 2. The quality of melon yields on mosaic symptomatic plants and healthy plants

Fruit quality	Symptomless		Mosaic	
	Number (fruits)	Percentage (%)	Number (fruits)	Percentage (%)
Healthy	287	89.68	198	61.87
Malformation	21	6.56	56	17.5
Discoloration	7	2.18	11	3.43
Holes and rotten	3	0.93	10	3.12
Color and shape	2	0.62	45	14.06

Table 3. Yield loss on some fields

AUDPC DS*	Yields (Kg/1000 m ²)	Yield loss (%)	Yield loss (IDR)
369	1761.8	14.35	4,287,720
572	1721.3	14.62	5,017,500
598	1537.1	18.01	8,332,500
673	1395.4	26.33	10,882,500
751	1126.6	30.15	15,721,500

*AUDPC DS= Area under diseases progress curve value of disease severity.

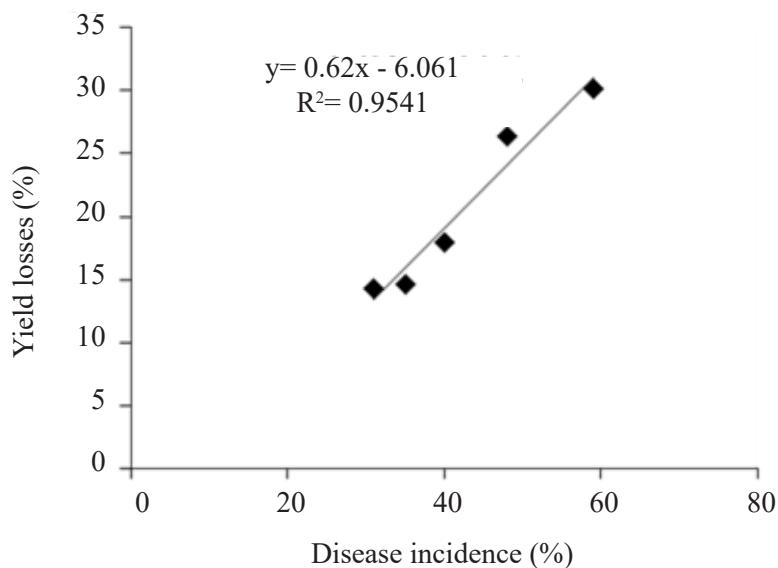


Figure 3. Relationship between yield loss and disease incidence (DI).

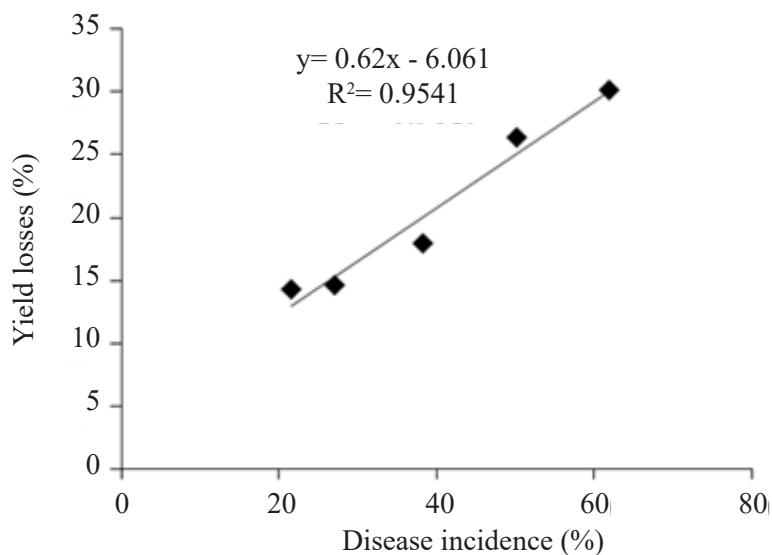


Figure 4. Relationship between yield loss and disease severity (DS).

and 96.19% of yield loss variation, respectively. R^2 values approaching 1 indicate a strong relationship between disease parameters and yield loss. Thus, increasing disease incidence and severity directly increase yield loss.

PRSV-W infection affected both the quantity and quality of melon fruits. In addition to reducing fruit size and weight, PRSV-W infection altered fruit skin color from yellow-orange to pale yellow and induced ringspot symptoms (Figure 5). These findings suggest that PRSV-W infects fruit tissues, including the fruit skin. RT-PCR analysis confirmed the presence of Potyvirus in symptomatic fruit skin. Sequencing results verified PRSV-W infection in fruit skin tissues

(data not shown). This ringspot symptom was observed exclusively in PRSV-W infected fruits, whereas healthy fruits retained a yellow-orange skin color.

Fruit flesh color was also affected. In healthy fruits, orange coloration extended from the seed cavity to the outer flesh, whereas PRSV-W infected fruits exhibited orange coloration only near the seeds. Fruit flesh color is associated with sweetness. PRSV-W infection reduced fruit sweetness to 9–12 °Brix, compared to 14–17 °Brix in healthy fruits.

PRSV-W infection caused significant changes in fruit skin and flesh color and reduced sweetness. These findings align with Tripathi et al. (2008), who reported that PRSV-P infection reduced fruit sweetness

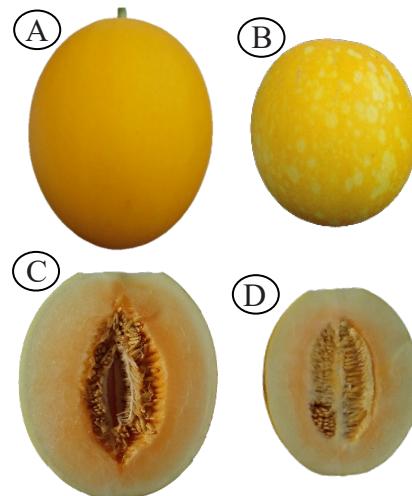


Figure 5. Skin and flesh color of melon fruits: healthy (A, C) and PRSV-W infected (B, D).

in papaya and melon by up to 50%. The present results suggest a relationship between viral symptom severity, cellular disruption, and deterioration of fruit quality in PRSV-W infected melon plants.

Given the severe impact of mosaic disease on melon production, dissemination of disease information and implementation of effective control strategies are essential. Control measures should emphasize the use of healthy planting materials, environmental sanitation, and insect vector management. Disease free seed selection is a critical initial step in preventing mosaic disease. Selecting larger seeds may promote better early plant growth. Environmental sanitation aims to eliminate virus inoculum sources in the field, including the use of mulch and multiple cropping systems. Hay mulch has been reported to reduce vector-borne disease incidence by up to 30% (Kirchner et al., 2014; Listihani, 2019). Another effective strategy involves adjusting planting time to periods of low aphid population density.

Insect vector presence strongly influences virus transmission. According to Listihani (2019), aphid vectors (*Aphis gossypii* and *Myzus persicae*) play a crucial role in mosaic virus spread and yield reduction in Cucurbitaceae crops. Similarly, Moya-Ruiz et al. (2023) reported that higher aphid populations increased PRSV-W incidence and reduced melon production in Spain. Vector population management strategies include planting barrier crops, which act as virus sinks and physical barriers to aphid movement (Damicone et al., 2007; Ramirez et al., 2023). Integrated disease management combining hay mulch, intercropping systems, and botanical insecticide application may further reduce disease incidence and yield loss.

CONCLUSION

The PRSV-W isolate from Bali caused a melon yield loss of 49.45%. The highest disease incidence and disease severity recorded at the end of the observation period were 58.4% and 61.8%, respectively. Yield loss showed strong correlations with disease incidence and disease severity, with correlation values of 95.41% and 96.19%, respectively. PRSV-W infection also altered the melon fruit skin color from yellow-orange to pale yellow and induced ringspot symptoms, and PRSV-W was detected in the skin tissues of diseased fruits. In addition, PRSV-W infection reduced fruit sweetness to 9–12 °Brix, whereas healthy fruits exhibited sweetness levels of 14–17 °Brix. Overall, increasing severity of PRSV-W infection resulted in greater yield losses in melon crops.

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AUTHORS' CONTRIBUTIONS

DGWS, IGRMT, GNAS, and IPS conceptualized and designed the experiment. DGWS, IGRMT, GNAS, IPS, and LL conducted yield loss observations at the

Pegok Experimental Farm, Faculty of Agriculture, Udayana University. DGWS and LL performed molecular identification and bioinformatics analyses. All authors contributed to manuscript preparation and approved the final version.

COMPETING INTEREST

The authors declare that they have no known financial or non-financial competing interests.

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