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Differential Biochemical Responses of Resistant and Susceptible Genotypes of Chili to Pepper Yellow Leaf Curl Thailand Virus

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Abstract

Chili (*Capsicum annuum* L.) production is threatened by the pepper yellow leaf curl virus (PepLCV), transmitted by whiteflies, leading to reduced yields. This study investigated the biochemical changes in two chili genotypes, PEP6 (tolerant to PepLCV) and Homsuphan (susceptible to PepLCV), following inoculation with the Thailand strain of PepLCV (known as Pepper Yellow Leaf Curl Thailand Virus, PepYLCTHV). Inoculation was performed using whitefly transmission (WF) and graft transmission (GT) methods, and disease severity was evaluated using a standardized index. The level of total phenolic compounds and the activities of peroxidase (POD) and polyphenol oxidase (PPO) enzymes were analyzed in virus-infected plants and compared with those in uninoculated controls. Both chili genotypes exhibited a more rapid increase in disease severity when inoculated with WF than with GT. In PEP6, disease severity was lower than Homsuphan in both WF and GT inoculations. Disease severity in WT-inoculated PEP6 plants increased gradually, reaching 100% by day 36, whereas Homsuphan plants had a more rapid progression, attaining 100% by day 21. The GT method led to slower disease severity progression in both genotypes, reaching 80–85% by day 36. In PEP6 plants, total phenolic compound content increased significantly following WF, indicating an active defense response, whereas levels remained stable in GT plants. Phenolic content in the tolerant genotype Homsuphan remained stable across all conditions. Notably, peroxidase (POD) activity was elevated in GT plants of both genotypes, which correlated with reduced disease severity. Polyphenol oxidase (PPO) activity was lowest in control plants, but WT and GT increased the PPO level. Among the treatments, GT induced the highest PPO activity, which was associated with the lowest disease severity. These findings suggest that GT may enhance disease resistance by modulating phenolic compound accumulation and increasing POD and PPO activity.

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Keywords: *Capsicum annuum* L; peroxidase; PepYLCTHV; PepLCV; phenolic compound; polyphenol oxidase

1. Introduction

Chili (*Capsicum annuum* L.) is an economically important vegetable and spice crop cultivated worldwide. In 2023, global chili production reached approximately 38 million tons [1], and the demand continues to increase due to its culinary, nutritional, and industrial values [2]. However, chili productivity has decreased in many regions due to the epidemic of pepper yellow leaf curl virus (PepLCV), a member of the genus *Begomovirus* [3]. Whiteflies (*Bemisia tabaci*) transmit PepLCV in a persistent and circulative manner [4], leading to yield losses ranging from 20 to 100% depending on disease severity [5]. pepper yellow leaf curl Indonesia virus (PepYLCIV) has been reported in Thailand since 1995, with a severe outbreak in Kanchanaburi province in 2014. Among various species of PepYLCV, pepper yellow leaf curl Kanchanaburi virus (PepYLCKaV) and pepper yellow leaf curl Thailand virus (PepYLCTHV) are the most prevalent, aggressive, and problematic [6]. Several chili germplasm lines resistant to begomoviruses, such as BG-3821 [7], Bhut Jolokia [8], DLS-Sel-10, WBC-Sel-5, PBC142 [9], PSP-11, KR-B/NP-46-A, PBC145 [10], 9853-123, and PSP11 [11,12] have been reported. These genotypes exhibit varying levels of tolerance, often characterized by reduced symptom severity, lower viral load, and sustained fruit yield under field conditions. For instance, despite more than four cycles of selfing, progenies of PSP11 continued segregated into highly resistant to highly susceptible plants against PepYLCTHV; however, a tolerant line designated as PEP6 was isolated [12]. Molecular studies have identified two genes involved in chili's defense against PepYLCV. The first, *pepy-7*, is a recessive gene that encodes the messenger RNA surveillance factor Pelota. The second, *Pepy-2*, is a dominant locus located on chromosome 7, which harbors the CaRDR3a gene—a strong candidate for conferring resistance to PepYLCV [13,14]. Chili plants possess diverse stress recognition mechanisms in response to viral infections [15], which include both physical barriers, such as cuticle thickness, wax deposition, and trichome density [16], and biochemical defense mechanisms.

The biochemical defense in chili plants could be either pre-existing or induced. The induced responses are rapid biochemical reactions triggered after pathogen infection. Examples include the production of reactive oxygen species (ROS), which eliminate infected cells and also act as signaling molecules to activate immune pathways [17]. The synthesis of antimicrobial phenolic compounds [18] and production of defense-related enzymes such as peroxidase (POD) [15], polyphenol oxidase (PPO), phenylalanine ammonia lyase (PAL), and catalase (CAT) are significantly increased following viral infection [19].

Under natural conditions, whiteflies (*Bemisia tabaci*) serve as efficient vectors for begomoviruses, resulting in systemic and wide-scale spread of virus across chili crops [20]. In contrast, grafting inoculation enables transmission of viruses through vascular connectivity between infected scion and healthy rootstock, thereby allowing detailed observation of symptomatology and host responses at an individual plant level [21]. Comparative evaluation of these two inoculation methods could distinguish ecological epidemiology from controlled infection dynamics, providing critical insight for targeted virus management strategies, resistance breeding programs, and diagnostic tools [22].

Previously, in a preliminary report, we described enhanced levels of phenols and peroxidase activities in a PepYLCTHV-resistant chili genotype [15]. In this communication, we present results of a comprehensive biochemical analysis on one each of resistant and susceptible chili genotypes following two distinct inoculation methods. We discuss the

observed biochemical changes and interpret them in the context of management strategies for PepYLCTHV.

2. Materials and Methods

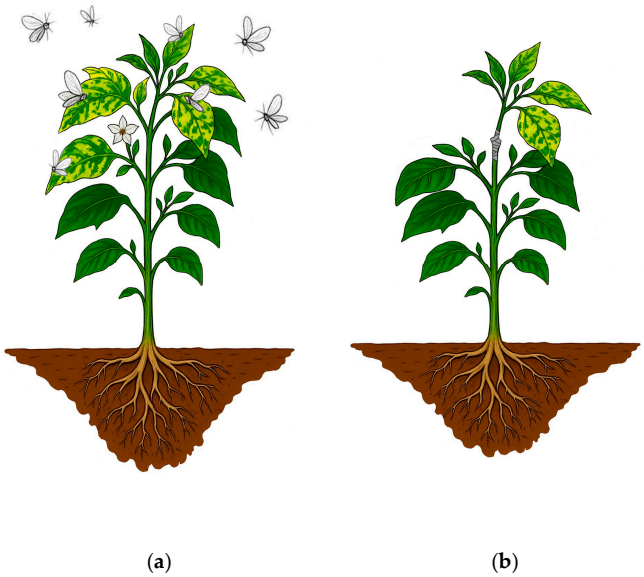
2.1. Plant Materials

The experiments were conducted at the greenhouse at the School of Agricultural Technology, King Mongkut's Institute of Technology, Ladkrabang (KMITL), Thailand, from August 2023 to July 2024. During this period, we maintained an average temperature of 30.66 °C and a relative humidity of 74.69%. Chili genotypes Homsuphan and PEP6, which are susceptible and tolerant genotypes against PepYLCTHV, respectively [12], were used. Seeds were sown in 60-cell germination trays with commercial peat moss media. Thirty-day-old seedlings with cotyledons and 3–4 leaves were transplanted in a 12-inch plot and manually watered every day with 600–1000 mL/pot. Ninety days after transplanting, the chili plants were inoculated.

A 2 × 3 Factorial Completely Randomized Design (CRD) with five replications was used. Factor A consisted of two chili genotypes (PEP6 and Homsupan), and Factor B included two virus inoculation methods: through whitefly transmission (WF) and graft transmission (GT), and control (non-inoculation). For each treatment, 120-day-old chili seedlings were assigned to six treatments: PEP6 + WF, PEP6 + GT, PEP6 + Control, Homsuphan + WF, Homsuphan + GT, and Homsuphan + Control.

2.2. Virus Inoculation and Disease Evaluation

Inoculum for both whitefly (WF) and grafting (GT) treatments was prepared through whitefly transmission. For the WF method, virus-free adult whiteflies (Figure 1), reared on cotton plants in insect-proof cages for 21 days, were given a 24-h acquisition access period (AAP) on pepper yellow leaf curl Thailand virus (PepYLCTHV)-infected plants. After AAP, 10–15 viruliferous whiteflies were transferred to each 120-day-old chili plant. These plants were maintained in a greenhouse, and whiteflies were allowed a 48-h inoculation access period (IAP) before being eliminated with a 20% acetamiprid SP spray [12]. The remaining treatments, i.e., the control and grafted plants, were also sprayed with 20% acetamiprid. For the GT method, PepYLCTHV-infected chili branches served as scions and were grafted onto the PEP6 and Homsupan plants. Disease severity was scored daily from 1 to 36 days post-inoculation using a 6-level scale [12] (Table 1 and Figure 2).



(**a**) (**b**)

Figure 1. Pictorial depiction of inoculation methods, (**a**) whitefly (WF) and (**b**) graft (GT) inoculation.

Table 1. Disease scale based on symptom severity.

Score	Symptoms	Disease Incidence (%)	Disease Response
0	No visible symptoms on leaves	0%	HR; highly resistant
1	Curling and clearing of upper leaves	0–5%	R; resistant
2	Curling and clearing of leaves, and swelling of veins	6–25%	MR; moderately resistant
3	Curling, puckering, and yellowing of leaves and swelling of veins	26–50%	MS; moderately susceptible
4	Leaf curling, stunted plant growth, and blistering of internodes	51–75%	S; susceptible
5	Curling and deformed small leaves, stunted plant growth without flowering	>75%	HS; highly susceptible

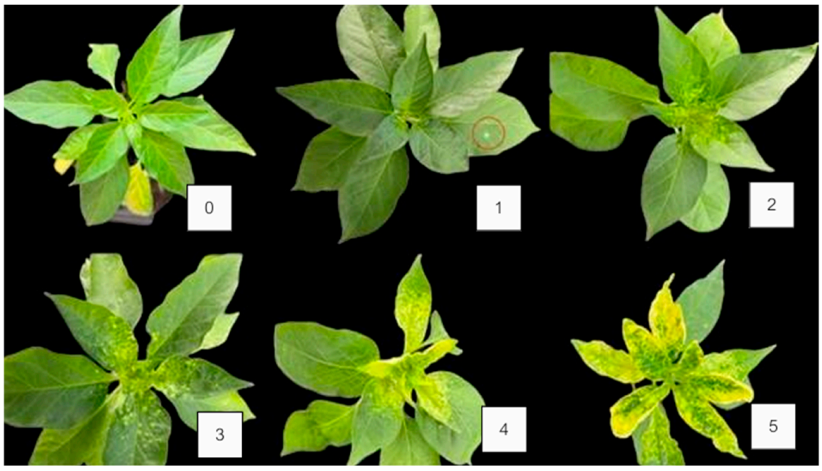


Figure 2. Pepper Yellow Leaf Curl Thailand Virus symptoms scoring scales in chili seedlings. 0 = HR (highly resistant); 1 = R (resistant); 2 = MR (moderately resistant); 3 = MS (moderately susceptible); 4 = S (susceptible) and 5 = HS (highly susceptible). The red circle indicates the initial symptom of pepper yellow leaf curl Thailand virus.

2.3. Determination of Total Phenolics Content (TPC)

The total phenolic content was analyzed by the Folin–Ciocalteu method [23]; 0.2 g of young, infected leaves was harvested, and phenol was extracted by adding 1 mL of 80% methanol. The mixture was vortexed for 10 min and subsequently centrifuged at 251.55 g for 20 min at 25 °C. The supernatant was transferred into a new tube. 50 µL of Folin–Ciocalteu reagent and 100 mL of Na_2CO_3 solution (75 g/L) were added, followed by 750 mL of distilled water. The reaction was incubated in a water bath at 50 °C for 16 min. The absorbance corresponding to total phenolic content was measured using a spectrophotometer at 765 nm (Shimadzu Corporation, Tokyo, Japan). The obtained values were extrapolated from a gallic acid standard curve and expressed as milligrams gallic acid equivalent (GAE) per gram of sample. The total phenolic content of the extract was calculated using the following Formula (1):

$$\text{Total phenolic content } (\text{mg GAE} \cdot \text{mL}^{-1}) = \frac{C \times V}{M} \quad (1)$$

where C = the concentration of gallic acid established from the calibration curve ($\text{mg} \cdot \text{mL}^{-1}$). V = the volume of the extract solution in mL, and M = the weight of the extract (g).

2.4. Estimation of Peroxidase Activity (POD)

The peroxidase activity (POD) was determined using a known protocol [24]; 0.2 g of young, infected leaves was collected from each treatment and homogenized in 1.5 mL of chilled 50 mM sodium phosphate buffer (pH 7.0). The homogenate was centrifuged at 9479× *g* for 20 min at 4 °C. The reaction mixture (3.0 mL total volume) contained 50 mM sodium phosphate buffer (pH 7.0), 4 mM H_2O_2, 20 mM guaiacol, and 200 µL of supernatant. The decomposition of H_2O_2 was monitored at a wavelength of 436 nm by measuring the decrease in absorbance per minute to calculate the POD activity. The POD activity was calculated as described by the following Formula (2):

$$\text{Peroxidase } (\text{U}\cdot\text{mL}^{-1}) = \frac{A_{F436} \times A_{I436}}{t \times V} \quad (2)$$

where A_{F436} = final absorbance at wavelength 436 nm, A_{I436} = initial absorbance at wavelength 436 nm, t = time (min), and V = volume of enzyme (g)

2.5. Determination of Polyphenol Oxidase Activity (PPO)

Polyphenol Oxidase (PPO) activity was analyzed according to the method described by Yadav et al. [25]. 0.1 g of young, infected leaves was homogenized, and the supernatant was obtained by centrifugation at 9479× *g* for 20 min at 4 °C. The reaction mixtures consisted of 2 mL of 10 mM sodium phosphate buffer (pH 6.5), 1.7 mL of 10 mM Catechol, and 0.05 M sodium phosphate buffer, which were mixed with 200 µL of the supernatant. The reaction was monitored by measuring the increase in absorbance at 495 nm every 15 s for a total of one minute. PPO enzyme activity was expressed as the change in absorbance per minute per milligram of protein. Enzyme activity was calculated using the following Formula (3):

$$\text{Polyphenol oxidase } (\text{U}\cdot\text{mL}^{-1}) = \frac{A_{F495} \times A_{I495}}{0.001 \times t \times V} \quad (3)$$

where A_{F495} = Final absorbance at 495 nm, A_{I495} = Initial absorbance at 495 nm, t = Reaction time in minutes, V = Volume (mL) of crude enzyme extract used in the assay, and 0.001 = Conversion factor representing a change in absorbance of 0.001 units per minute.

2.6. Data Analysis

The disease severity index of each genotype was calculated as a percentage of disease incidence by the following formula:

$$\text{DSI } (\%) = \frac{\sum(ni \times vi)}{(N \times V)} \quad (4)$$

where i: 0–5, ni = number of symptomatic plants to value of a particular score, vi = value symptom score, N = the total number of plants were observed and V = the highest score value. Analysis of variance and mean analysis of the incidence of the chili pepper yellow leaf curl Thailand virus disease using IBM SPSS version 29 and comparison of means using the LSD (Least Significant Difference) test at a 95% confidence level. The biochemical data (TPC, POD and PPO) were collected daily over a 10-day period, with all treatments replicated three times. The resulting data will be analyzed using IBM SPSS version 29.

3. Results

3.1. Disease Severity of PepYLCTHV

The disease severity index in tolerant (PEP6) and susceptible (Homsuphan) chili genotypes inoculated by whitefly (WF) increased sharply compared to the graft inoculation method (GT) only after 3 days of inoculation (Figure 3). The percentage of disease index

(DSI%) of PEP6 was lower than that of Homsuphan, and the disease increased over time in both genotypes. A sharp increase in DSI (%) was recorded between the third and seventh day after WF inoculation, reaching a peak of 50% by day 10. Homsuphan, upon WF inoculation, DSI remained low in the initial days (around 0% until day 3) and then increased sharply, reaching a plateau at the highest level of 100% from day 21 onwards. The DSI in both genotypes also showed an increase in disease severity over time. By day 36, the disease severity index in the grafted group in both varieties was around 80–85% (Figures 3 and 4).

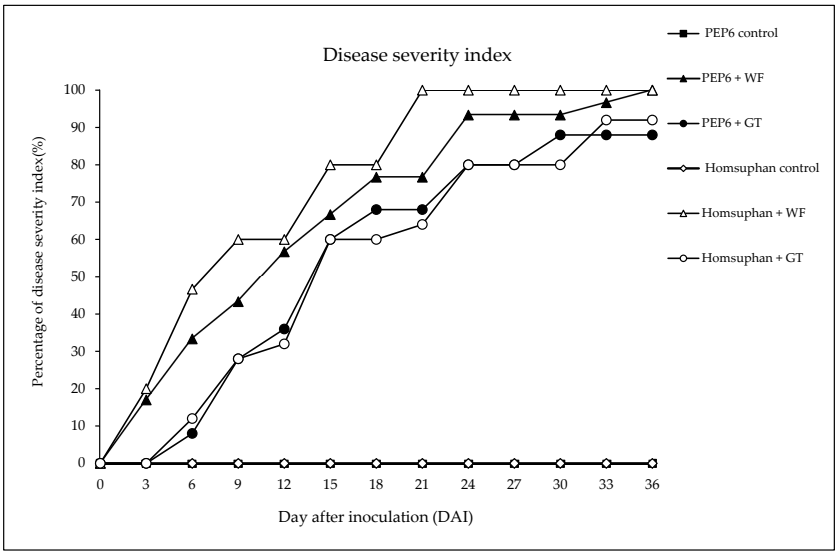


Figure 3. The disease severity index of two chili genotypes inoculated with PepYLCTHV. Each value is expressed as the means of replication in PEP6 and Homsuphan after inoculation by whitefly (WF), grafting (GT), and control.



(**a**) (**b**)

Figure 4. Severe symptoms of pepper yellow leaf curl Thailand virus (red circle) after inoculation in PEP6 upon whitefly inoculation (**a**) and graft inoculation (**b**).

3.2. Total Phenolic Content (TPC) in Response to PepYLCTHV

PEP6 exhibited a significantly higher total phenolic content (TPC) than Homsupan on the first day post-inoculation, measuring 7.68 ± 0.17 mg GAE·mL^{-1} and 6.77 ± 0.17 mg GAE·mL^{-1}, respectively. From the 2nd to the 10th day, Homsupan showed a gradual increase in TPC, reaching levels comparable to PEP6, with no significant differences observed ($p > 0.05$) (Tables 2 and 3).

Table 2. Mean square of analysis of the variable of inoculation treatment in resistant and susceptible chili leaves.

S.O.V	DF	Day 1			Day 2			Day 3			Day 4			Day 5		
		TPC	**POD**	**PPO**	**TPC**	**POD**	**PPO**	**TPC**	**POD**	**PPO**	**TPC**	**POD**	**PPO**	**TPC**	**POD**	**PPO**
Variety (V)	1	4.47 **	0.10 ^{ns}	1360.51 ^{ns}	0.43 ^{ns}	0.28 ^{ns}	7255.43 ^{ns}	0.47 ^{ns}	0.31 ^{ns}	201.16 ^{ns}	2.21 ^{ns}	0.30 ^{ns}	10,451.42 *	0.001 ^{ns}	1.51 **	1572.66 ^{ns}
Inoculation (I)	2	0.43 **	0.11 ^{ns}	1907.78 ^{ns}	0.057 ^{ns}	0.23 ^{ns}	10,667.86 ^{ns}	0.23 ^{ns}	0.12 **	1578.17 ^{ns}	0.06 ^{ns}	0.17 ^{ns}	6413.72 *	0.47 ^{ns}	0.57 ^{ns}	2454.84 ^{ns}
V × I	2	0.22 ^{ns}	0.07 ^{ns}	605.56 ^{ns}	0.012 ^{ns}	0.41 ^{ns}	10,998.32 ^{ns}	0.31 ^{ns}	0.11 *	1113.60 ^{ns}	0.002 ^{ns}	0.15 ^{ns}	948.52 ^{ns}	0.19 ^{ns}	0.41 ^{ns}	17,894.02 ^{ns}
Error	18	0.27	0.105	1917.76	0.33	0.151	3631.36	0.19	0.023	900.32	0.19	0.18	1456.95	0.21	0.213	7218.01
C.V.%		7.21	60.01	130.87	7.43	69.39	128.39	5.71	29.16	118.56	5.95	73.15	32.76	6.21	60.73	64.13
S.O.V	DF	Day 6			Day 7			Day 8			Day 9			Day 10		
		TPC	POD	PPO	TPC	POD	PPO	TPC	POD	PPO	TPC	POD	PPO	TPC	POD	PPO
Variety (V)	1	0.77 ^{ns}	0.87 **	14,602.07 *	4.34 ^{ns}	0.10 ^{ns}	1120.38 ^{ns}	0.52 ^{ns}	0.004 ^{ns}	305.72 ^{ns}	0.005 ^{ns}	0.04 ^{ns}	735.53 ^{ns}	1.13 ^{ns}	0.20 ^{ns}	17,238.78 **
Inoculation (I)	2	3.53 *	0.24 **	10,165.28 *	0.75 ^{ns}	0.26 ^{ns}	1343.69 ^{ns}	0.14 ^{ns}	0.09 ^{ns}	58.56 ^{ns}	0.86 ^{ns}	0.02 ^{ns}	959.05 ^{ns}	0.80 ^{ns}	0.04 ^{ns}	6109.27 ^{ns}
V × I	2	1.35 ^{ns}	0.08 ^{ns}	8760.68 *	0.43 ^{ns}	0.17 ^{ns}	1579.69 ^{ns}	0.06 ^{ns}	0.003 ^{ns}	445.41 ^{ns}	1.17 ^{ns}	0.005 ^{ns}	793.08 ^{ns}	0.62 ^{ns}	0.06 ^{ns}	9122.83 *
Error	18	0.92	0.041	2124.79	1.03	0.15	637.66	0.82	0.076	715.30	0.71	0.037	650.53	0.36	0.042	1888.01
C.V.%		10.27	51.92	69.88	10.76	49.65	54.82	9.61	44.46	50.70	9.26	37.72	44.80	7.08	37.26	52.92

* = Significant at 5% probability level, ** = significant at 1% probability level, ns = non-significant, DF = degree of freedom, C.V. = coefficient of variation.

Table 3. Total phenolic content, peroxidase (POD), and polyphenol oxidase (PPO) in PEP6 and Homsuphan following PepYLCTHV inoculations.

Varieties	Day-1			Day-2			Day-3			Day-4			Day-5		
	TPC	**POD**	**PPO**	**TPC**	**POD**	**PPO**	**TPC**	**POD**	**PPO**	**TPC**	**POD**	**PPO**	**TPC**	**POD**	**PPO**
PEP6 (V_1)	7.68 ± 0.17 ^{a}	0.53 ± 0.09	24.70 ± 12.5	7.89 ± 0.18	0.63 ± 0.11	36.63 ± 17.20	7.77 ± 0.13	0.62 ± 0.04	25.75 ± 7.88	7.03 ± 0.14	0.71 ± 0.12	131.73 ± 109.01 ^{a}	7.25 ± 0.14	0.96 ± 0.13	137.73 ± 87.14
Homsuphan (V_2)	6.77 ± 0.17 ^{b}	0.56 ± 0.09	39.59 ± 12.5	7.63 ± 0.18	0.50 ± 0.11	71.02 ± 17.20	7.50 ± 0.13	0.42 ± 0.04	20.02 ± 2.16	7.69 ± 0.14	0.45 ± 0.12	90.47 ± 67.74 ^{b}	7.34 ± 0.14	0.50 ± 0.13	121.72 ± 71.13
F-test	**	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	*	ns	ns	ns
Treatment															
Control (B_1)	6.89 ± 0.26 ^{b}	0.65 ± 0.13	23.61 ± 17.88	7.78 ± 0.29 ^{ab}	0.80 ± 0.16	98.61 ± 24.60	7.37 ± 0.22 ^{b}	0.56 ± 0.06 ^{ab}	7.14 ± 12.25	7.24 ± 0.22 ^{b}	0.74 ± 0.17	75.97 ± 15.58 ^{b}	7.01 ± 0.23 ^{b}	1.10 ± 0.19	111.85 ± 34.68
Whitefly (B_2)	7.42 ± 0.17 ^{a}	0.43 ± 0.10	24.05 ± 13.85	7.83 ± 0.18 ^{ab}	0.44 ± 0.12	36.47 ± 19.06	7.60 ± 0.14 ^{ab}	0.61 ± 0.05 ^{a}	25.37 ± 9.49	7.43 ± 0.14 ^{a}	0.45 ± 0.13	129.45 ± 12.07 ^{a}	7.52 ± 0.14 ^{a}	0.55 ± 0.15	147.58 ± 26.87
Grafting (B_3)	7.17 ± 0.17 ^{ab}	0.59 ± 0.10	48.78 ± 13.85	7.68 ^{b} ± 0.18	0.54 ± 0.12	26.40 ± 19.06	7.76 ± 0.14 ^{a}	0.40 ± 0.05 ^{b}	36.15 ± 9.49	7.34 ± 0.14 ^{ab}	0.61 ± 0.13	127.88 ± 12.07 ^{a}	7.19 ± 0.14 ^{b}	0.76 ± 0.15	129.73 ± 26.87
F-test	**	ns	ns	**	ns	ns	**	**	ns	**	ns	*	**	ns	ns

Table 3. *Cont.*

Varieties	Day-6			Day-7			Day-8			Day-9			Day-10		
	TPC	POD	PPO	TPC	POD	PPO	TPC	POD	PPO	TPC	POD	PPO	TPC	POD	PPO
PEP6 (V_1)	9.03 ± 0.30	0.57 ± 0.06 ^{a}	90.09 ± 62.64 ^{a}	9.00 ± 0.32	0.85 ± 0.11	37.77 ± 22.73	9.24 ± 0.29	0.60 ± 0.08	56.66 ± 40.73	9.13 ± 0.27	0.54 ± 0.06	52.28 ± 37.09	8.17 ± 0.19	0.65 ± 0.06	60.71 ± 34.84 ^{b}
Homsuphan (V_2)	9.67 ± 0.30	0.22 ± 0.06 ^{b}	41.31 ± 13.86 ^{b}	9.86 ± 0.32	0.71 ± 0.11	51.28 ± 36.24	9.57 ± 0.29	0.64 ± 0.08	49.60 ± 33.67	9.12 ± 0.27	0.47 ± 0.06	63.2 ± 48.04	8.77 ± 0.19	0.45 ± 0.06	113.72 ± 87.85 ^{a}
F-test	ns	**	*	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	**
Treatment															
Control (B_1)	8.99 ± 0.48	0.58 ± 0.08 ^{a}	63.94 ± 18.82 ^{ab}	9.29 ± 0.51	0.59 ± 0.16	34.50 ± 10.31	9.20 ± 0.45	0.67 ± 0.11	55.53 ± 10.92	8.53 ± 0.42	0.57 ± 0.08	63.06 ± 10.41	8.61 ± 0.3	0.64 ± 0.08	120.42 ± 17.74
Whitefly (B_2)	9.90 ± 0.30	0.45 ± 0.06 ^{a}	98.43 ± 14.58 ^{a}	9.72 ± 0.32	0.74 ± 0.12	58.60 ± 7.99	9.38 ± 0.29	0.70 ± 0.09	50.23 ± 8.46	9.24 ± 0.27	0.48 ± 0.06	64.12 ± 8.07	8.29 ± 0.19	0.49 ± 0.07	76.83 ± 13.74
Grafting (B_3)	8.85 ± 0.30	0.24 ± 0.06 ^{b}	34.72 ± 14.58 ^{b}	9.20 ± 0.32	0.95 ± 0.12	40.47 ± 7.99	9.51 ± 0.29	0.52 ± 0.09	53.62 ± 8.46	9.24 ± 0.27	0.49 ± 0.06	46.08 ± 8.07	8.77 ± 0.19	0.55 ± 0.07	64.40 ± 13.74
F-test	ns	**	*	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns	ns

* = Significant at 5% probability level, ** = significant at 1% probability level, ns = non-significant, DF = degree of freedom. Values in each column followed by different letters indicate they are significantly different at $p < 0.05$. Least Significant Difference (LSD) was the method of mean separation used.

Figure 5A,D illustrates the changes in total phenolic compound (TPC) content (mg GAE·mL^{-1}) and disease severity index (% DSI) over a 10-day period following inoculation (DAI). Both varieties responded to the three experimental treatments—Control, Whitefly infestation (WF), and Grafted (GT)—with varied TPC dynamics.

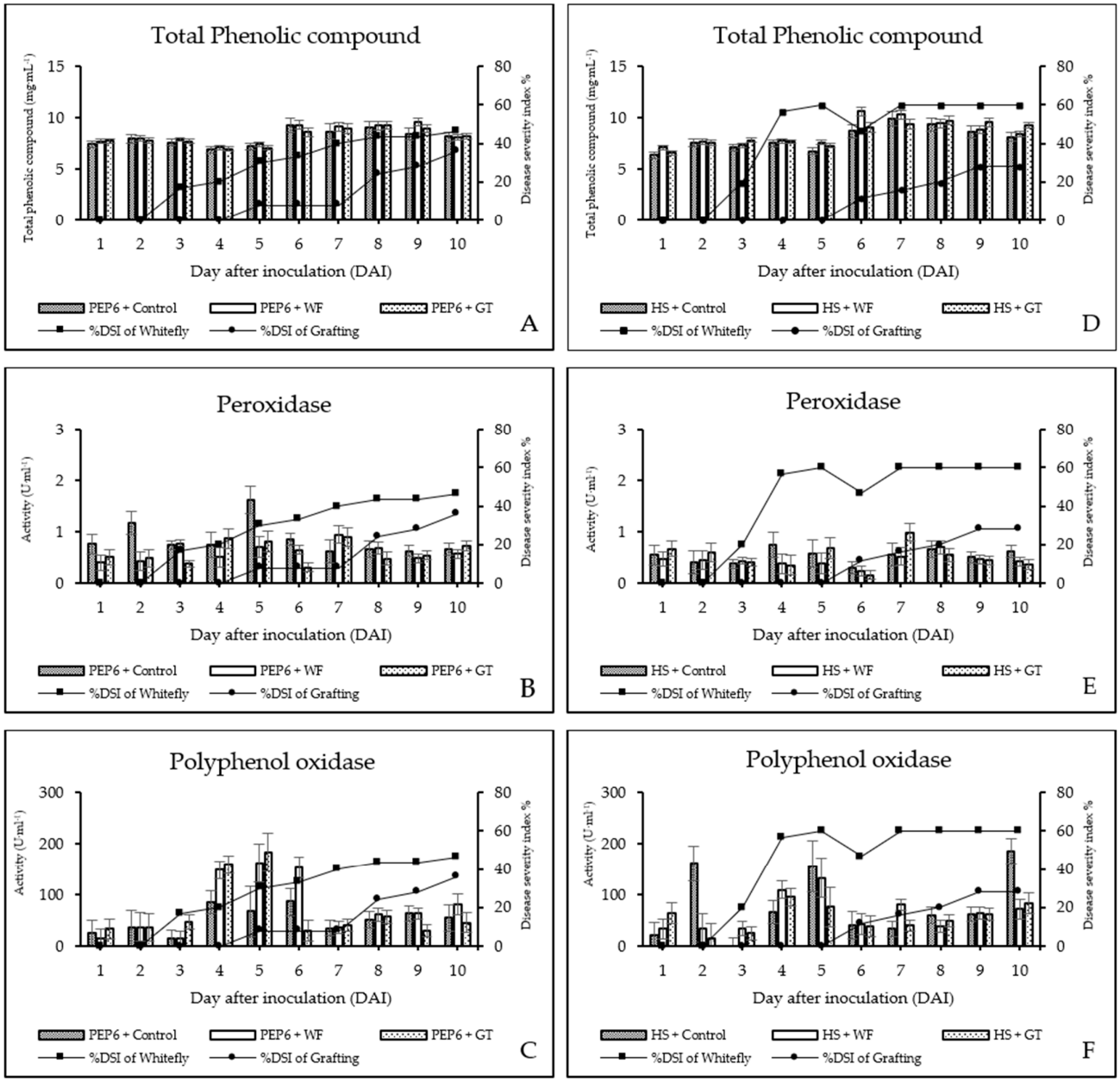


Figure 5. Changes in total phenolic compounds, peroxidase, and polyphenol oxidase activity in response to viral infection over a period of 10 days. Data are expressed as the means of treatment, with a vertical bar designating the standard deviation. (**A**–**C**) represents PEP6 (**A**–**C**), and (**D**–**F**) represent Homsuphan after inoculation by whitefly (WF), grafting (GT), and control.

In the PEP6 + Control, TPC increased slightly from 7.42 ± 0.37 mg GAE·mL^{-1} on day 1 to about 8.17 ± 0.42 mg GAE·mL^{-1} by day 10, remaining relatively stable throughout the experiment. In contrast, the PEP6 + WF group exhibited a distinct trend: TPC levels began at approximately 7.72 ± 0.23 mg GAE·mL^{-1} on day 1, remained steady until day 3, and then increased sharply, peaking at around 8.13 ± 0.27 mg GAE·mL^{-1} by day 10 (Figure 5A). The PEP6 + GT group followed a similar trajectory to the control, with a slight increase from around 7.76 ± 0.23 mg GAE·mL^{-1} to 8.21 ± 0.27 mg GAE·mL^{-1} over the 10 days.

TPC levels in this group consistently remained below those observed in the WF group. A strong correlation was observed between whitefly infestation and increased accumulation of phenolic compounds. The PEP6 + WF group, which exhibited the highest disease severity, also recorded the highest TPC levels. In contrast, the PEP6 + GT group showed lower disease severity and a milder rise in TPC compared to the WF group (Figure 5A). In the Homsupan variety, TPC showed a modest increase across all three treatments during the 10-day post-inoculation period. However, the differences among control, WF, and GT conditions were not statistically significant, with phenolic content remaining relatively consistent throughout the experiment (Figure 5D).

3.3. Defense Enzyme Assay

3.3.1. Peroxidase (POD) Activity

The temporal progression of peroxidase activity (POD) and disease severity index (DSI) varied significantly across genotypes and inoculation strategies. On Day 6, PEP6 exhibited notably higher POD compared to Homsupan (HS) (Table 2), suggesting a stronger oxidative response. Both whitefly (WF) and grafting (GT) methods influenced POD levels, with WF-induced inoculation showing significantly elevated POD on Days 3 and 6 (Table 3). In PEP6, POD increased from Day 1 through Day 7, then declined thereafter (Figure 5B). Under controlled conditions, POD remained low, with a minor spike around Day 5, and DSI reached ~50% by Day 10. Whitefly exposure moderately elevated POD between Days 4 and 7, with DSI stabilizing at ~45%. Grafting induced the highest POD levels, peaking on Day 5, while maintaining the lowest DSI (~40%) throughout the 10-day span. Conversely, HS plants showed a reversed POD trend: activity decreased until Day 6 and then rose slightly by Day 10. Control plants maintained low POD throughout, with DSI nearing ~50%. Whitefly exposure resulted in mild increases in POD at earlier time points (Days 3 and 7), with a final DSI of ~50%. Grafted HS plants demonstrated a significant POD surge around Day 7 and achieved a dramatically reduced DSI (~20%) by Day 10 (Figure 5E). Taken together, grafting consistently enhanced POD levels and suppressed disease severity across both varieties. PEP6 responded more swiftly and robustly to biotic stress, while HS benefited significantly from graft-induced defense activation at later stages.

3.3.2. Polyphenol Oxidase (PPO) Activity

Polyphenol oxidase (PPO) activity exhibited significant differences across chili genotypes and inoculation methods at days 4, 6, and 10 (Table 2). PEP6 consistently showed higher PPO activity than Homsupan (HS) on days 4 and 6, a trend that reversed by Day 10. Inoculation method further influenced PPO levels, with virus-infected plants—via whitefly transmission (WF) and grafting (GT)—displaying higher PPO activity on Day 4 than their respective controls (Table 3). In PEP6, control plants maintained low PPO activity throughout, with a mild increase around Day 7. WF-inoculated plants exhibited elevated PPO activity, peaking near day 7, and recorded a DSI of ~40% by day 10. GT-inoculated plants demonstrated the highest PPO activity, peaking around Day 5, accompanied by the lowest disease severity (<30%) across the timeline. In contrast, the HS control plants showed consistently low PPO activity, with subtle peaks occurring around Days 3 and 7. WF inoculation resulted in a moderate increase in PPO, peaking near Day 7, but corresponded with a notably high DSI (~60%) by Day 10. GT treatment in HS led to sustained high PPO activity from day 7 onward, culminating in a marked reduction in DSI (~20%) at Day 10. These results confirm that grafting effectively enhances PPO activity and suppresses disease progression in both chili varieties. PEP6 showed faster and stronger PPO induction post-grafting, while HS benefited from delayed yet sustained enzymatic defense, reinforcing the strategic advantage of GT-mediated inoculation in managing viral susceptibility.

4. Discussion

The comparative analysis of disease progression across two chili genotypes, PEP6 (tolerant to PepLCV and Homsuphan (susceptible to PepLCV), revealed a distinct disparity in response to whitefly-transmitted (WT) and grafting (GT) inoculation methods. Rapid onset and escalation of disease severity in both genotypes under WT conditions underscores the efficiency of whiteflies as viral vectors and validates the known susceptibility gradient, with HS being highly susceptible and PEP6 showing mild susceptibility. In contrast, grafted plants (PEP6 + GT and HS + GT) displayed a more protracted disease trajectory, with DSI plateauing at 80–85% by day 36. This delay suggests that grafting confers partial disease mitigation, likely by activating defense mechanisms through physiological reprogramming. As supported by Suwor et al. [26], the grafting process can induce systemic resistance through interactions between the scion and rootstock. Wound repair, vascular reconnection, and translocated signaling compounds may collectively upregulate defense-related metabolites or trigger transcriptional changes in defense genes [7,27–31]. While grafting did not fully prevent disease establishment, the slower pace of symptom progression points to a form of acquired tolerance rather than absolute resistance. This tempered disease response may be modulated by multiple factors, including pathogen virulence, the initial inoculum load, or the compatibility between the scion and rootstock. The contrasting outcomes observed between WT and GT treatments further highlight the distinct transmission pathways and host-plant response mechanisms. Whitefly inoculation delivers the pathogen directly into the vascular tissue, facilitating rapid systemic infection and symptom development [32]. The efficiency of viral transmission via whitefly is remarkably high; autophagic processes within the insect vector modulate viral titers [33], and even a single whitefly can transmit PepLCV to 66.6% of plants, with complete transmission reported using eight vectors [34]. By comparison, grafting may activate a multilayered defensive response, possibly involving oxidative bursts, metabolite accumulation, and transcriptional priming [35].

The total phenolic compound (TPC) profiles of PEP6 and Homsupan revealed distinct temporal patterns following inoculation, with notable genotype-specific differences in the early defense phase. PEP6 exhibited a significantly higher TPC (7.68 ± 0.17 mg GAE·mL^{-1}) than Homsupan (6.77 ± 0.17 mg GAE·mL^{-1}) on day 1 post-inoculation (Table 2), suggesting a faster or more robust early activation of phenolic-mediated defense mechanisms. However, Homsupan showed progressive upregulation of TPC levels, reaching parity with PEP6 from day 2 to day 10 (Table 3), indicating its capacity for sustained defense induction. This phenolic escalation coincided with the onset and advancement of symptoms under whitefly-transmitted (WT) conditions, implying a stress-induced biosynthetic response [36]. In WT treatments, both genotypes showed elevated TPC compared to controls, aligning with the known role of phenolic compounds as antioxidants, antimicrobial agents, and defense signaling molecules [37]. Notably, PEP6 + WF plants exhibited high DSI and high TPC, whereas PEP6 + GT plants maintained lower DSI alongside moderate phenolic accumulation, suggesting that phenolic levels alone may not confer effective disease suppression. This reflects the complex interplay between biochemical response and pathogen pressure, where TPC accumulation is indicative of defense activation but not necessarily resistance outcome [38].

The consistent observation that grafted plants of both PEP6 and Homsupan exhibited elevated peroxidase (POD) activity and reduced disease severity index (DSI) compared to control and whitefly-exposed counterparts represents a significant defense-enhancement outcome. This finding strongly supports the hypothesis that grafting can induce a resistant defense mechanism [39]. PODs are pivotal in reinforcing plant defenses through multiple biochemical pathways—namely, cell wall lignification, cross-linking of structural proteins,

and the generation of reactive oxygen species (ROS). These functions not only strengthen physical barriers but also initiate cascades of defense-related signaling. The distinct temporal profiles between genotypes—PEP6 with a more pronounced and delayed POD peak under whitefly challenge, and Homsupan with less responsive dynamics—underscore the genotype-specific defense architectures at play. Importantly, grafting consistently enhanced POD levels and suppressed DSI across both varieties, irrespective of their inherent susceptibility. This suggests grafting initiates a universal physiological remodeling, potentially involving vascular reprogramming, ROS signaling, and localized defense priming, ultimately amplifying stress resilience. The elevated POD activity in grafted plants, as supported by the roles of peroxidases in lignin biosynthesis and pathogen-triggered ROS bursts [40,41], likely forms a cornerstone of this graft-induced defense enhancement.

This study consistently highlights the pivotal role of polyphenol oxidase (PPO) in the defense mechanisms of chili plants against viral infection. Both cultivars demonstrated an inducible PPO response following both WT and GT inoculations. Notably, PEP6 exhibited a faster and more pronounced early induction of PPO, whereas Homsupan showed a more prolonged and sustained activation. The most compelling observation is that grafting consistently resulted in significantly higher PPO activity and substantially lower disease severity across both genotypes, suggesting that grafting is an effective strategy for enhancing plant resistance, likely by priming the PPO-mediated defense pathway [42,43]. However, it would be critical to further investigate the induced resistance mechanism by considering the cost of such management practice.

5. Conclusions

Grafting and whitefly inoculation significantly impact disease progression and defense responses in PEP6 and Homsupan chili genotypes. Whitefly-transmitted inoculation induced rapid and severe disease development, especially in the highly susceptible Homsupan. In contrast, grafting disease progresses slowly in both genotypes. Key biochemical markers, peroxidase activity, and total phenolic content exhibited distinct temporal patterns and genotypic responses. Elevated POD activity in grafted plants, particularly in PEP6, was associated with strengthened structural barriers and defense signaling. Phenolic accumulation correlated with pathogen pressure but varied in effectiveness depending on the transmission route. Together, these findings emphasize the complex interplay between inoculation method, varietal susceptibility, and enzymatic defense responses.

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