agronomy

MDPI

Article

Influence of Weather Conditions and the Aphid Population on the Potato Virus Y Infection of Tobacco in the Field

Marcin Przybyś [1,*], Teresa Doroszewska [1], Andrzej Doroszewski [2] and Tomasz Erlichowski [3]

[1] Institute of Soil Science and Plant Cultivation—State Research Institute, Department of Plant Breeding and Biotechnology, 24-100 Puławy, Poland; dorter@iung.pulawy.pl
[2] Institute of Soil Science and Plant Cultivation—State Research Institute, Department of Agrometeorology and Applied Informatics, 24-100 Puławy, Poland; ador@iung.pulawy.pl
[3] Plant Breeding and Acclimatization Institute—National Research Institute in Radzików, Bonin Division, Department of Potato Protection and Seed Science at Bonin, 76-009 Bonin, Poland; t.erlichowski@ihar.edu.pl
* Correspondence: mprzybys@iung.pulawy.pl

Abstract: Potato virus Y (PVY) is a major tobacco (*Nicotiana tabacum* L.) pathogen that causes severe crop losses. We studied the influence of meteorological factors and a population of twelve aphid species on the development of PVY in field-grown tobacco from 1996 to 2010 in Poland. Three PVY-susceptible tobacco varieties were used in the study. The mean virus incidence ranged from 18% in 2010 to almost 99% in 1996, 2004, and 2009. For determining the relationship between tobacco plant infection and meteorological conditions and aphid populations, logistic regression analysis was used. It was found that the probability of PVY infection is significantly dependent on the average air temperature, relative humidity, number of days with an average temperature of at least 25 °C, and the abundance of *Aphis fabae* and *Brachycaudus helichrysi*. The probability of infection of tobacco plants with potato virus Y decreased with increasing air temperature and relative humidity. In addition, with each subsequent day with a temperature of at least 25 °C, the risk of infection decreased by 24%. Furthermore, it was often observed that high populations of *Aphis fabae* and *Brachycaudus helichrysi* were associated with a high incidence of virus infection in tobacco plants.

Keywords: potato virus Y; PVY; epidemiology; meteorological factors; aphid; logistic regression

Citation: Przybyś, M.; Doroszewska, T.; Doroszewski, A.; Erlichowski, T. Influence of Weather Conditions and the Aphid Population on the Potato Virus Y Infection of Tobacco in the Field. *Agronomy* **2024**, *14*, 1725. https://doi.org/10.3390/agronomy14081725

Academic Editors: Zuqing Hu, Huiyan Zhao and Lei Zhao

Received: 10 June 2024
Revised: 1 August 2024
Accepted: 2 August 2024
Published: 5 August 2024

1. Introduction

Virus disease epidemiology depends on the interactions among viruses, vectors, plants, and the environment. Potato virus Y, a member of the family Potyviridae and the genus Potyvirus [1], infects many solanaceous crops such as potato (*Solanum tuberosum*), tobacco (*Nicotiana tabacum*), pepper (*Capsicum annuum*), and tomato (*Lycopersicon esculentum*) [2,3], as well as solanaceous weeds and ornamentals [4]. It is naturally transmitted by aphids in a non-persistent manner, causing epidemics in crops [3].

PVY originated in South America which is where potatoes were first domesticated. This pathogen was probably first taken from South America to Europe in the 16th century in tubers of potatoes [5], while it was discovered in Europe in 1931 [6]. The PVY^N strain was first found in 1935 on tobacco [7] and, since then, has been reported worldwide, causing significant yield losses [8,9] of up to 70% on potato [10,11] and almost 100% on susceptible cultivars of tobacco [12]. The annual economic losses caused by PVY in potato production in the UK alone is estimated at over GBP 30 million [13]. In the European Union, the annual losses were estimated at EUR 187 million [14], and in Idaho (USA) at USD 19.5 million [15]. Unfortunately, there are no estimates of the economic losses caused by PVY in different crops on a global scale.

The PVY genome contains a single strand of RNA of approximately 9700 nucleotides in length, encoding a polyprotein of 3063 amino acids [16]. Inside the host cell, the polyprotein is digested by viral proteases. This results in the formation of active viral proteins. The

multifunctional Helper Component Protease (HC-Pro) protein plays a key role in the transmission of the virus [17,18]. The C-terminal domain of the HC-Pro protein has been demonstrated to exhibit protease activity [19]. In contrast, the N-terminal and middle part of the protein forms the helper component (HC) domain [20,21]. Consequently, the HC-Pro protein is involved in several processes during aphid feeding on infected plants [20,22,23], including virus movement from cell to cell [24] and long-distance movement [25,26]. The primary function of the coat protein (CP) is to safeguard the genomic RNA of the virus and facilitate its transmission through the vector [27]. The capability of the aphid to transmit the virus is accountable upon the DAG motif present in the N-terminal region of the protein [23,28,29]. It should be noted that the PVY genome codes many proteins, including those present in the cytoplasm of infected cells, such as the CI (Cylindrical Inclusion) protein [30], as well as those present in the host cell nucleus such as NIa (Nuclear Inclusion a) [31,32] and NIb (Nuclear Inclusion b) proteins [33]. Other proteins include 6K1, which acts as a viroporin [34]; 6K2, which is engaged in viral particle long-distance movement and viral genome replication [35,36]; P1 protein, which is an auxiliary element in viral genome replication [37] and is associated with the movement of the virus from cell to cell [38]; and P3 protein, which is part of the replication complex [39,40].PVY isolates can be divided into several strain groups according to serological properties, host response, and resistance gene interactions [41]. The strains include two main biological groups: ordinary strain (PVY^{O}), tobacco vein necrosis strain (PVY^{N}), and several minor strains such as PVY^{C}, PVY^{Z}, PVY^{E}, and PVY^{D} [42–44]. The origin of PVY^{N} is Andean, but that of PVY^{O} and PVY^{C} is unknown [5]. Mutations and recombination events between some of these strains led to the formation of new important strains: $PVY^{N:O}$ and $PVY^{N\text{-}Wi}$, which have a PVY^{N} pathotype but a PVY^{O} serotype, and PVY^{NTN}, which induces systemic vein necrosis on tobacco and tuber necrotic ringspot disease (PTNRD) on susceptible cultivars of potato [2]. In tobacco, PVY induces vein necrosis, which inhibits the transport of water and mineral salts to leaf tissues, while leaf blade necrosis reduces the capacity for assimilation and gas exchange, thus inhibiting plant growth [45,46]. Serological and molecular techniques are widely used to detect infections with various PVY strains. Recently, metabolomic analyses based on the gas chromatography–mass spectrometry (GC-MS) technique were used for this purpose. It has been shown that infection with PVY^{NTN}, $PVY^{N\text{-}Wi}$, and PVY^{O} strains causes changes in the regulation of carbohydrate metabolism and the production of several common and strain-specific metabolites in the host. As a result, infection caused by different strains of PVY can be distinguished by metabolomic analysis [47].

The dissemination of PVY in the environment is markedly affected by meteorological conditions; e.g., air temperature, humidity, and precipitation affect viral infections of plants. For viral infections, the biggest impact is the air temperature and humidity. The optimal temperature for the growth of different viruses can vary considerably. For example, the optimal temperature for potato virus X is within the range of 20–24 °C. A temperature exceeding 25 °C resulted in a reduction in virus concentration within tissues and a decrease in disease symptoms. However, a temperature above 34 °C was observed to completely prevent the multiplication of the virus in the plant [48]. The temperature at which PVY multiplication occurs in the plant and disease symptoms develop varies depending on the virus strain. The optimal temperature for multiplication is between 22 °C and 26 °C with PVYN, while it falls between 14 °C and 22 °C with PVYO [49]. Similar results were obtained by Del Torro [50], studying the effects of growing *N. benthamiana* plants infected with PVY at 25 °C or 30 °C on virus accumulation and symptom expression. PVY accumulation decreased markedly at 30 °C and there were few or no symptoms.

The development of viral infections is also affected by the relative air humidity (RH). The optimum RH is 80% both before and after infection by aphids. A high relative humidity of 80% in both pre and post inoculation phases enhanced host susceptibility. The efficiency of virus transmission was reduced by almost 50% when the relative humidity was 50% [51].

The impact of meteorological conditions on the incidence of PVY infection in the field remains inadequately documented. It is widely acknowledged that air temperature

is a significant factor that can influence the disease in infected plants [49,52]. In field conditions, PVY is transmitted through the feeding of aphids, which act as vectors. The efficiency of virus infection is dependent upon the temperature at which the aphids feed, which must be within an optimal range. The efficiency of virus acquisition by aphids and transmission to the host plant is optimal at 20 °C for the PVYO strain. An increase in temperature to 25 °C has been demonstrated to result in a significant reduction in the efficiency of virus acquisition [53]. Winged aphids are known to fly when conditions are quite warm; however, temperatures that are too high can result in a reduction in certain aphid populations [54]. Based on data from the meteorological station covering 152 years (1871–2022), it was found that the mean air temperature in Puławy, Poland, increased in this period by 2.0 °C, and during last 100 years by 1.35 °C [55]. Models of climate change indicate a gradual increase in global average temperatures to 4.6 °C by 2100 [56]. This, in conjunction with the direct consequences of climate change, such as increases in temperature and, indirectly, the abundance and activity of vectors, could have a significant impact on plant virus epidemics [53,57]. Aphids exhibit a pronounced response to minor fluctuations in mean temperatures. It is anticipated that an additional five generations of aphids will emerge in temperate zones with a warming of 2 °C. Consequently, the probability of significant epidemics of aphid-transmitted viruses rises in tandem with the expansion of their populations and activities. In temperate regions, the survival of aphid vectors is anticipated to improve with more moderate mean winter temperatures, while higher mean summer temperatures are likely to enhance their development and reproductive rates. A reduction in the number of days with frost and the duration of cold spells will enhance their capacity to survive the winter period, thereby enabling them to expand their geographic range and extend the duration of their activity each year [58]. Aphid flights are monitored in many countries. The risk of virus transmission is frequently assessed in terms of vector pressure, which is determined by multiplying the population of each aphid species by its associated relative transmission efficiency factor (REF value) [59,60] and then adding the results together for all species [61–64]. While species-specific REF values are obtained in laboratory experiments, they are inadequate for capturing the full range of behavioural aspects of vectors that could impact virus epidemics in the field [65]. In fact, it has been postulated that aphid species that are unable to settle on potato or tobacco may play a more significant role as PVY vectors than is currently considered based on REF values alone [66–68]. The aphid species that act as the most significant PVY vectors may differ geographically. In the field, Aphis fabae [64] has been identified as the primary vector in Finland, while Brachycaudus helichrysi and Phorodon humuli [65] have been documented as the predominant vectors in Switzerland. Surprisingly, Myzus persicae, considered the most efficient PVY vector, was not correlated to virus infection in the field in any of these countries [65].

The creation of strategies to prevent and control PVY necessitates an understanding of viruses, vectors, and plants and the interactions between them. The application of pesticides is not advised for the control of PVY, given the brief period of time that aphids require to transmit the virus [69–71]. Furthermore, in accordance with IPM (Integrated Pest Management) principles, the usage of pesticides should be minimised in order to safeguard the environment from contamination. An effective strategy for controlling the virus is to employ the use of virus-resistant cultivars, physical barriers, oil sprays, and crop rotation as an integrated approach [70–72]. The best strategy to manage diseases seems to be to prevent PVY infections by breeding resistant cultivars. In the case of PVY on tobacco and other plant species, the eukaryotic transcription initiation factor 4E (eIF4E), critical for virus replication, has been identified as a recessive resistance gene [73–75], although another resistance/tolerance gene from the wild species *Nicotiana africana* is also known [76,77]. In tobacco, the eIF4E gene family comprises six members [78]. CRISPR/Cas9-based combinatorial editing of eIF4E1 and eIF4E2 genes enhanced resistance to PVY [78,79].

Currently, there are insufficient data in the literature on the effects of the meteorological conditions on the tobacco PVY infection in the field. Prerequisite to undertaking this

research was the progressing global climate change. This work takes the opportunity of field tests in natural conditions grown at Puławy, Poland, in which the PVY infection was recorded, to investigate the relations between weather conditions, aphid populations, and potato virus Y development.

2. Materials and Methods

2.1. Plant Material

In this study, three cultivars of tobacco susceptible to PVY were used: Burley 21, K326, and NC 95. The experiment was conducted at the Institute of Soil Science and Plant Cultivation—State Research Institute at Puławy, Poland, in the years 1996 to 2010 under natural infection pressure. The production of seedlings was carried out in multi-pallets in a glasshouse. Seedlings were grown in a greenhouse under appropriate phytosanitary conditions. The planting of seedlings at the experimental field was performed in the second half of May. The experiments were conducted using a completely randomised block with 4 replications of 32 plants per plot, giving a total of 128 plants of each cultivar with a row-to-row spacing of 90 cm and plant-to-plant distance of 45 cm under standard conditions of fertilisation, without the use of plant protection products. The final infection of the tobacco plants was determined in the last week of August of each year when observations and serological tests were carried out.

2.2. Meteorological Data

Meteorological factors, including mean value of air temperature, maximum value of air temperature, sum of atmospheric precipitation, mean value of relative air humidity, number of days with mean temperature higher than or equal to 20 °C, number of days with mean temperature higher than or equal 25 °C, and interaction between mean value of temperature and precipitation, were acquired from the meteorological station of the Institute of Soil Science and Plant Cultivation—State Research Institute located in Puławy. The data used for the analysis were averages from 10-day periods from 1995 to 2010. The mean values of air temperature were transformed into the absolute scale expressed in Kelvin degrees so that none of the temperature values would be negative.

2.3. Collection and Identification of Aphid Flight

From mid-May to the end of August, aphid flight activity was observed using yellow traps (YPTs). The traps contained 1.5 litres of water with the addition of 0.03% Tween 20. The size of the YPTs was 25 cm × 35 cm × 8 cm. Four traps were situated at the periphery of the field, and the total yield of aphids captured in the traps was used for the subsequent analysis. During the midpoint of the season, the traps were replaced with new ones due to the fading of the yellow colour caused by sunlight [80], in order to prevent any alteration in the aphids' response to landing [81,82]. All captured aphids were identified using the relevant taxonomic keys [83–89].

2.4. Serological Tests

All plants were serologically tested to confirm that they were infected with PVY. Tobacco leaf samples were collected in the last week of August. The leaf extracts were tested by a double-antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA) [90]. Monoclonal antibodies (PVY monoclonal cocktail IgG, Bioreba AG, Reinach, Switzerland) were used according to the manufacturer's instructions. The antibodies were able to detect all PVY strains and, after improvement by the manufacturer in 2002, also atypical isolates such as PVY^{O}768 belonging to the PVY^{Ob} group. The absorbance of ELISA plates at 405 nm was measured at 30 min using a Tecan Sunrise™ ELISA reader (Tecan Group Ltd., Männedorf, Switzerland). The positive–negative result determination method was taken from Bioreba's ELISA data analysis instructions. The absorbance of each sample was measured twice and the average of the two readings was calculated. The averages were then sorted and a histogram was constructed. Data showing a small increase in optical

density (OD) were considered as the background. A sudden increase in OD determined the "step" that distinguished the potentially positive samples from the background. For values less than the "step", the mean and standard deviation (s) were determined, while the cut-off value was calculated using Equation (1):

$$\text{cut-off} = (\text{mean} + 3\,\text{s}) \times 1.1 \quad (1)$$

2.5. Statistical Analysis

We conducted data analysis using Statistica 13.3 (Tibco Software, Santa Clara, CA, USA). First, we examined the eventual differences in infection among the three susceptible cultivars. In the analysis of variance for infection, the cultivar effect was non-significant ($p = 0.1876$). Therefore, in a further analysis, these three susceptible varieties were analysed jointly. The dependent variable was the average virus incidence per year in the last week of August in a selected set of susceptible cultivars in all cases, with virus incidence defined here as the number of plants infected with the virus out of 32 plants in each replicate. The logistic regression model included the values of meteorological factors from 10-day periods and abundances of the aphid species as predictor variables. The statistical significance of individual regression coefficients was assessed using the odds ratio (OR) with 95% confidence interval and Wald statistic [91,92]. The classification table was used to validate the predictive accuracy of the logistic regression model [93]. For goodness-of-fit statistics, the Hosmer–Lemeshow (H-L) test was used. The accuracy was evaluated by the size of the area under the curve (AUC) using receiver operating characteristics (ROCs). AUC indicates the ability to discriminate the plant infection status (0.9–1: excellent, 0.7–0.9: good, 0.5–0.7: poor, <0.50: fail) [94]. The diagnostic sensitivity in the ROC curve is the actual number of plants among those with infection. The diagnostic specificity is calculated by the number of plants among those without infection.

The general prediction equation used was (2):

$$y = \frac{1}{1 + \exp\left(-\left(\beta_0 + \beta_t t + \beta_p p + \beta_{tp} tp + \beta_h h + \beta_{t20} t_{20} + \beta_{t25} t_{25} + \beta_{tmax} t_{max} + \beta_{Mp} Mp + \cdots + \beta_{Ap} Ap\right)\right)} \quad (2)$$

where β_0—intercept; t—mean value of air temperature from 10-day period; p—precipitation during 10-day period; tp—interaction between mean value of temperature and precipitation during 10-day period; h—relative humidity from 10-day period; t_{20}—number of days with temperature greater than or equal to 20 °C; t_{25}—number of days with temperature greater than or equal to 25 °C; t_{max}—value of max air temperature during 10-day period; Mp—the sum of the *Myzus persicae* Sulz. aphids caught in the YPTs; An—the sum of the *Aphis nasturtii* Kalt. aphids caught in the YPTs; Afr—the sum of the *Aphis frangulae* Kalt. aphids caught in the YPTs; Af—the sum of the *Aphis fabae* Scop. aphids caught in the YPTs; Bh—the sum of the *Brachycaudus helichrysi* Kalt. aphids caught in the YPTs; Hl—the sum of the *Hyperomyzus lactucae* L. aphids caught in the YPTs; Cg—the sum of the *Cryptomyzus galeopsidis* Kalt. aphids caught in the YPTs; Ca—the sum of the *Cavariella aegopodii* Scop. aphids caught in the YPTs; Bb—the sum of the *Brevicoryne brassicae* L. aphids caught in the YPTs; Ct—the sum of the *Cavariella theobaldi* Gill. Bragg aphids caught in the YPTs; Rp—the sum of the *Rhopalosiphum padi* L. aphids caught in the YPTs; Ap—the sum of the *Acyrthosiphon pisum* Harris aphids caught in the YPTs.

3. Results

3.1. Variation in Virus Incidence among Years

In the years 1996–2010, important and significant variations in the number of plants with disease symptoms were observed. Generally, the first symptoms of PVY infection were observed 8 weeks (late June and July) after planting in the field, showing as vein clearing and then passing to vein necrosis. Vein necrosis was accompanied by chlorotic and necrotic spots on the leaf lamina, often leading to drying out of the leaves, and sometimes even stems. The increase in the number of infections was observed until the third week

of August. The final observations were carried out in the last week of August and are considered for this study. The mean virus incidence (percentage of plants with symptoms caused by PVY) per year ranged from 18% and 23% in 2010 and 2003 to almost 99% in 1996, 2004, and 2009, respectively (Figure 1).

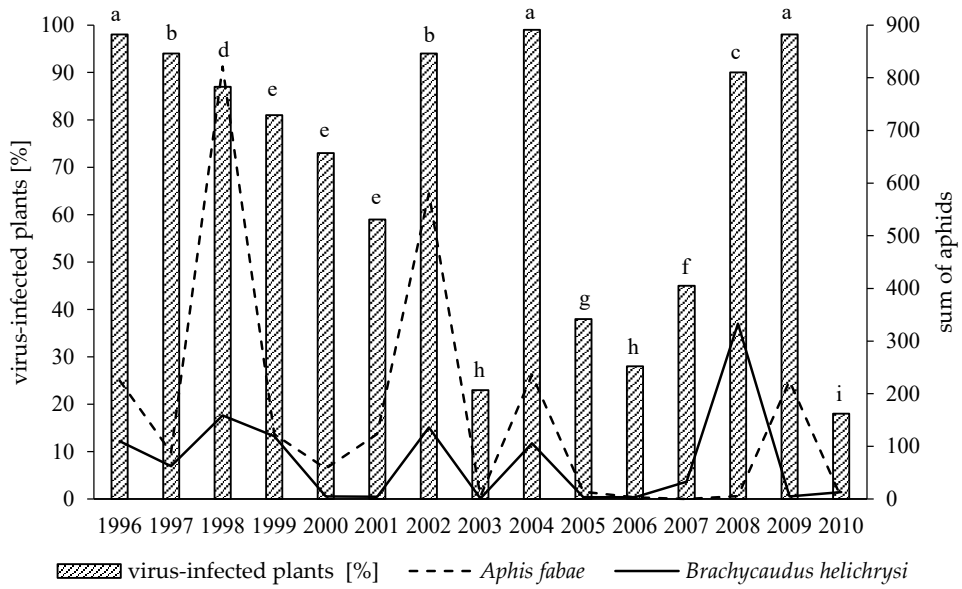

Figure 1. PVY infection of susceptible varieties of tobacco plants and abundance of aphid vectors. Values marked with different letters (a–i) for virus-infected plants are significantly different (Tukey's HSD, $\alpha = 0.05$).

3.2. Effect of Weather Conditions and Aphid Population on Virus Infections Caused by PVY

The potential impact of meteorological factors in all months of the year was investigated. The results from the tobacco plant infection were used in determining the relationship with the meteorological conditions and aphid population. For this purpose, logistic regression analysis was used. A total of thirty-six logistic regression models were tested. The overall model evaluation was based on the Hosmer–Lemeshow goodness-of-fit test. The results of the H-L test ($p > 0.05$) suggested that only one model (the first 10 days of July) fitted well to the data (Table 1).

Table 1. Test of significance of Hosmer–Lemeshow goodness-of-fit statistics.

	χ^2	df	*p*
Goodness-of-fit test	3.325	9	0.853

The Wald statistic and odds ratio with 95% confidence interval (CI) were used to assess the statistical significance of individual regression coefficients in the model (Table 2).

The probability of PVY infection is significantly dependent on the average air temperature, relative humidity, the number of days with an average temperature of at least 25 °C, and the abundance of *Aphis fabae* and *Brachycaudus helichrysi* (Table 2). The probability of infection of tobacco plants with potato virus Y decreases with increasing average air temperature and relative humidity (Figures 2 and 3). With each subsequent day with a temperature of at least 25 °C, the risk of infection decreased by 24%. Based on the statistical analyses (Table 2), a significant impact of two aphid species *Aphis fabae* and *Brachycaudus helichrysi* on the infection of tobacco plants was found. Unexpectedly, *M. persicae* was not significantly associated with virus incidence. The year-to-year variation in aphid abundance was large. Cumulative counts ranged from 0 (2007) to 822 (1998) for *A. fabae*, and from 1 (2003) to 333 (2008) for *B. helichrysi* (Figure 1). As the population of these aphids increased, the virus infection of plants also increased.

Table 2. Statistical tests of individual predictors.

Predictor	β	Wald Statistic	df	*p*	OR	95% CI for OR	
						Lower	Upper
intercept	1850.27	23.08	1	<0.001			
mean temperature (t)	−6.20	12.82	1	<0.001	0.002	0.000	0.004
precipitation (p)	−60.90	2.20	1	0.138	0.000	0.000	315.661
relative humidity (h)	−0.63	29.15	1	<0.001	0.535	0.426	1.000
max. temperature (max.t)	1.38	0.50	1	0.480	3.956	0.087	179
number of days t ≥ 20 (t_{20})	−2.13	0.08	1	0.774	0.119	0.000	242.013
number of days t ≥ 25 (t_{25})	−1.42	12.67	1	<0.001	0.241	0.110	1.000
interaction temp. x prec. (tp)	0.21	2.24	1	0.134	1.233	0.937	2.000
Myzus persicae (Mp)	−0.02	0.96	1	0.327	0.976	0.931	1.000
Aphis nasturtii An	−0.06	0.09	1	0.763	0.943	0.644	1.000
Aphis frangulae Afr	0.03	0.22	1	0.984	1.030	0.055	19.000
Aphis fabae Af	0.20	7.69	1	<0.001	1.224	1.220	1.228
Brachycaudus helichrysi Bh	0.57	6.54	1	<0.001	1.773	1.708	1.841
Hyperomyzus lactucae Hl	−0.18	1.32	1	0.148	0.832	0.629	1.101
Cryptomyzus galeopsidis Cg	−0.01	0.39	1	0.516	0.799	0.745	1.313
Cavariella aegopodii Ca	0.03	2.84	1	0.251	0.192	0.187	0.196
Brevicoryne brassicae Bb	−0.23	0.76	1	0.087	0.794	0.580	1.087
Cavariella theobaldi Ct	0.01	2.91	1	0.362	1.011	0.737	1.388
Rhopalosiphum padi Rp	−0.12	3.56	1	0.060	0.886	0.781	1.005
Acyrthosiphon pisum Ap	−0.01	1.74	1	0.862	0.989	0.879	1.114

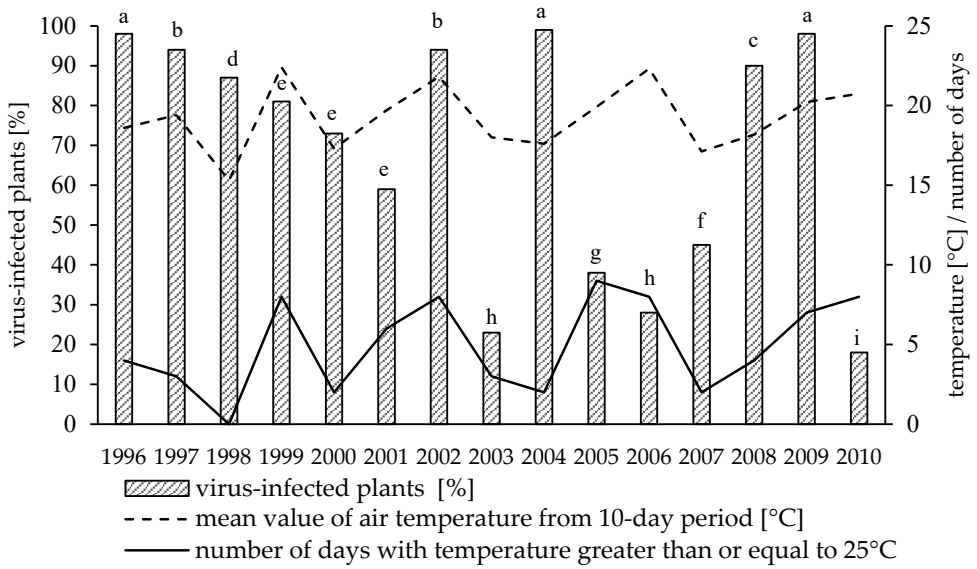

Figure 2. PVY infection of susceptible varieties of tobacco plants, mean value of air temperature, and number of days with temperature greater than or equal to 25 °C (meteorological conditions from first 10 days of July). Values marked with different letters (a–i) for virus-infected plants are significantly different (Tukey's HSD, $\alpha = 0.05$).

To evaluate the predictive accuracy of the logistic regression model, the classification table method and discrimination with ROC curves were used. The overall correct prediction was 81.68%. It shows an improvement over the chance level, which is 50% (Table 3). The diagnostic sensitivity of the model, that is, the percentage of correctly classified occurrences of plant infection, was 84.95%, while the diagnostic specificity, that is, the percentage of correctly classified lack of plant infection with the virus, was 73.72%. The evaluated logistic regression model classified 26.28% of false positive events and 15.05% of false negative events.

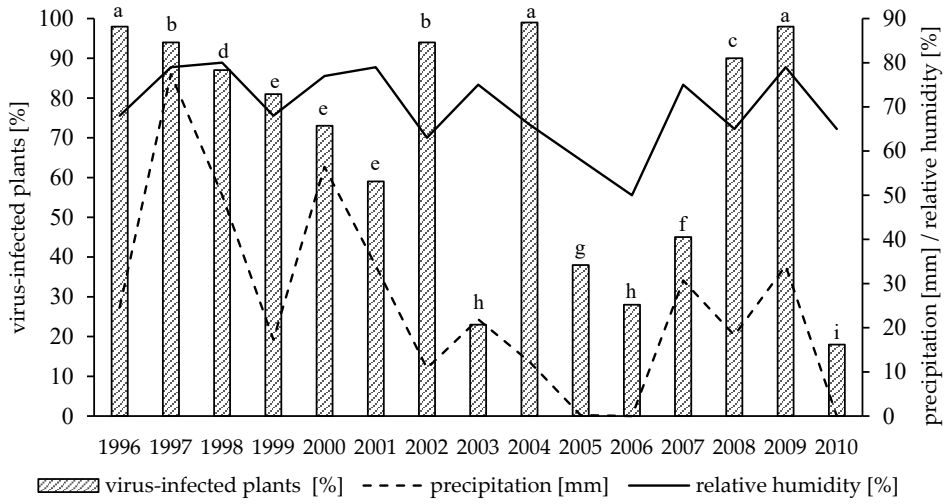

Figure 3. PVY infection of susceptible varieties of tobacco plants, precipitation, and relative humidity (meteorological conditions from first 10 days of July). Values marked with different letters (a–i) for virus-infected plants are significantly different (Tukey's HSD, $\alpha = 0.05$).

Table 3. A classification table of predictive accuracy of the logistic regression model.

Observed	Predicted		% Correct
	Yes	No	
Yes	852	151	84.95
No	108	303	73.72
Overall % correct			81.68

Diagnostic sensitivity = 852/(852 + 151) = 84.95%; diagnostic specificity = 303/(108 + 303) = 73.72%; false positive = 108/(108 + 303) = 26.28%; false negative = 151/(852 + 151) = 15.05%.

As a result of the statistical analyses, a ROC curve was plotted (Figure 4). The AUC value was 0.8739, which indicates a good model fit [95].

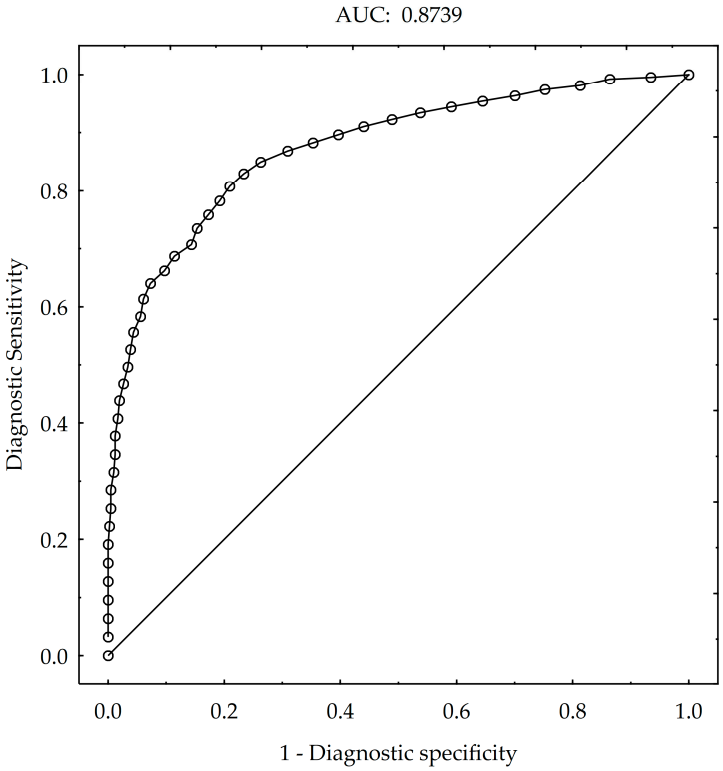

Figure 4. The ROC curve of fit of the model.

The final prediction equation used was (3):

$$y = \frac{1}{1 + \exp(-(1850.27 - 6.2t - 0.63h - 1.42t_{25} + 0.2Af + 0.57Bh))} \quad (3)$$

4. Discussion

The main aim of this study was to identify the most important weather conditions and aphid species influencing PVY tobacco plant infections in Poland. The performed statistical analysis has shown that the PVY infection of tobacco plants was dependent on weather conditions. According to Köppen's classification, the area of Poland is located in a humid continental climate zone (Dfb) [96,97]. The average summer daily air temperatures in the analysed period (1996–2010) were 19.3 °C and 18.4 °C in July and August, respectively, but there were days with the maximum temperature even reaching 35.0 °C, such as in July and August 2010. The average winter temperature was −2.1 °C and −2.0 °C in January and February, respectively, but there were days when the minimum temperature dropped to −29.5 °C, such as in January 2006.

Temperature is an environmental parameter that differentially affects the interaction of hosts with RNA viruses [50]. The weather conditions prevailing at the beginning of July in Puławy had a significant impact on the infection of tobacco plants with the virus. Both high average air temperature ($p < 0.001$), relative humidity ($p < 0.001$) and the number of days with a persistent high average temperature above 25 °C ($p < 0.001$) resulted in lower frequency of PVY symptoms on susceptible tobacco plants (Table 2). The reduction in the ability of aphids to acquire PVY, the establishment of infection, and the accumulation of the virus in plant tissues are the primary causes [53]. It has been proposed that elevated temperatures may enhance the efficacy of gene-silencing-based antiviral defences. However, in the case of infection of *Nicotiana benthamiana* by a PVY isolate, it was observed that, at relatively elevated temperatures (30 °C), the antiviral silencing defence could be suppressed by the virus [53,98,99]. In the study conducted by Choi et al., systemic PVY infection was only observed at temperatures between 16 °C and 32 °C. An increase in temperature led to a reduction in the time required to induce the systemic infection of plants. At 35 °C, the complete inhibition of infection was observed [100]. Due to observed climate change, temperature is one of the environmental variables expected to influence the severity or prevalence of viral diseases [101–103]. For example, the incidence of viruses in the summer months was associated with the average temperature in winter. Higher winter temperatures increased the persistence of weeds, which are reservoirs for viruses and shelter for insects. This resulted in the earlier establishment of larger aphid populations, leading to a higher virus incidence [103–105]. The impact of weather conditions in January and February, the coldest months in Poland, was also examined. It was shown that the average temperature in this period was statistically significant, but it was not included in the final model due to the insufficient goodness-of-fit of the model (Hosmer–Lemeshow test, $p < 0.05$).

Although meteorological conditions exert a considerable influence on PVY infection in tobacco, our results suggest that only a portion of the observed variability in PVY infection may be attributable to them. This is indicated by the results obtained, which show that seven variables (meteorological factors) were initially entered into the regression analysis, but only three of them were statistically significant. It is also important to consider other factors that may influence the outcome, such as the initial source of the virus inoculum, the efficiency of PVY transmission, the variable composition of virus strains, and the phenomenon of mature plant resistance. A very important factor affecting the spread of PVY to healthy plants is the initial source of inoculum in the field [106]. The source of inoculum in the field may originate from PVY-infected potato seed tubers or weeds that subsequently provide virus-infected foliage to the aphids [45,77]. In the field, a mixture of different strains of PVY occurs. The optimal temperature for virus development varies depending on the dominant strain, which affects the infection severity. The efficiency of PVY transmission is determined by the specific strain of the virus. Previous research has indicated

that PVY^{N} is more effectively transmitted than PVYO [107], and that $PVY^{N\text{-}Wi}$ is more efficient at transmitting the infection to progeny potato tubers than PVY^{NTN} [108]. Our additional research, conducted on tobacco, indicates a gradual increase in the proportion of the PVY^{NTN} strain in Poland. This strain has been observed to exhibit a greater ability to overcome the resistance of many cultivars [109]. A similar situation has been observed in Western Europe [110,111]. Furthermore, in the field, mixed infections involving various strains of PVY are frequently observed. Additionally, the maturity of the infected plant may influence the severity of the PVY infection. This specific phenomenon is referred to as mature plant resistance and is characterised by the restriction of the cell-to-cell movement of virus particles [112,113]. In the scenario of delayed aphid activity, the probability of plant inoculation is decreased due to the development of mature plant resistance [67]. Nevertheless, alterations in temperature have been observed to impact cell-to-cell movement, systemic movement, and replication, which subsequently affects the dissemination of the virus within the host [58,101,114].

A comprehensive understanding of the principal viral vectors is essential for the development of a predictive system for viral infections in tobacco production. The strong correlation between the annual incidence of the virus over 15 years and the abundance of *Aphis fabae* and *Brachycaudus helichrysi* suggests that these species may be the most important vectors of PVY on tobacco in Poland (Table 2). The abundance of *Myzus persicae*, typically regarded as the most efficient vector of PVY [67,115], was not found to be associated with the incidence of the virus. The highest numbers of *M. persicae* during the study period were caught in 2003 and 2007—622 and 738 individuals, respectively—although the virus prevalence was below average in both years (Figure 1). In these years, the emergence of *Myzus persicae* was observed later—more than a month after tobacco was planted in the field. This suggests that *M. persicae* was not the main PVY vector in our study. A similar phenomenon has also been observed in other countries [71,116–119]. In Poland, tobacco seedlings are planted in the field in the second half of May. The years 1998, 2002, 2004, 2008, and 2009 were characterised by the highest numbers of two species of aphids: *Aphis fabae* and *Brachycaudus helichrysi*. The high number of aphids of these two species coincided with the high infection of tobacco plants by PVY. In these years, only in 2008 was the population of *B. helichrysi* larger than that *A. fabae*.

The aphid species *M. persicae* had no significant effect on the PVY infection of tobacco plants. A similarly insignificant effect of *M. persicae* on the infection of potato plants by PVY was observed in the studies of Steinger [65]. They noted that *B. helichrysi* is a species that regularly alights on potato crops, despite its inability to colonise potato plants, and therefore concluded that non-colonisers can be efficient vectors of PVY because they engage in repeated cycles of alighting and probing during host plant selection [65,67,120,121]. The presence of *A. fabae* and *B. helichrysi* is already noticeable at the time of tobacco planting, while the appearance of *M. persicae* in greater numbers occurs a few weeks later. It seems reasonable to posit that the initial period after planting tobacco in the field may be crucial for the emergence of virus outbreaks [64,122,123].

This study has demonstrated that meteorological data and count data of winged aphids can be used to predict the risk of PVY infection in tobacco plantations.

5. Conclusions

A 15-year field study was conducted in south-eastern Poland to investigate the effects of weather conditions and aphid populations on PVY infection in tobacco plants. A total of three meteorological factors were identified as having a statistically significant impact on the prevalence of PVY in tobacco plants. The mean air temperature, relative humidity, and the number of days with a sustained temperature above 25 °C were found to have a significant effect. It was determined that temperatures higher than 25 °C and high relative humidity limit virus infection. Among the 12 aphid species, *Aphis fabae* and *Brachycaudus helichrysi* were identified as having a significantly positive effect on PVY infections.

Author Contributions: M.P.: Conceptualisation, methodology, software, formal analysis, investigation, data curation, visualisation, writing—original draft preparation. T.D.: Conceptualisation, methodology, investigation, resources, supervision, project administration, writing—review and editing. A.D.: Conceptualisation, methodology, investigation, resources, writing—review and editing. T.E.: methodology, resources, writing—review and editing. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Data Availability Statement: The data presented in this study are available on request from the corresponding author.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Aramburu, J.; Galipienso, L.; Matas, M. Characterization of potato virus Y isolates from tomato crops in northeast Spain. *Eur. J. Plant Pathol.* **2006**, *115*, 247–258. [CrossRef]
2. Nikolaeva, O.V.; Roop, D.J.; Galvino-Costa, S.B.F.; Figueira, A.R.; Gray, S.M.; Karasev, A.V. Epitope Mapping for Monoclonal Antibodies Recognizing Tuber Necrotic Isolates of Potato Virus Y. *Am. J. Potato Res.* **2012**, *89*, 121–128. [CrossRef]
3. DeBokx, J.A.; Huttinga, H. *Potato virus Y. CMI/AAB Descriptions of Plant Viruses No.242*; Commonwealth Microbiology Institute and Association of Applied Biology: Kew, UK, 1981.
4. Kaliciak, A.; Syller, J. New hosts of Potato virus Y (PVY) among common wild plants in Europe. *Eur. J. Plant Pathol.* **2009**, *124*, 707–713. [CrossRef]
5. Fuentes, S.; Jones, R.A.C.; Matsuoka, H.; Ohshima, K.; Kreuze, J.; Gibbs, A.J. Potato virus Y; the Andean connection. *Virus Evol.* **2019**, *5*, vez037. [CrossRef] [PubMed]
6. Smith, K.M. Composite nature of certain potato viruses of the mosaic group. *Nature* **1931**, *127*, 702. [CrossRef]
7. Smith, K.M.; Dennis, R.W.G. Some notes on a suspected variant of solanum virus 2 (potato virus Y). *Ann. Appl. Biol.* **1940**, *27*, 65–70. [CrossRef]
8. Shukla, D.D.; Ward, C.W.; Brunt, A.A. *The Potyviridae*; CAB International: Wallingford, UK, 1994; 516p.
9. Choi, H.-S.; Bhat, H.-I.; Park, J.-W.; Cheon, J.-U.; Kim, J.-S.; Pappu, H.-R.; Choi, J.-K.; Takanami, Y. Studies on Potato virus Y isolates infecting potato and tobacco in Korea. *J. Fac. Agric. Kyushu Univ.* **2004**, *49*, 253–262. [CrossRef] [PubMed]
10. Jacquot, E.; Tribodet, M.; Croizat, F.; Balme-Sinibaldi, V.; Kerlan, C. A single nucleotide polymorphism-based technique for specific characterization of Y^O and Y^N isolates of Potato virus Y (PVY). *J. Virol. Methods* **2005**, *125*, 83–93. [CrossRef] [PubMed]
11. Van der Zaag, D.E. Yield reduction in relation to virus infection. In *Viruses of Potatoes and Seed-Potato Production*, 2nd ed.; De Bokx, J.A., van des Want, J.P.H., Eds.; Pudoc: Wageningen, The Netherlands, 1987; pp. 149–150.
12. Latore, B.A.; Flores, V.; Marholz, G. Effect of Potato virus Y on growth, yield and chemical composition of flue-cured tobacco in Chile. *Plant Dis.* **1984**, *68*, 884–886. [CrossRef]
13. Valkonen, J.P.T. Viruses: Economical losses and biotechnological potential. In *Potato Biology and Biotechnology: Advances and Perspectives*; Elsevier BV: Amsterdam, The Netherlands, 2007; pp. 619–641.
14. Dupuis, B.; Nkuriyingoma, P.; Ballmer, T. Economic Impact of Potato Virus Y (PVY) in Europe. *Potato Res.* **2024**, *67*, 55–72. [CrossRef]
15. McIntosh, C. The economics of PVY. In Proceedings of the Idaho Potato Conferences, Pocatello, ID, USA, 22–24 January 2014.
16. Powell, A.P.; Nelson, R.S.; Barun de Hoffmann, N.; Rogers, S.G.; Fraley, R.T.; Beachy, R.N. Delay of disease development in transgenic plants that express the tobacco mosaic virus coat protein gene. *Science* **1986**, *232*, 738–743.
17. Carrington, J.C.; Freed, D.D. Cap-independent enhancement of translation by a plant potyvirus 5′ non translated region. *J. Virol.* **1990**, *64*, 1590–1597. [CrossRef] [PubMed]
18. Verchot, J.M.; Koonin, E.V.; Carrington, J.C. The 35-kDa protein from the N-terminus of a potyviral polyprotein functions as a third virus encoded proteinase. *Virology* **1991**, *185*, 527–535. [CrossRef] [PubMed]
19. Oh, C.S.; Carrington, J.C. Identification of essential residues in potyvirus proteinase HC-Pro by site-directed mutagenesis. *Virology* **1989**, *173*, 692–724. [CrossRef] [PubMed]
20. Atreya, C.D.; Atreya, P.L.; Thornbury, D.W.; Pirone, T.P. Site-directed mutations in the potyvirus HC-Pro gene affect helper component activity, virus accumulation and symptom expression in infected tobacco plants. *Virology* **1992**, *191*, 106–111. [CrossRef] [PubMed]
21. Atreya, C.D.; Pirone, T.P. Mutational analysis of the helper component-proteinase gene of a potyvirus: Effects of amino acid substitutions, deletions and gene replacement on virulence and aphid transmissibility. *Proc. Natl. Acad. Sci. USA* **1993**, *90*, 11919–11923. [CrossRef] [PubMed]
22. Wang, R.Y.; Ammar, E.D.; Thornbury, D.W.; Lopez-Moya, J.J.; Pirone, T.P. Loss off potyvirus transmissibility and helper-component activity correlate with non-retention of virions in aphid stylets. *J. Gen. Virol.* **1996**, *77*, 861–867. [CrossRef] [PubMed]
23. Blanc, S.; Lopez-Moya, J.J.; Pirone, T.P. *La Transmission par Pucerons du Tobacco Vein Mottling Virus Necessite une Interaction Entre HC-Pro et la Protéine de Capside*; Résumés, 48; Sixiemes Rencontres de Virologie Végétale: Aussois, France, 1997.

24. Rojas, M.R.; Zerbini, F.M.; Allison, R.F.; Gilbertson, R.L.; Lucas, W.J. Capsid protein and helper component-proteinase function as potyvirus cell-to-cell movement proteins. *Virology* **1997**, *237*, 283–295. [CrossRef]
25. Cronin, S.; Verchot, J.; Haldeman-Cahill, R.; Schaad, M.C.; Carrington, J.C. Long-distance movement factor: A transport function of the potyvirus helper component proteinase. *Plant Cell* **1995**, *7*, 549–559.
26. Saenz, P.; Salvador, B.; Simon-Mateo, C.; Kasschau, K.D.; Carrington, J.C.; Garcia, J.A. Host-specific involvement of the HC protein in the long-distance movement of potyviruses. *J. Virol.* **2002**, *76*, 1922–1931. [CrossRef]
27. Dolja, V.V.; Haldemann-Cahill, R.; Montgomery, A.E.; Vandenbosch, K.A.; Carrington, J.C. Capsid proteins determinants involved in cell-to-cell and long distance movement of tobacco etch potyvirus. *Virology* **1995**, *206*, 1007–1016. [CrossRef] [PubMed]
28. Roudet-Tavert, G.; Grerman-Retana, S.; Delaunay, T.; Delecolle, B.; Candresse, T.; Le Gall, O. Interaction between potyvirus helper component-proteinase and capsid protein in infected plants. *J. Gen. Virol.* **2002**, *83*, 1765–1770. [CrossRef] [PubMed]
29. Flasinski, S.; Cassidy, B.G. Potyvirus aphid transmission requires helper component and homologous coat protein for maximal efficiency. *Arch. Virol.* **1998**, *143*, 2159–2172. [CrossRef]
30. Shand, K.; Theodoropoulos, C.; Stenzel, D.; Dale, J.L.; Harrison, M.D. Expression of potato virus Y cytoplasmic inclusion protein in tobacco results in disorganization of parenchyma cells, distortion of epidermal cells and induces mitochondrial and chloroplast abnormalities, formation of membrane whorls and atypical lipid accumulation. *Micron* **2009**, *40*, 730–736. [PubMed]
31. Shahabuddin, M.; Shaw, J.G.; Rhoads, R.E. Mapping of the tobacco vein mottling virus VPg cistron. *Virology* **1988**, *173*, 499–508. [CrossRef] [PubMed]
32. Barrett, A.J.; Rawlings, N.D. Families and clans of cysteine peptidases. *Perspect. Drug Discov. Des.* **1996**, *6*, 1–11. [CrossRef] [PubMed]
33. Koonin, E.V. The phylogeny of RNA-dependent RNA polymerases of positive-strand RNA viruses. *J. Gen. Virol.* **1991**, *72*, 2197–2206. [CrossRef]
34. Chai, M.; Li, L.; Li, Y.; Yang, Y.; Wang, Y.; Jiang, X.; Luan, Y.; Li, F.; Cui, H.; Wang, A.; et al. The 6-kilodalton peptide 1 in plant viruses of the family Potyviridae is a viroporin. *Proc. Natl. Acad. Sci. USA* **2024**, *121*, 21. [CrossRef]
35. Tomimura, K.; Špak, J.; Katis, N.; Jenner, C.E.; Walsh, J.A.; Gibbs, A.; Ohshima, K. Comparisons of the genetic structure of populations of turnip mosaic virus in west and east Eurasia. *Virology* **2004**, *330*, 408–423. [CrossRef]
36. Merits, A.; Rajamäki, M.L.; Lindholm, P.; Runeberg-Roos, P.; Kekarainen, T.; Puustinen, P.; Mäkeläinen, K.; Valkonen, J.P.T.; Saarma, M. Proteolytic processing of potyviral proteins and polyprotein processing intermediates in insect and plant cells. *J. Gen. Virol.* **2002**, *83*, 1211–1221. [CrossRef]
37. Verchot, J.; Carrington, J.C. Debilitation of plant potyvirus infectivity by P1 proteinase-inactivating mutations and restoration by second-site modifications. *J. Virol.* **1995**, *69*, 1582–1590. [CrossRef] [PubMed]
38. Brantley, J.D.; Hunt, A.G. The N-terminal protein of the polyprotein encoded by by the potyvirus tobacco vein mottling virus is an RNA-binding protein. *J. Gen. Virol.* **1993**, *74*, 1157–1162. [CrossRef] [PubMed]
39. Klein, P.G.; Klein, R.R.; Rodriguez-Cerezo, E.; Hunt, A.G.; Shaw, J.G. Mutational analysis of the tobacco vein mottling virus genome. *Virology* **1994**, *204*, 759–769. [CrossRef] [PubMed]
40. Martin, M.T.; Cervera, M.T.; García, J.A.; Bonay, P. Properties of the active plum pox potyvirus RNA polymerase complex in defined glycerol gradient fractions. *Virus Res.* **1995**, *37*, 127–137. [CrossRef] [PubMed]
41. Coutts, B.A.; Jones, R.A. Potato virus Y: Contact transmission, stability, inactivation, and infection sources. *Plant Dis.* **2015**, *100*, 387–394. [CrossRef]
42. Singh, R.P.; Valkonen, J.P.; Gray, S.M.; Boonham, N.; Jones, R.A.; Kerlan, C.; Schubert, J. Discussion paper: The naming of Potato virus Y strains infecting potato. *Arch. Virol.* **2008**, *153*, 1–13. [CrossRef]
43. Kehoe, M.A.; Jones, R.A.C. Improving Potato virus Y strain nomenclature: Lessons from comparing isolates obtained over a 73-year period. *Plant Path* **2016**, *65*, 322–333. [CrossRef]
44. Jones, R.A.C.; Vincent, S.J. Strain-specific hypersensitive and extreme resistance phenotypes elicited by Potato virus Y among 39 potato cultivars released in three world regions over a 11-year period. *Plant Dis.* **2018**, *102*, 185–196. [CrossRef]
45. Przybyś, M.; Doroszewska, T.; Berbeć, A. Point mutation in the viral genome-linked protein (VPg) of Potato virus Y probably correspond with ability to overcome resistance of tobacco. *J. Food Agric. Environ.* **2013**, *11*, 132–135.
46. Verrier, J.L.; Marchand, V.; Cailleteau, B.; Delon, R. Chemical change and cigarette smoke mutagenicity increase associated with CMV-DTL and PVY-N infection in burley tobacco. In Proceedings of the Cooperation Centre for Scientific Research Relative to Tobacco Meeting Agro-Phyto Groups, Cape Town, South Africa, 30 September–4 October 2001.
47. Manasseh, R.; Berim, A.; Kappagantu, M.; Moyo, L.; Gang, D.R.; Pappu, H.R. Pathogen-triggered metabolic adjustments to potato virus Y infection in potato. *Front. Plant Sci.* **2023**, *13*, 1031629. [CrossRef]
48. Close, R. Some effects of other viruses and of temperature on the multiplication of potato virus X. *Ann. Appl. Biol.* **1964**, *53*, 151–164. [CrossRef]
49. DeBokx, J.A.; Piron, P.G.M. Effect of temperature on symptom expression and relative virus concentration in potato plants infected with potato virus YN and YO. *Potato Res.* **1977**, *20*, 207–213. [CrossRef]
50. Del Torro, F.J.; Aguilar, E.; Hernandez-Wallas, F.J.; Tenellado, F.; Chung, B.-N.; Canto, T. High temperature, high ambient CO2, affect the interactions between three positive-sense RNA viruses and a compatible host differentially, but not their silencing suppression efficiencies. *PLoS ONE* **2015**, *10*, e0136062. [CrossRef] [PubMed]

51. Singh, M.N.; Khurana, S.M.P.; Nagaich, B.B.; Agrawal, H.O. Environmental factors influencing aphid transmission of potato virus Y and potato leafroll virus. *Potato Res.* **1988**, *31*, 501–509. [CrossRef]
52. Glasa, M.; Labonne, G.; Quiot, J.B. Effect of temperature on plum pox virus infection. *Acta Virol.* **2003**, *47*, 49–52. [PubMed]
53. Chung, B.N.; Canto, T.; Tenllado, F.; Choi, K.S.; Joa, J.H.; Ahn, J.J.; Kim, C.H.; Do, K.S. The effects of high temperature on infection by Potato virus Y, Potato virus A and Potato leafroll virus. *Plant Pathol. J.* **2016**, *32*, 321–328. [CrossRef] [PubMed]
54. Matthews, R.E.F. *Plant Virology*, 3rd ed.; Elsevier BV: Amsterdam, The Netherlands, 1991; 864p.
55. Doroszewski, A. Warunki termiczne i opadowe w Puławach w latach z ekstremalnymi suszami rolniczymi w okresie 2006–2022. *Studia i Raporty IUNG-PIB* **2023**, *71*, 9–25.
56. IPCC. *Climate Change 2013: The Physical Science Basis. Contribution of Working Group I to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change*; Stocker, T.F., Qin, D., Plattner, G.K., Tignor, M., Allen, S.K., Boschung, J., Nauels, A., Xia, Y., Bex, V., Midgley, P.M., Eds.; Cambridge University Press: Cambridge, UK; New York, NY, USA, 2013; 1535p.
57. Jones, R.A.C. Plant virus emergence and evolution: Origins, new encounter scenarios, factors driving emergence, effects of changing world conditions, and prospects for control. *Virus Res.* **2009**, *141*, 113–130. [CrossRef]
58. Jones, R.A.C. Future Scenarios for Plant Virus Pathogens as Climate Change Progresses. *Adv. Virus Res.* **2016**, *95*, 87–147.
59. Van Harten, A. The relation between aphid flights and the spread of Potato virus YN(PVYN) in the Netherlands. *Potato Res.* **1983**, *26*, 1–15. [CrossRef]
60. Verbeek, M.; Piron, P.; Dullemans, A.; Cuperus, C.; van derVlugt, R. Determination of aphid transmission efficiencies for N, NTN and Wilga strains of Potato virus Y. *Ann. Appl. Biol.* **2010**, *156*, 39–49. [CrossRef]
61. Basky, Z. The relationship between aphid dynamics and two prominent potato viruses (PVY and PLRV) in seed potatoes in Hungary. *Crop Prot.* **2002**, *21*, 823–827. [CrossRef]
62. Basky, Z. Cumulative vector intensity and seed potato virus infection in Hungary. *Int. J. Hortic. Sci.* **2006**, *12*, 61–64. [CrossRef]
63. Northing, P. Extensive field based aphid monitoring asan information tool for the UK seed potato industry. *Asp. Appl. Biol.* **2009**, *94*, 31–34.
64. Kirchner, S.M.; Döring, T.F.; Hiltunen, L.H.; Virtanen, E.; Valkonen, J.P.T. Information-theory-based model selection for determining the main vector and period of transmission of Potato virus Y. *Ann. Appl. Biol.* **2011**, *159*, 414–427. [CrossRef]
65. Steinger, T.; Goy, G.; Gilliand, H.; Hebeisen, T.; Derron, J. Forecasting virus disease in seed potatoes using flight activity data of aphid vectors. *Ann. Appl. Biol.* **2015**, *166*, 410–419. [CrossRef]
66. Nemecek, T. The Role of Aphid Behaviour in the Epidemiology of Potato Virus Y: A Simulation Study. Ph.D. Thesis, ETH Zurich, Zurich, Switzerland, 1993.
67. Robert, Y.; Woodford, J.A.T.; Ducray-Bourdin, D.G. Some epidemiological approaches to the control of aphid-borne virus diseases in seed potato crops in northern Europe. *Virus Res.* **2000**, *71*, 33–47. [CrossRef] [PubMed]
68. Boquel, S.; Delayen, C.; Couty, A.; Giordanengo, P.; Ameline, A. Modulation of aphid vector activity by potato virus Y on in vitro potato plants. *Plant Dis.* **2012**, *96*, 82–86. [CrossRef]
69. Cambra, M.; Vidal, E. Sharka, a vector-borne disease caused by plum pox virus: Vector species, transmission mechanism, epidemiology and mitigation strategies to reduce its natural spread. *Acta Hortic.* **2017**, *1163*, 57–68. [CrossRef]
70. Dupuis, B. The movement of potato virus Y (PVY) in the vascular system of potato plants. *Eur. J. Plant Pathol.* **2017**, *147*, 365–373. [CrossRef]
71. Gadhave, K.R.; Gautam, S.; Rasmussen, D.A.; Srinivasan, R. Aphid Transmission of Potyvirus: The Largest Plant-Infecting RNA Virus Genus. *Viruses* **2020**, *12*, 773. [CrossRef]
72. Jeger, M.J. The Epidemiology of Plant Virus Disease: Towards a New Synthesis. *Plants* **2020**, *9*, 1768. [CrossRef] [PubMed]
73. Ruffel, S.; Gallois, J.L.; Lesage, M.L.; Caranta, C. The recessive potyvirus resistance gene pot-1 is the tomato orthologue of the pepper pvr2-eIF4E gene. *Mol. Genet. Genom.* **2005**, *274*, 346–353. [CrossRef]
74. Hashimoto, M.; Neriya, Y.; Yamaji, Y.; Namba, S. Recessive Resistance to Plant Viruses: Potential Resistance Genes Beyond Translation Initiation Factors. *Front. Microbiol.* **2016**, *7*, 1695. [CrossRef]
75. Michel, V.; Julio, E.; Candresse, T.; Cotucheau, J.; Decorps, C.; Volpatti, R.; Moury, B.; Glais, L.; Jacquot, E.; de Borne, F.D.; et al. A complex eIF4E locus impacts the durability of va resistance to Potato virus Y in tobacco. *Mol. Plant Pathol.* **2019**, *20*, 1051–1066. [CrossRef]
76. Doroszewska, T. Transfer of tolerance to different Potato virus Y (PVY) isolates from Nicotiana africana Merxm. to *Nicotiana tabacum* L. *Plant Breed.* **2009**, *129*, 76–81. [CrossRef]
77. Korbecka-Glinka, G.; Czubacka, A.; Depta, A.; Doroszewska, T. Inheritance of Potato virus Y tolerance introgressed from Nicotiana africana to cultivated tobacco. *Pol. J. Agron.* **2017**, *31*, 39–44.
78. Le, N.T.; Tran, H.T.; Bui, T.P.; Nguyen, G.T.; Van Nguyen, D.; Ta, D.T.; Trinh, D.D.; Molnar, A.; Pham, N.B.; Chu, H.H.; et al. Simultaneously induced mutations in eIF4E genes by CRISPR/Cas9 enhance PVY resistance in tobacco. *Sci. Rep.* **2022**, *12*, 14627. [CrossRef] [PubMed]
79. Zhou, L.X.; Tian, Y.P.; Ren, L.L. A natural substitution of a conserved amino acid in eIF4E confers resistance against multiple potyviruses. *Mol. Plant Pathol.* **2024**, *25*, e13418. [CrossRef]
80. Thieme, T.; Steiner, H.; Busch, T. Vergleich der Blattlausfänge in verschiedenen Gelbschalen. *Nachrichtenblatt Dtsch. Pflanzenschutzd.* **1994**, *46*, 65–68.
81. Chittka, L.; Döring, T.F. Are autumn foliage colors red signals to aphids? *PLoS Biol.* **2007**, *5*, e187. [CrossRef]

82. Döring, T.F.; Archetti, M.; Hardie, J. Autumn leaves seen through herbivore eyes. *Proc. R. Soc. Lond. B Biol.* **2009**, *276*, 121–127. [CrossRef]
83. Heie, O.E. *The Aphidoidea (Hemiptera) of Fennoscandia and Denmark. I. General Part. The Families Mindaridae, Hormaphididae, Thelaxidae, Anoeciidae, and Pemphigidae*; Scandinavian Science Press: Klampenborg, Denmark, 1980; 236p.
84. Heie, O.E. *The Aphidoidea (Hemiptera) of Fennoscandia and Denmark. II. Family Drepanosiphidae*; Scandinavian Science Press: Klampenborg, Denmark, 1982; 176p.
85. Heie, O.E. *The Aphidoidea (Hemiptera) of Fennoscandia and Denmark. III. Family Aphididae: Pterocommatinae and Tribe Aphidini of Subfamily Aphidinae*; Brill: Leiden, The Netherlands, 1986; 314p.
86. Heie, O.E. *The Aphidoidea (Hemiptera) of Fennoscandia and Denmark. IV. Family Aphididae: Part 1 of Tribe Macrosiphini of Subfamily Aphidinae*; Brill: Leiden, The Netherlands, 1992; 189p.
87. Heie, O.E. *The Aphidoidea (Hemiptera) of Fennoscandia and Denmark. V. Family Aphididae: Part 2 of Tribe Macrosiphini of Subfamily Aphidinae*; Brill: Leiden, The Netherlands, 1994; 242p.
88. Heie, O.E. *The Aphidoidea (Hemiptera) of Fennoscandia and Denmark. VI. Family Aphididae: Part 3 of Tribe Macrosiphini of Subfamily Aphidinae and Family Lachnidae*; Brill: Leiden, The Netherlands, 1995; 222p.
89. Taylor, L.R. *A Handbook for Aphid Identification*; Rothamsted Experimental Station: Harpenden, UK, 1984; 171p.
90. Clark, M.F.; Adams, A.N. Characteristic of the microplate of enzyme-linked immunosorbent assay for the detection of plant viruses. *J. Gen. Virol.* **1977**, *34*, 475–483. [CrossRef]
91. Hosmer, D.W.; Lemeshow, S. *Applied Logistic Regression*, 2nd ed.; John Wiley & Sons Inc.: New York, NY, USA, 2000.
92. Bewick, V.; Cheek, L.; Ball, J. Statistics review 14: Logistic regression. *Crit. Care* **2005**, *9*, 112–118. [CrossRef] [PubMed]
93. Peng, C.J.; So, T.H. Logistic regression analysis and reporting: A primer. *Underst. Stat.* **2002**, *1*, 31–70. [CrossRef]
94. Song, S.W. Using the receiver operating characteristic curve to measure sensitivity and specificity. *Korean J. Fam. Med.* **2009**, *30*, 841–842. [CrossRef]
95. Bewick, V.; Cheek, L.; Ball, J. Statistics review 13: Receiver operating characteristic curves. *Crit. Care* **2004**, *8*, 508–512. [CrossRef]
96. Peel, M.C.; Finlayson, B.L.; McMahon, T.A. Updated world map of the Köppen-Geiger climate classification. *Hydrol. Earth Syst. Sci.* **2007**, *11*, 1633–1644. [CrossRef]
97. Arnfield, A.J. Köppen Climate Classification. Encyclopedia Britannica. 28 May 2024. Available online: https://www.britannica.com/science/Koppen-climate-classification (accessed on 16 July 2024).
98. Chellappan, P.; Vanitharani, R.; Ogbe, F.; Fauquet, C.M. Effect of temperature on geminivirus-induced RNA silencing in plants. *Plant Physiol.* **2005**, *138*, 1828–1841. [CrossRef]
99. Szittya, G.; Silhavy, D.; Molnár, A.; Havelda, Z.; Lovas, Á.; Lakatos, L.; Bánfalvi, Z.; Burgyán, J. Low temperature inhibits RNA silencing-mediated defence by the control of siRNA generation. *EMBO J.* **2003**, *22*, 633–640. [CrossRef]
100. Choi, K.S.; Del Toro, F.; Tenllado, F.; Canto, T.; Nam Chung, B. A Model to Explain Temperature Dependent Systemic Infection of Potato Plants by Potato virus Y. *Plant Pathol. J.* **2017**, *33*, 206–211. [CrossRef]
101. Tsai, W.-A.; Brosnan, C.A.; Mitter, N.; Dietzgen, R.G. Perspectives on plant virus diseases in a climate change scenario of elevated temperatures. *Stress Biol.* **2022**, *2*, 37. [CrossRef] [PubMed]
102. Hunjan, M.S.; Lore, J.S. Climate Change: Impact on Plant Pathogens, Diseases and Their Management. In *Crop Protection under Changing Climate*; Jabran, K., Florentine, S., Chauhan, B.S., Eds.; Springer International Publishing: Cham, Switzerland, 2020; pp. 85–100.
103. Jeger, M.J.; Madden, L.V.; van den Bosch, F. Plant virus epidemiology: Applications and prospects for mathematical modeling and analysis to improve understanding and disease control. *Plant Dis.* **2018**, *102*, 837–854. [CrossRef] [PubMed]
104. Korbecka-Glinka, G.; Przybyś, M.; Feledyn-Szewczyk, B.A. Survey of Five Plant Viruses in Weeds and Tobacco in Poland. *Agronomy* **2021**, *11*, 1667. [CrossRef]
105. Chappell, T.M.; Beaudoin, A.L.P.; Kennedy, G.G. Interacting virus abundance and transmission intensity underlie tomato spotted wilt virus incidence: An example weather-based model for cultivated tobacco. *PLoS ONE* **2013**, *8*, e73321. [CrossRef] [PubMed]
106. Steinger, T.; Gilliand, H.; Hebeisen, T. Epidemiological analysis of risk factors for the spread of potato viruses in Switzerland. *Ann. Appl. Biol.* **2014**, *164*, 200–207. [CrossRef]
107. Beemster, A.B.R. Translocation of the potato viruses YN and YO in some potato varieties. *Potato Res.* **1976**, *19*, 169–172. [CrossRef]
108. Lindner, K.; Trautwein, F.; Kellermann, A.; Bauch, G. Potato virus Y (PVY) in seed potato certification. *J. Plant Dis. Prot.* **2015**, *122*, 109–119. [CrossRef]
109. Przybyś, M. Characterization and the Occurrence of PVY in the Main Tobacco Growing Areas in Poland. Ph.D. Thesis, Institute of Soil Science and Plant Cultivation—State Research Institute, Puławy, Poland, 2011.
110. Doroszewska, T.; Depta, A. Resistance of wild Nicotiana species to different PVY isolates. *Phytopathologia* **2011**, *59*, 9–24.
111. Rigotti, S.; Balmelli, C.; Gugerli, P. Census Report of the Potato Virus Y (PVY) Population in Swiss Seed Potato Production in 2003 and 2008. *Potato Res* **2011**, *54*, 105–117. [CrossRef]
112. Draper, M.D.; Pasche, J.S.; Gudmestad, N.C. Factors influencing PVY development and disease expression in three potato cultivars. *Am. J. Potato Res.* **2002**, *79*, 155–165. [CrossRef]
113. Valkonen, J.P.T.; Rokka, V.M. Combination and expression of two virus resistance mechanisms in interspecific somatic hybrids of potato. *Plant Sci.* **1998**, *131*, 85–94. [CrossRef]

114. Amari, K.; Huang, C.; Heinlein, M. Potential impact of global warming on virus propagation in infected plants and agricultural productivity. *Front. Plant Sci.* **2021**, *12*, 478. [CrossRef] [PubMed]
115. Radcliffe, E.B.; Ragsdale, D.W. Aphid-transmitted potato viruses: The importance of understanding vector biology. *Am. J. Potato Res.* **2002**, *79*, 353–386. [CrossRef]
116. Van Hoof, H.A. Determination of the infection pressure of potato virus YN. *Eur. J. Plant Pathol.* **1977**, *83*, 123–127.
117. Rydén, K.; Brishammar, S.; Sigvald, R. The infection pressure of potato virus YO and the occurrence of winged aphids in potato fields in Sweden. *Potato Res.* **1983**, *26*, 229–235. [CrossRef]
118. Sigvald, R. Relationship between aphid occurrence and spread of potato virus Yo (PVY) in field experiments in southern Sweden. *J. Appl. Entomol.* **1989**, *108*, 35–43. [CrossRef]
119. Kirchner, S.M.; Hiltunen, L.; Döring, T.F.; Virtanen, E.; Palohuhta, J.P.; Valkonen, J.P.T. Seasonal phenology and species composition of the aphid fauna in a northern crop production area. *PLoS ONE* **2013**, *8*, e71030. [CrossRef]
120. DiFonzo, C.D.; Ragsdale, D.W.; Radcliffe, E.B.; Gudmestad, N.C.; Secor, G.A. Seasonal abundance of aphid vectors of potato virus Y in the Red River Valley of Minnesota and North Dakota. *J. Econ. Entomol.* **1997**, *90*, 824–831. [CrossRef]
121. Heimbach, U.; Thieme, T.; Weidemann, H.L.; Thieme, R. Transmission of potato virus Y by aphid species which do not colonise potatoes. In *Aphids in Natural and Managed Ecosystems*; Dixon., A.F.G., Ed.; Universidad de León: León, Spain, 1998; pp. 555–559.
122. Woodford, J.A.T. Virus transmission by aphids in potato crops. *Neth. J. Plant Pathol.* **1992**, *98*, 47–54. [CrossRef]
123. MacKenzie, T.D.B.; Fageria, M.S.; Nie, X. Effects of crop management practices on current-season spread of potato virus Y. *Plant Dis.* **2014**, *98*, 213–222. [CrossRef] [PubMed]