Journal of Integrative Agriculture 2020, 19(2): 376–388

Available online at www.sciencedirect.com

ScienceDirect

RESEARCH ARTICLE

Simulation modelling of potato virus Y spread in relation to initial inoculum and vector activity

Andrew GALIMBERTI[1, 2], Andrei ALYOKHIN[1], Hongchun QU[1, 3], Jason ROSE[1]

[1] *School of Biology and Ecology, University of Maine, Orono, ME 04469, USA*
[2] *Eastern New York Commercial Horticulture Program, Cornell Cooperative Extension, Plattsburgh, NY 12901, USA*
[3] *College of Automation, Chongqing University of Posts and Telecommunications, Chongqing 400065, P.R.China*

Abstract

Potato virus Y (PVY) is a non-persistent virus that is transmitted by many aphid species and causes significant damage to potato production. We constructed a spatially-explicit model simulating PVY spread in a potato field and used it to investigate possible effects of transmission efficiency, initial inoculum levels, vector behavior, vector abundance, and timing of peak vector activity on PVY incidence at the end of a simulated growing season. Lower PVY incidence in planted seed resulted in lower virus infection at the end of the season. However, when populations of efficient PVY vectors were high, significant PVY spread occurred even when initial virus inoculum was low. Non-colonizing aphids were more important for PVY spread compared to colonizing aphids, particularly at high densities. An early-season peak in the numbers of non-colonizing aphids resulted in the highest number of infected plants in the end of the season, while mid- and late-season peaks caused relatively little virus spread. Our results highlight the importance of integrating different techniques to prevent the number of PVY-infected plants from exceeding economically acceptable levels instead of trying to control aphids within potato fields. Such management plans should be implemented very early in a growing season.

Keywords: potato virus Y, spatially-explicit modelling, aphids, virus spread, integrated pest management

1. Introduction

Potato virus Y (PVY) is the most economically important virus infecting potato (Gray *et al.* 2010; Scholthof *et al.* 2011). PVY is transmitted non-persistently by aphids (Hemiptera: Aphididae). Therefore, the virus is acquired in brief probes even in the absence of sustained feeding (Gray and Banerjee 1999). The virus has a complex epidemiology, with over 65 species of aphids serving as potential vectors (Pelletier *et al.* 2012). Because of this, control of the disease is difficult, with few reliable methods available (Davidson *et al.* 2013). Efficacy of control strategies can vary significantly from year to year or between different locations. Understanding the dynamics driving PVY spread could help clarify which factors should be the focus of control efforts.

PVY can be transmitted both by potato-colonizing aphids, as well as by aphids that do not colonize potato plants. However, the relative importance of potato colonizers and non-colonizers is uncertain. On one hand, colonizing aphid populations are able to build up within potato fields,

Received 21 September, 2018 Accepted 21 February, 2019
Correspondence Andrei ALYOKHIN, Tel: +1-207-5812977, Fax: +1-207-5812537, E-mail: alyokhin@maine.edu

© 2020 CAAS. Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).
doi: 10.1016/S2095-3119(19)62656-0

and these aphids often have high transmission efficiencies (Al-Mrabeh *et al.* 2010). On the other hand, non-colonizers will likely probe more plants within a potato field, leading to more opportunities to transmit the virus (Boquel *et al.* 2014). Individual species can also differ in characteristics such as transmission efficiency and propensity to move among plants, which could also affect PVY spread (Boquel *et al.* 2011, 2014). However, many characteristics related to vector behavior are nearly impossible to measure (Ferriss and Berger 1993), making it difficult to know which species are most responsible for spreading the virus. This is unfortunate because having a good idea of which vector species is epidemiologically important could inform decisions about which species to target for control.

Mineral oil is a tool used by many potato growers to reduce the spread of PVY. Mineral oil hinders the attachment of the virus particle to the aphid's mouthparts, thus reducing transmission efficiency by aphid vectors (Wang and Pirone 1996; Boquel *et al.* 2013). The ability of mineral oil to reduce PVY spread has been frequently demonstrated (Bradley *et al.* 1966; Boiteau and Singh 1982; Kirchner *et al.* 2014). While oil consistently lowers PVY incidence in the field, the magnitude of control can vary considerably from year to year, and oil is often insufficient for keeping PVY at economically acceptable levels on its own (Hansen and Nielsen 2012; MacKenzie *et al.* 2017). It is likely that the variation can be explained by outside factors, such as inoculum levels and vector abundance (MacKenzie *et al.* 2017). A clearer understanding of these processes could help optimize the use of mineral oil along with other management strategies.

A common strategy for PVY control is limiting the amount of inoculum in the field. This can be done through seed certification programs, in which tolerance limits for the amount of PVY allowed in seed potato lots are set by government agencies (Davidson *et al.* 2013). Rouging, or the physical removal of plants showing PVY symptoms, can be used to lower virus inoculum in fields during the growing season (Radcliffe and Ragsdale 2002). However, the spread of strains showing milder foliar symptoms has made these strategies more difficult, thus increasing virus inoculum in seed potato lots and causing more of them to be rejected (Lorenzen *et al.* 2006; Lacomme *et al.* 2014).

PVY outbreaks have complex dynamics that are driven by a variety of factors, often acting on a large-scale landscape level. These include, but not limited to, abundance, species composition, and activity of aphid vectors, the amount of initial inoculum within and around potato fields, location and spatial arrangement of potato fields within the landscape matrix, weather, genetic characteristics and physiological maturity of potato plants and surrounding vegetation, and movement of agricultural equipment within potato fields. Although many of the individual factors can be manipulated experimentally under controlled conditions, empirical testing of their effects and interactions under meaningfully realistic conditions is technically impossible. For example, influx of aphid vectors may depend on their abundance on wild hosts in unknown locations at considerable distances away from experimental plots, as well as on prevailing wind patterns. Similarly, the amount of initial inoculum may be affected by its presence in weedy hosts and in commercial potato fields and home gardens away from the immediate vicinity of the study area. Also, individual aphids are almost impossible to track under field conditions because of their small size. As a result, there are many potential nuisance variables that make deciphering the "big picture" of PVY epidemiology based solely on interpreting experimental field data very difficult and potentially misleading.

Simulation modeling is a tool with a variety of applications in the study of plant disease epidemiology. Modeling cannot be used as a substitute for collecting empirical data under field, semi-field, and laboratory conditions. However, it is beneficial for determining general mechanisms underlying disease spread among its hosts and for identifying individual factors that may be potentially responsible for disease outbreaks. Furthermore, simulation modeling allows the user to test variables which are extremely difficult to manipulate in laboratory or field experiments (Ferriss and Berger 1993). In particular, spatially-explicit agent-based simulation modeling can be used to incorporate biological and behavioral characteristics of different disease vectors. This approach involves constructing a virtual field with plants represented by cells in a matrix. The field contains agents, representing vectors, which move among the cells (Qu *et al.* 2017; Qu and Drummond 2018).

Bertschinger *et al.* (1995) developed a mathematical model that used temperature and aphid trap counts to simulate degeneration of potato seed lots due to viral infection. Although an important contribution to plant disease epidemiology, that model had several important limitations. It was rather general and did not distinguish between different viruses or transmission types. Also, the only two input variables possible in that study were aphid abundance based on trap captures and temperature. Furthermore, while the authors provided model description and results of sensitivity analysis, they did not use it to test any disease transmission parameters.

In the present study, we built a spatially-explicit model to simulate PVY spread among potato plants. Our major objectives were to gain insights into possible effects of transmission efficiency, initial inoculum levels, vector behavior, vector abundance, and timing of peak vector activity on disease levels at the end of a simulated growing season. Clarifying the relationships between the various factors influencing PVY spread may have important

implications for designing integrated pest management plans targeting virus reduction in commercial potato fields.

2. Materials and methods

2.1. Model description

The model constructed for the present study was a spatially-explicit model simulating PVY spread in a potato field. It was based on the existing empirical information on behavior of individual aphids and their interactions with potato plants, as described in Section 1. The field was set up as a square grid of cells that represent potato plants. Aphids occupied plants within the field; multiple aphids could be residing on the same plant. Aphids moved among plants as the model ran. Plants existed in two states: virus-infected or uninfected. Likewise, aphids could be either viruliferous or aviruliferous.

Aphids moved following the algorithm shown in Fig. 1. During each movement step, each aphid moved to a random plant within the field. The maximum distance of that move was determined by the parameter set for that species prior to running the model (see below). If the plant where the aphid landed was infected, the aphid became viruliferous. If the aphid was viruliferous and the plant was uninfected, the plant had a chance of becoming infected depending on

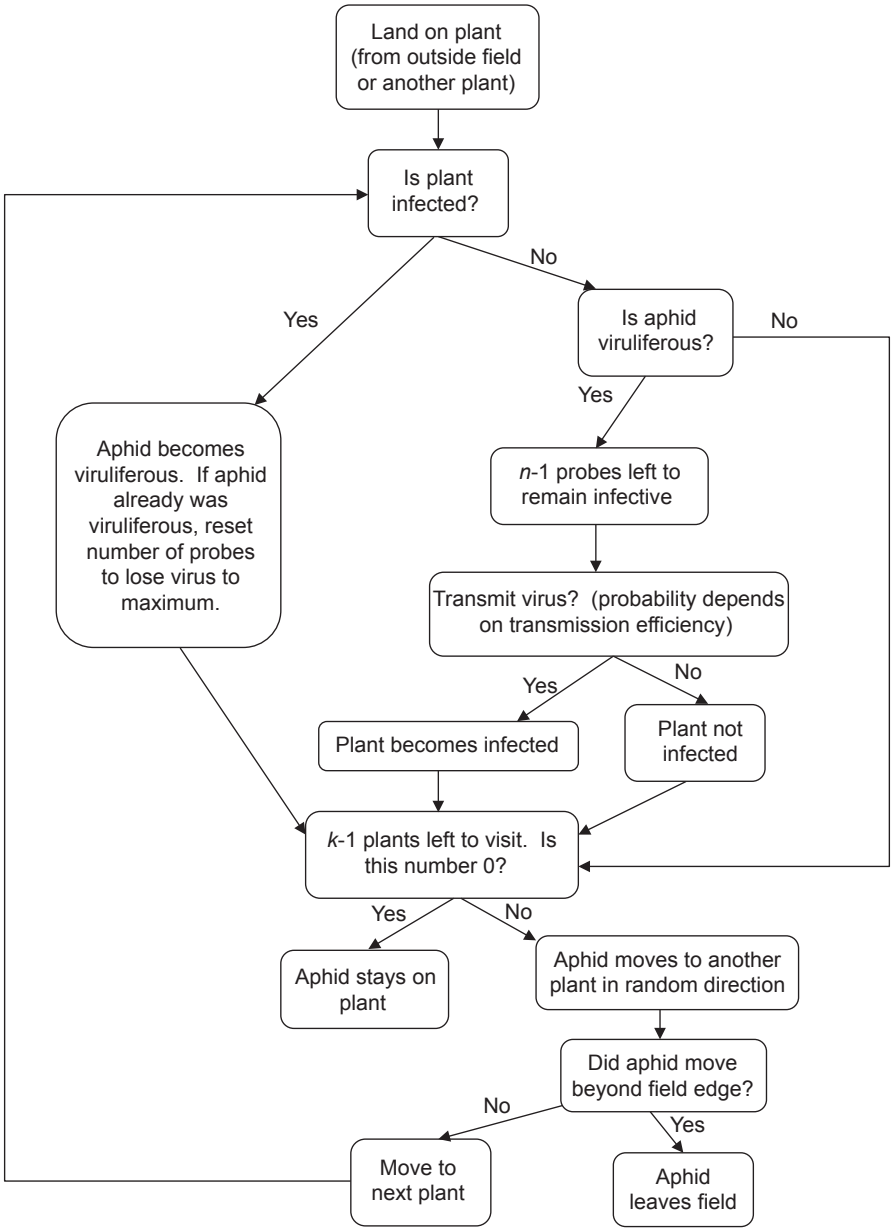

Fig. 1 Algorithm of aphid movement and virus transmission in the simulation model.

the transmission efficiency of the aphid. If the aphid was not viruliferous and the plant was not infected, the aphid continued moving throughout the field without transmitting PVY. Each aphid repeated this process until it settled on a plant after the specified number of plants visited for that species, until it reached the field edge and left the field, or until the simulation ended. Aphids leaving the field did not return back. Viruliferous aphids lost their ability to transmit viruses after landing on three uninfected plants (Bradley and Rideout 1953; Wróbel 2007). When a viruliferous aphid landed on an uninfected plant and then moved to an infected plant, it acquired the virus again and needed three additional probes to lose it.

Each replication of the model included a series of simulations, with each simulation representing one week in a field season. A simulation consisted of a set number of movement steps; each movement step was a single movement of every aphid in the field (Fig. 1). Once all movement steps in a simulation were complete, the next simulation began with the same field as in the end of the previous simulation, but with a different group of aphids. Each simulation could have a different group of aphids with different parameters. This process continued until the desired number of simulations was reached. Once all replications had finished, the model tallied and recorded the proportion of infected plants at each movement step.

The computer program to run the model was written in the computer programming language GAML, version 1.7 (Grignard *et al.* 2013). It is a high-level and intuitive agent-based language for the GAMA open-source modeling and simulation platform.

The following assumptions were made when designing and executing the model:

1) The field was square in shape and consisted of a grid of cells that represented potato plants;

2) Multiple aphids could be residing on the same plant

3) All inoculum originated within the field;

4) At the beginning of each replication, virus-infected plants were randomly distributed throughout the field, and aphids were randomly placed on plants;

5) Aphids did not show any preference for plants within the field; therefore, they moved onto each subsequent plant at random;

6) Aphids did not return to the field after leaving it;

7) Viruliferous aphids lost their ability to transmit viruses after landing on three uninfected plants;

8) Field season consisted of fourteen weeks (simulations);

9) Each simulation consisted of fifteen movement steps.

Parameters inputted into the model for the field and aphid community are listed in Table 1. Field parameters were field size and initial proportion of virus-infected plants. Field size was represented by a number of cells in a square grid. In this study, the field size was kept constant at 660×660 cells to approximate a 12-ha potato field. Aphid parameters were population size, transmission efficiency (i.e., the probability of a viruliferous aphid transmitting the virus when it landed on an uninfected plant), number of probes before a viruliferous aphid lost the virus, number of plants an aphid visited, and maximum number of spaces the aphid moved in a single movement step. Of those, transmission efficiency (Al-Mrabeh *et al.* 2010) and the number of probes before infectivity is lost (Bradley 1959; Wróbel 2007) were based on the results of laboratory experiments. Population sizes were extrapolated from the results of field trapping surveys (Difonzo *et al.* 1997; Mondal *et al.* 2016). Number of plants visited by each aphid was determined based on a normal distribution within one standard deviation of the set mean. For colonizing species, the parameter was selected to concur with the observations reported for the green peach aphid, *Myzus persicae* Sulzer (Harrewijn *et al.* 1981). For non-colonizing species, we assumed a larger number of plants visited in search of suitable hosts compared to colonizers. Distances traveled within the field were selected to fall within the ranges reported by Alyokhin and Sewell (2003) and Harrewijn *et al.* (1981). In the model, number of spaces moved during each movement step was randomly selected from a uniform distribution ranging from one to the set maximum. The model allowed for multiple aphid species with different parameters to be present at the same time.

Table 1 List of parameters included in the model

Parameter	Value	Distribution	Description
Field size	660×660 cells	Constant	Potato plants spaced at 30 cm from each other
Initial inoculum	0.001–0.2	Constant	Proportion of plants infected
Transmission efficiency	0.01–0.75	Constant	Probability of probe resulting in successful virus transmission
Plants visited before losing virus	3	Constant	Number of plants that a viruliferous aphid can infect
Number of plants visited	2 (colonizers); 10 (non-colonizers)	Normal	Plants visited per simulated week
Distance moved per movement step	1–50	Uniform	Number of plants moved per movement step

2.2. Transmission efficiency *vs*. initial inoculum

A series of runs was performed with different levels of initial inoculum and transmission efficiency to test their relative importance in the spread of PVY. Virus spread was tested at initial inoculum levels (defined as proportions of infected plants at the beginning of the first run before any transmission by aphids) of 0.001, 0.005, 0.01, 0.05, and 0.1. At each inoculum level, five aphid transmission efficiencies were tested: 0.01, 0.1, 0.25, 0.5, and 0.75. The aphid population was kept constant at 10 000 winged individuals belonging to the same species during each week. Aphids visited a mean of ten plants per week. Each transmission efficiency by initial inoculum level scenario was replicated 30 times.

2.3. Colonizing *vs*. non-colonizing aphids

Another series of runs was used to compare the relative importance of potato-colonizing aphids and non-colonizing aphids in spreading PVY. Three treatments were tested in this experiment. The first treatment represented colonizing aphids, specifically green peach aphids, which are the most efficient PVY vectors among potato colonizers. Transmission efficiency was set at 0.71 (Piron 1986). Green peach aphids were set to visit a mean of two plants per simulated week. Ten thousand aphids were used for each simulation. The second treatment consisted of non-colonizing aphids. In that treatment, twenty different non-colonizing potato aphid species were represented (Table 2). Transmission efficiencies for each species were taken from Al-Mrabeh *et al.* (2010). Species were selected to get a range of genera and transmission efficiencies. When multiple values were reported, the maximum transmission efficiency was used. All non-colonizing species visited a mean of ten plants per simulated week. Five hundred individuals of each species were used, totaling 10 000 winged aphids per week. The third treatment represented both colonizing and non-colonizing aphids present within the field together. Five thousand green peach aphids were included in the simulation using the same parameter settings as in the first treatment. In addition, 250 winged individuals of each non-colonizing aphid species used in the second treatment were included. The experiment was performed at three initial inoculum levels: 0.01, 0.05, and 0.2. Each treatment was replicated 30 times per initial inoculum level.

2.4. Abundance of non-colonizing aphids

Since the previous experiment identified non-colonizing aphids to be more important for PVY spread than colonizing aphids (see Section 3.2), next we tested the effects of their abundances on the proportion of infected plants. Initial inoculum was set at 0.05, which is the maximum proportion of infected tubers allowed for certification of seed potatoes in Maine. Population densities were set at 1 000, 10 000, 50 000, or 100 000 individuals per week, evenly distributed among the 20 non-colonizing species (Table 2). All other parameters were the same as set in the previous experiment for non-colonizing aphids.

Table 2 List of non-colonizing aphid species represented in the model, with corresponding transmission efficiencies expressed as probability of probes resulting in successful virus transmission

Species	Transmission efficiency[1)]
Acyrthosiphon pisum	0.14
Aphis fabae	0.24
Aphis glycines	0.75
Aphis gossypii	0.31
Aphis sambuci	0.12
Brachycaudus helichrysi	0.125
Capitophorus eleagni	0.2
Cavariella aegopodii	0.04
Cryptomyzus galeopsidis	0.174
Diuraphis noxia	0.07
Hyalopterus pruni	0.139
Hyperomyzus lactucae	0.174
Metopolophium dirhodum	0.03
Myzus cerasi	0.032
Phorodon humuli	0.35
Rhopalosiphum maidis	0.015
Rhopalosiphum padi	0.115
Sitobion avenae	0.018
Sitobion fragariae	0.101
Uroleucon spp.	0.083

[1)] Transmission efficiencies were compiled by Al-Mrabeh *et al*. (2010).

2.5. Timing of peak aphid abundance

The last experiment investigated whether timing of peak aphid abundance affected PVY spread within a simulated potato field. Four treatments were tested: (1) 10 000 aphids each week; (2) 40 000 aphids per week during the first two weeks of the season, followed by 5 000 aphids per week during the remaining 12 weeks; (3) 5 000 aphids per week during the first six weeks, then 40 000 aphids per week during the seventh and eighth weeks, and then 5 000 aphids per week during the remaining six weeks until the end of the season; and (4) 5 000 aphids per week during the first 12 weeks of the season, followed by 40 000 aphids per week during the thirteenth and fourteenth weeks. Thus, a total number of aphids summed up over the entire season was 140 000 in each treatment. All other parameters were the

same as set in the previous experiment.

2.6. Statistical analyses

For the first two experiments, two-way ANOVAs were performed to test the effect of each variable tested in that experiment on both final proportion of plants infected with PVY in the end of a simulated growing season, as well as on their proportional increase throughout the simulated growing season. Proportional increase was defined as the proportion of PVY-infected plants at the end of the season compared to the initial inoculum level. When a significant interaction was found, additional one-way ANOVAs were performed. For final proportion infected, one-way ANOVAs tested the effects of each variable (i.e., transmission efficiency or colonization behavior) on final proportion of plants infected at each initial inoculum level. For proportional increase, one-way ANOVAs tested the effects of initial inoculum on proportional increase in infection for each individual treatment. For the last two experiments, one-way ANOVAs were performed on final proportions of plants infected with PVY. Tukey's test was performed when one-way ANOVA results were significant. All analyses were performed in R (R Core Team 2016).

Increase in PVY inoculum over time was analyzed by fitting the data to linear, exponential, and sigmoid curves using TableCurve 2D (Systat Software 2002). Best-fit equations based on the Akaike Information Criteria, R^2 values, and significance of fit based on ANOVA were obtained from the analyses.

3. Results

3.1. Transmission efficiency *vs*. initial inoculum

Increasing transmission efficiency (F=1 933, df=4, 740, P<0.001) and initial inoculum (F=14 876, df=1, 740, P<0.001) each significantly increased the proportion of plants infected with PVY at the end of a simulated field season (Table 3). The interaction between the two factors was significant (F=1 379, df=4, 740, P<0.001). Increasing transmission efficiency significantly increased final infection rates at all initial inoculum levels.

Transmission efficiency (F=23 174, df=1, 740, P<0.001) and initial inoculum (F=449.6, df=4, 740, P<0.001) also significantly affected the proportional increase in virus inoculum over a simulated field season (Table 4). The interaction between transmission efficiency and initial inoculum was significant (F=653.5, df=4, 740, P<0.001). Increasing initial inoculum did not affect the proportional increase at a transmission efficiency of 0.01. However, greater initial inoculum levels showed lower proportional increase at all other transmission efficiencies.

Proportion of infected plants increased at an exponential rate (Fig. 2; P<0.05 for all fitted models). The rates of increase got progressively steeper with decreasing initial inoculum and increasing transmission efficiency. Exponential curves explained the majority of variation except at the lowest transmission efficiency with the lowest initial inoculum level.

3.2. Colonizing *vs*. non-colonizing aphids

Aphid colonizing behavior (F=3 622, df=2, 264, P<0.001) and initial inoculum (F=168 346, df=1, 264, P<0.001) significantly affected final proportion of infected plants at the end of a simulated growing season (Table 5). Their interaction was also significant (F=2 052, df=2, 264, P<0.001). Activity of non-colonizing aphids led to significantly greater final number of infected plants compared to the activity of colonizing aphids and to the activity of a mix of colonizing and non-colonizing aphids at all initial inoculum levels.

Aphid colonizing behavior (F=39 505, df=2, 261, P<0.001) and initial inoculum (F=4 497, df=2, 261, P<0.001) also significantly affected the proportional increase in inoculum throughout the season (Table 6). The interaction between colonizing behavior and initial inoculum was also significant

Table 3 Proportion of plants infected with potato virus Y (PVY) at the end of a simulated field season at each transmission efficiency and initial inoculum level

Transmission efficiency	Initial inoculum (proportion of plants infected)				
	0.001	0.005	0.01	0.05	0.1
0.01	0.0011±0.0001 a	0.0053±0.0001 a	0.011±0.0001 a	0.053±0.0004 a	0.11±0.0004 a
0.1	0.0019±0.0001 b	0.0093±0.0002 b	0.018±0.0003 b	0.089±0.0006 b	0.17±0.0007 b
0.25	0.004±0.0003 c	0.02±0.0005 c	0.039±0.0008 c	0.17±0.0015 c	0.3±0.0016 c
0.5	0.01±0.0005 d	0.048±0.001 d	0.092±0.0017 d	0.35±0.002 d	0.54±0.002 d
0.75	0.018±0.001 e	0.086±0.002 e	0.16±0.003 e	0.52±0.002 e	0.7±0.002 e
F-value	5 398	36 762	55 512	450 021	951 211
df	4, 145	4, 145	4, 145	4, 145	4, 145
P-value	<0.001	<0.001	<0.001	<0.001	<0.001

Data are presented as mean±SD, along with ANOVA results. Means followed by the same letters within columns are not significantly different from each other (P<0.05) based on Tukey's test.

Table 4 Proportional increase from initial virus inoculum to final infection rate at the end of a simulated field season at each transmission efficiency and initial inoculum level

Initial Inoculum (proportion of plants infected)	Transmission efficiency				
	0.01	0.1	0.25	0.5	0.75
0.001	1.07±0.05 a	1.87±0.09 a	4.02±0.28 a	9.89±0.47 a	18.4±1.1 a
0.005	1.07±0.02 a	1.86±0.05 a	3.94±0.1 ab	9.61±0.21 b	17.1±0.36 b
0.01	1.06±0.02 a	1.84±0.03 a	3.89±0.08 b	9.24±0.17 c	16±0.26 c
0.05	1.06±0.007 a	1.78±0.01 b	3.46±0.03 c	7.02±0.04 d	10.3±0.05 d
0.1	1.06±0.004 a	1.7±0.007 c	3.05±0.02 d	5.35±0.02 e	7.02±0.017 e
F-value	0.63	68.4	264.1	1 926	2 675
df	4, 145	4, 145	4, 145	4, 145	4, 145
P-value	0.643	<0.001	<0.001	<0.001	<0.001

Data are presented as mean±SD, along with ANOVA results. Means followed by the same letters within columns are not significantly different from each other (*P*<0.05) based on Tukey's test.

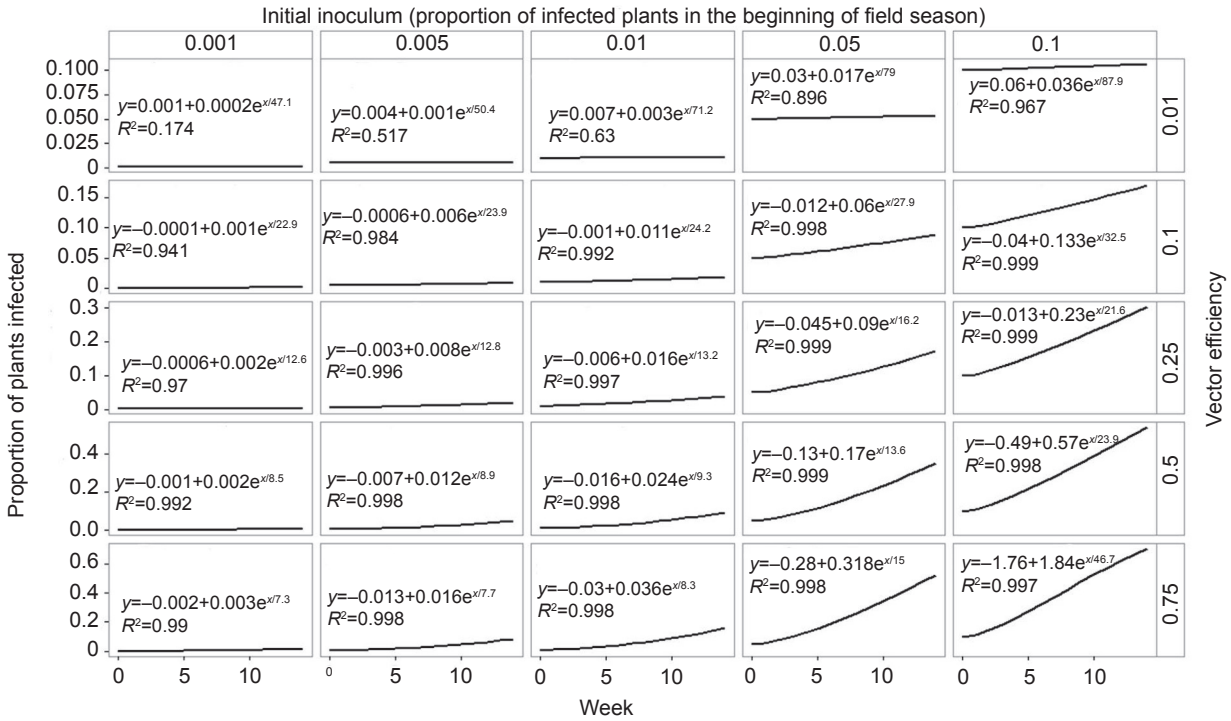

Fig. 2 Increase in proportion of infected plants throughout a simulated growing season at each transmission efficiency and initial inoculum level. Exponential equation and R^2 value for each curve are shown.

Table 5 Proportion of plants infected with potato virus Y (PVY) at the end of a simulated field season at each colonization type and initial inoculum level

Colonization behavior	Initial inoculum (proportion of plants infected)		
	0.01	0.05	0.2
Colonizers	0.013±0.0002 a	0.063±0.0005 a	0.24±0.0007 a
Non-colonizers	0.024±0.0004 b	0.11±0.001 b	0.37±0.001 b
Both	0.018±0.0004 c	0.086±0.0006 c	0.3±0.001 c
F-value	7 892	34 173	111 294
df	2, 87	2, 87	2, 87
P-value	<0.001	<0.001	<0.001

Data are presented as mean±SD, along with ANOVA results. Means followed by the same letters within columns are not significantly different from each other (*P*<0.05) based on Tukey's test.

(*F*=1 045, *df*=4, 261, *P*<0.001). Increasing initial inoculum significantly lowered proportional increase for colonizing aphids, non-colonizing aphids, and a combination of colonizers and non-colonizers.

Table 6 Proportional increase from initial virus inoculum to final infection rate at the end of a simulated field season at each colonization behavior and initial inoculum level

Initial Inoculum (proportion of plants infected)	Colonization behavior		
	Colonizers	Non-colonizers	Both
0.01	1.28±0.02 a	2.39±0.04 a	1.79±0.04 a
0.05	1.25±0.01 b	2.25±0.02 b	1.72±0.01 b
0.2	1.21±0.004 c	1.84±0.01 c	1.52±0.005 c
F-value	248.8	3 134	1 198
df	2, 87	2, 87	2, 87
P-value	<0.001	<0.001	<0.001

Data are presented as mean±SD, along with ANOVA results. Means followed by the same letters within columns are not significantly different from each other (P<0.05) based on Tukey's test.

In each treatment, the proportion of infected plants increased exponentially over time (Fig. 3; P<0.05 for all fitted models). At each initial inoculum level, non-colonizing aphids caused PVY to be spread at a faster rate compared to colonizing aphids. The equations explained most of the variation in all cases.

3.3. Abundance of non-colonizing aphids

Higher aphid populations resulted in higher proportions of infected plants in the end of the growing season. There was very little transmission at the population density of 1 000 aphids per week, while almost all plants became infected at the population density of 100 000 aphids per week (Table 7). PVY spread exponentially for all but the highest aphid density. At 100 000 aphids per week, a sigmoid curve provided a better fit to the generated data (Fig. 4).

3.4. Timing of peak aphid abundance

An early peak in aphid abundance resulted in most PVY spread, with almost three times as many infected plants in the end of the season as under constant aphid densities throughout the season. Mid- and late-season peaks had the lowest PVY spread, which was significantly smaller than that under constant densities (Table 8). Mid-season peak resulted in disease progression that best fitted a sigmoid curve. Under the other conditions, proportion of infected plants grew exponentially (Fig. 5).

4. Discussion

Our simulations confirmed that planting seed with lower PVY incidence would result in lower virus infection at the end of the season. However, greater transmission efficiency of aphid vectors also significantly increased final virus infection levels regardless of initial inoculum. With enough efficient vectors, significant PVY spread could occur even when initial virus inoculum was low. Such a result emphasizes the importance of using mineral oil, which reduces the transmission efficiency of aphids probing oil-treated plants (Wang and Pirone 1996; Boquel *et al.* 2013). This agrees with recent field studies, which demonstrated that PVY can be effectively controlled in the field using mineral oil (MacKenzie *et al.* 2014, 2016, 2017), especially when used in combination with insecticides. However, in those studies virus reduction was less consistent than suggested in our model. In the field, incomplete coverage may be an issue (Boiteau *et al.* 2009), and virus spread may be higher or lower depending on aphid population size or vector species present. Still, oil does effectively reduce aphid transmission efficiency and contribute to PVY control.

Results from the curve-fitting analyses supported the importance of reducing transmission efficiency and maintaining low virus levels to reduce PVY spread. While all curves showed exponential increase, rates of increase grew dramatically with increasing transmission efficiency. In addition, when both initial inoculum and transmission efficiency were kept low, the models were a poor fit and curves remained relatively flat throughout the simulated growing season. It is likely that virus levels under those conditions were, in large part, stochastically driven. Similarly, MacKenzie *et al.* (2014) identified interaction between initial inoculum and vector activity as an important parameter affecting PVY spread on commercial potato fields.

The proportional increase in the incidence of infected plants between the beginning and the end of the field season supported similar conclusions. While the final viral incidence was higher when initial inoculum was large, at higher transmission efficiencies the ratio of final to initial number of infected plants decreased with increasing virus levels at the start of the season (Table 4). Thus, highly efficient vectors spread the virus rapidly even when initial inoculum levels were low. At the lowest transmission efficiency, initial inoculum did not affect proportional increase, and virus spread was small regardless of amount of PVY present.

Non-colonizing aphids played a greater role in the spread of PVY compared to colonizing aphids. This is consistent with the empirical evidence suggesting that potato colonizers may be less important than non-colonizers

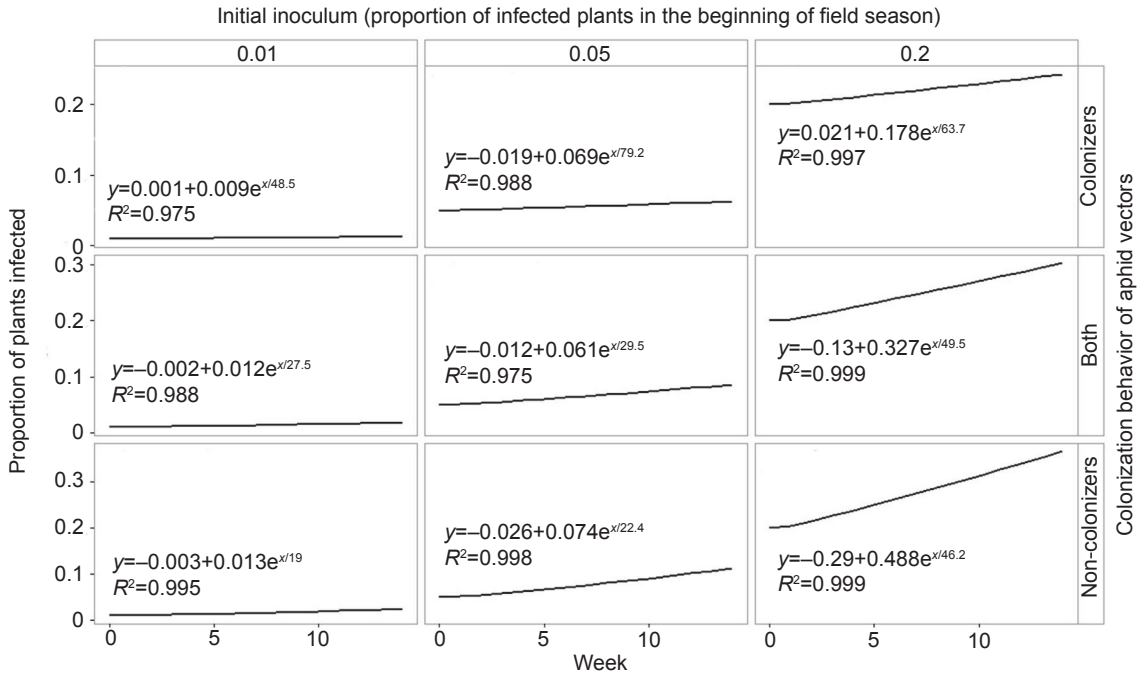

Fig. 3 Increase in proportion of infected plants for each colonization type and initial inoculum level. Exponential equation and R^2 value for each curve are shown.

Table 7 Proportion of plants infected with potato virus Y (PVY) at the end of a simulated field season at each density of non-colonizing aphid populations

Total number of aphids	Infection rate (proportion of plants infected)
1 000	0.0551±0.0004 a
10 000	0.1126±0.0008 b
50 000	0.5731±0.0028 c
100 000	0.9386±0.0013 d
F-value	1 963 643
df	3, 116
P-value	<0.001

Data are presented as mean±SD, along with ANOVA results. Means followed by the same letters within columns are not significantly different from each other (P<0.05) based on Tukey's test.

in PVY epidemiology. Significant PVY infection has been observed in the field despite a lack of colonizing aphids (Kirchner *et al.* 2011). Even when green peach aphids were present in high numbers, PVY spread was not correlated with green peach aphid abundance (Steinger *et al.* 2015). To the contrary, there was a significant correlation between PVY infection rate and captures of non-colonizing aphids (Mondal *et al.* 2016).

Colonizing aphids settle on a potato plant when searching the field. Thus, even if they acquire the virus, they may not leave the infected plant to spread it to other plants. This contrasts with non-colonizing aphids; each individual is more likely to land on another plant and potentially infect it. While colonizing green peach aphids are much more efficient vectors than non-colonizing aphids, they have little opportunity to spread the virus compared to non-colonizers. This could help explain why insecticides are often ineffective at suppressing PVY in the field by themselves (MacKenzie *et al.* 2017). These sprays primarily target colonizing aphids. Non-colonizers coming from outside the field, which are responsible for most of the PVY spread, are unaffected.

As discussed earlier, transmission efficiency is still important, at least with regards to non-colonizing species. Non-colonizing aphids with very low efficiencies may have little impact on PVY spread even when initial inoculum is relatively high (see graph in the upper right corner on Fig. 2); these inefficient vectors may therefore be less critical to target for control. Behavioral differences could also exist among individual species within groups, making some of them more important than the others. The model assumed that all non-colonizers had the same propensity for movement; in reality, this is uncertain. Plant factors could also affect the probability of transmission (Boquel *et al.* 2010). Regardless, non-colonizing aphids still appear to have a greater impact on PVY spread in most cases.

Not surprisingly, higher populations of non-colonizing aphids caused significantly higher virus spread in our simulations. At the highest density of 500 000 aphids passing through the field each week, the rate of increase

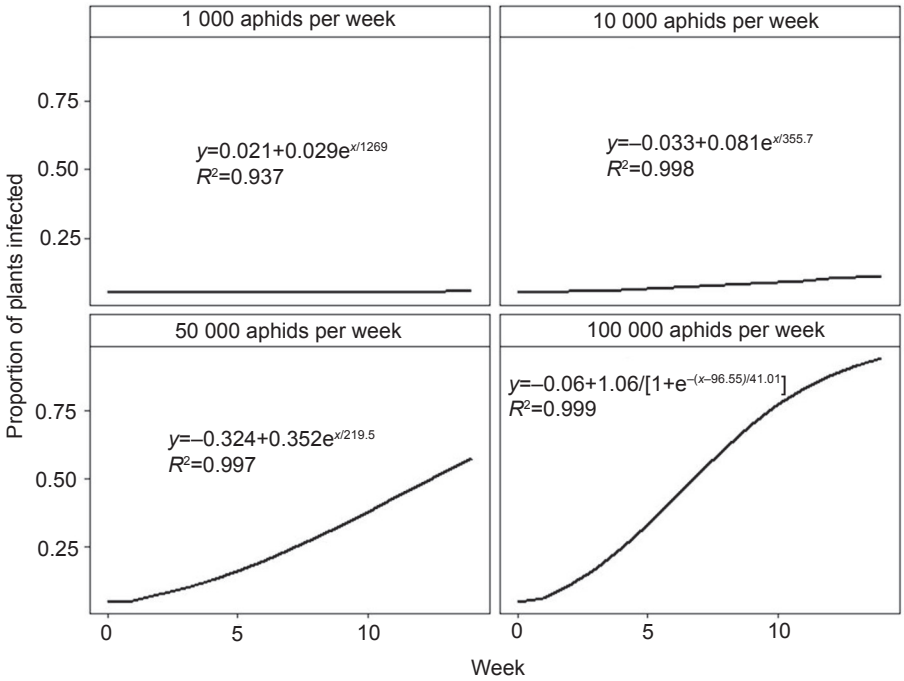

Fig. 4 Increase in proportion of infected plants at each density of non-colonizing aphid populations. Exponential or sigmoid equation and R^2 value for each curve are shown.

Table 8 Proportion of plants infected with potato virus Y (PVY) at the end of a simulated field season at each timing of peak density of non-colonizing aphid populations

Timing of aphid peak	Infection rate (proportion of plants infected)
Early	0.3034±0.0019 a
Middle	0.0888±0.0007 b
Late	0.0889±0.0005 b
No peak	0.1126±0.0009 c
F-value	255 312
df	3, 116
P-value	<0.001

Data are presented as mean±SD, along with ANOVA results. Means followed by the same letters within columns are not significantly different from each other (P<0.05) based on Tukey's test.

in the number of PVY-infected plants started to slow down mid-season (Fig. 4). That was different from the exponential growth observed in most other cases. Most likely, the field became saturated with infected plants. As a result, many aphids moved from one infected plant to another, with little effect on further spread of the disease. Still, almost all plants ended up having the virus in the end of the growing season (Fig. 4; Table 7).

Since non-colonizing aphids cannot be controlled in potato fields, their possible influx represents a challenge for potato production. One option for addressing this challenge is to use an area-wide approach and control aphids in other crops (Mondal *et al.* 2016). However, this may not be always logistically possible and/or economically feasible. Furthermore, a considerable number of non-colonizing vectors may originate from non-cultivated vegetation and travel over large distances before arriving to potato fields (Mondal *et al.* 2016).

An early-season peak in the numbers of non-colonizing aphids resulted in the highest number of infected plants towards the time of the harvest, while mid- and late-season peaks were relatively harmless. Apparently, early build up in virus inoculum was sufficient to ensure steady PVY spread for the remainder of the season, despite a substantial drop in the abundance of aphid vectors. A previous field study (MacKenzie *et al.* 2016) also demonstrated a significant increase in PVY infection when aphid populations were high early in the season, but not when they were high late in the season.

In our model, mid-season aphid peak caused a short-lived temporary boost in infection spread during the two high-density weeks, resulting in a sigmoid shape of epidemic progression (Fig. 5). However, the final infection rates were low (Table 8). This is different from the finding by MacKenzie *et al.* (2016), who reported higher PVY spread when mid-season aphid populations were high. The extent of PVY transmission by aphids at different times of the season may vary depending on potato cultivars and aphid species present (Karasev and Gray 2013). Therefore, the

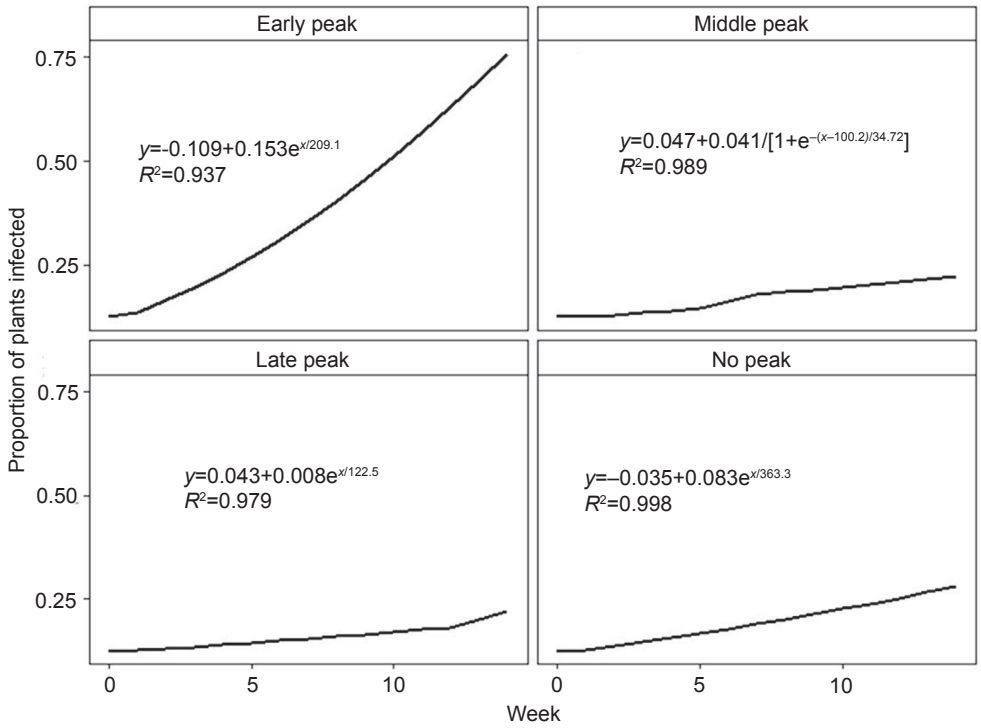

Fig. 5 Increase in proportion of infected plants at each timing of peak density of non-colonizing aphid populations. Exponential or sigmoid equation and R^2 value for each curve are shown.

window of potato crop vulnerability is not likely to be always limited to the early season. Still, our results emphasize that PVY management should be implemented from the very beginning of a growing season.

Based on the results of our modelling simulation, as well as the aforementioned empirical evidence, aphid control is not the best approach for protecting potato fields from PVY. That is not to say that colonizing aphids should be completely ignored; they are important vectors of persistent viruses and did cause some PVY spread in the model. However, a more complicated management approach that integrates several different techniques is needed to ensure that the number of PVY-infected plants does not exceed economically acceptable levels. Reducing initial inoculum and using mineral oil to interfere with its transmission is likely to provide more effective PVY control than simply targeting colonizing aphid populations. Insecticides may further increase efficiency of oils, possibly through incapacitating aphid vectors (MacKenzie *et al.* 2017). Border crops around potato fields that intercept arriving aphids and make them lose infectivity after several probes is another valuable technique in PVY management, especially in combination with the sprays of mineral oils (Boiteau *et al.* 2009).

Our model relies on parameters for which exact measurements are nearly impossible to obtain. For example, abundance of flying aphids is usually investigated by using different traps. While such data may be useful to detect aphid presence and compare relative abundance between species or times of the year, its relation to absolute abundance of aphids flying in a given area is unclear. Therefore, it can at best provide guidance on aphid populations, as was the case in the present study. Similarly, tracking individual aphids and recording the number of plants that they visit in the field is technically impossible. Furthermore, a plethora of external factors, such as weather or abundance of natural enemies, are likely to affect aphid numbers. Indeed, there is a well-documented variation in aphid populations between years (Difonzo *et al.* 1997) and between geographic locations (Mondal *et al.* 2016). Vector behavior may also be affected (Narayandas and Alyokhin 2006). Therefore, parameter estimates and model assumptions may be realistic only under certain, often unknown, conditions.

On one hand, this difficulty makes our approach highly beneficial because it allows testing hypotheses which could not be easily studied in laboratory or field experiments. However, this also makes validating the model extremely difficult. Thus, its results are more useful as estimates showing general patterns of PVY spread rather than as exact predictors of how many plants are infected with PVY on specific potato fields. Nevertheless, this model generates data that are in general agreement with a number of field

studies (Kirchner *et al.* 2011; MacKenzie *et al.* 2014, 2016, 2017; Steinger *et al.* 2015; Mondal *et al.* 2016). Therefore, it can be an effective theoretical tool which can improve understanding of PVY epidemiology.

5. Conclusion

PVY spread in potato fields can be curtailed only through integrating different techniques that reduce initial virus inoculum and interfere with virus transmission by its aphid vectors. Particular attention should be paid to protecting plants early in the growing season. Chemical control of aphid vectors residing within potato fields is unlikely to be effective on its own.

Acknowledgements

This research was supported in part by the United States Department of Agriculture National Institute of Food and Agriculture Special Crops Research Initiative (Award # 2014-51181-22373). Funding for Hongchun Qu's stay at the University of Maine was received from the National Natural Science Foundation of China (Award # 61871061).

References

Al-Mrabeh A, Anderson E, Torrance L, Evans A, Fenton B. 2010. A literature review of insecticide and mineral oil use in preventing the spread of non-persistent viruses in potato crops. In: *Report to the Potato Council of the Agriculture and Horticulture Development Board.* Stoneleigh Park, Warwickshire, United Kinngdom.

Alyokhin A, Sewell G. 2003. On-soil movement and plant colonization by walking wingless morphs of three aphid species (Homoptera: Aphididae) in greenhouse arenas. *Environmental Entomology*, **32**, 1393–1398.

Bertschinger L, Keller E R, Gessler C. 1995. Development of EPIVIT, a simulation model for contact- and aphid-transmitted potato viruses. *Phytopathology*, **85**, 801–814.

Boquel S, Giguere M A, Clark C, Nanayakkara U, Zhang J H, Pelletier Y. 2013. Effect of mineral oil on *Potato virus Y* acquisition by *Rhopalosiphum padi*. *Entomologia Experimentalis et Applicata*, **148**, 48–55.

Boquel S, Giordanengo P, Ameline A. 2010. Divergent effects of PVY-infected potato plant on aphids. *European Journal of Plant Pathology*, **129**, 507–510.

Boquel S, Giordanengo P, Ameline A. 2011. Probing behavior of apterous and alate morphs of two potato-colonizing aphids. *Journal of Insect Science*, **11**, 1–10.

Boquel S, Giordanengo P, Ameline A. 2014. Vector activity of three aphid species (Hemiptera: Aphididae) modulated by host plant selection behaviour on potato (Solanales: Solanaceae). *Annales de la Societe Entomologique de France*, **50**, 141–148.

Boiteau G, Singh R. 1982. Evaluation of mineral oil sprays for reduction of virus Y spread in potatoes. *American Potato Journal*, **59**, 253–262.

Boiteau G, Singh M, Lavoie J. 2009. Crop border and mineral oil sprays used in combination as physical control methods of the aphid-transmitted potato virus Y in potato. *Pest Management Science*, **65**, 255–259.

Bradley R H E. 1959. Loss of virus from the stylets of aphids. *Virology*, **8**, 308–318.

Bradley R H E, Moore C A, Pond D D. 1966. Spread of potato virus Y cutailed by oil. *Nature*, **209**, 1370–1371.

Bradley R H E, Rideout D W. 1953. Comparative transmission of potato virus Y by four aphid species that infest potatoes. *Canadian Journal of Zoology*, **31**, 333–341.

Davidson R D, Houser A J, Sather K, Haslar R. 2013. Controlling PVY in seed: What works and what does not. *American Journal of Potato Research*, **90**, 28–32.

Difonzo C D, Ragsdale D W, Radcliffe E B, Gudmestad N C, Secor G A. 1997. Seasonal abundance of aphid vectors of potato virus Y in the Red River Valley of Minnesota and North Dakota. *Journal of Economic Entomology*, **90**, 824–831.

Ferriss R S, Berger P H. 1993. A stochastic simulation model of epidemics of arthropod-vectored plant viruses. *Phytopathology*, **83**, 1269–1278.

Gray S, De Boer S, Lorenzen J, Karasev A, Whitworth J, Nolte P, Singh R, Boucher A, Xu H. 2010. PVY: An evolving concern for potato crops in the United States and Canada. *Plant Disease*, **94**, 1384–1397.

Gray S M, Banerjee N. 1999. Mechanisms of arthropod transmission of plant and animal viruses. *Microbiology and Molecular Biology Reviews*, **63**, 128–48.

Grignard A, Taillandier P, Gaudou B, Vo D A, Huynh N Q, Drogoul A. 2013. GAMA 1.6: Advancing the art of complex agent-based modeling and simulation. In: Boella G, Elkind E, Savarimuthu B T R, Dignum F, Purvis M K, eds., *PRIMA 2013*: *Principles and Practice of Multi-Agent Systems*. Springer-Verlag, Berlin. pp. 117–131.

Hansen L M, Nielsen S L. 2012. Efficacy of mineral oil combined with insecticides for the control of aphid virus vectors to reduce potato virus Y infections in seed potatoes (*Solanum tuberosum*). *Acta Agriculturae Scandinavica* (Section B: Soil & Plant Science), **62**, 132–137.

Harrewijn P, Hoof H A, Noordink J P W. 1981. Flight behaviour of the aphid *Myzus persicae* during its maiden flight. *Netherlands Journal of Plant Pathology*, **87**, 111–117.

Karasev A V, Gray S M. 2013. Continuous and emerging challenges of *Potato virus Y* in potato. *Annual Review of Phytopathology*, **51**, 571–586.

Kirchner S M, Döring T F, Hiltunen L H, Virtanen E, Valkonen J P T. 2011. Information-theory-based model selection for determining the main vector and period of transmission of *Potato virus Y*. *Annals of Applied Biology*, **159**, 414–427.

Kirchner S M, Hiltunen L H, Santala J, Döring T F, Ketola J, Kankaala A, Virtanen E, Valkonen J P T. 2014. Comparison of straw mulch, insecticides, mineral oil, and birch extract

for control of transmission of *Potato virus Y* in seed potato crops. *Potato Research*, **57**, 59–75.

Lacomme C, Davie K, Holmes R, Pickup J. 2014. PVY^{N} prevalence in potato crops: Impact of strain competition and differential ability to overcome plant resistance mechanisms. In: *Proceedings of The Dundee Conference on Crop Protection in Northern Britain 2014*, *Dundee*, *UK*, *25-26 February 2014*. United Kingdom. pp. 203–208.

Lorenzen J H, Meacham T, Berger P H, Shiel P J, Crosslin J M, Hamm P B, Kopp H. 2006. Whole genome characterization of *Potato virus Y* isolates collected in the western USA and their comparison to isolates from Europe and Canada. *Archives of Virology*, **151**, 1055–1074.

MacKenzie T D B, Fageria M S, Nie X, Singh M. 2014. Effects of crop management practices on current-season spread of *Potato virus Y*. *Plant Disease*, **98**, 213–222.

MacKenzie T D B, Lavoie J, Nie X, Singh M. 2017. Effectiveness of combined use of mineral oil and insecticide spray in reducing *Potato virus Y* (PVY) spread under field conditions in New Brunswick, Canada. *American Journal of Potato Research*, **94**, 70–80.

MacKenzie T D B, Nie X, Singh M. 2016. Crop management practices and reduction of on-farm spread of *Potato virus Y*: A 5-year study in commercial potato fields in New Brunswick, Canada. *American Journal of Potato Research*, **93**, 552–563.

Mondal S, Wenninger E J, Hutchinson P J S, Weibe M A, Eigenbrode S D, Bosque-Pérez N A. 2016. Contribution of noncolonizing aphids to *Potato virus Y* prevalence in potato in Idaho. *Environmental Entomology*, **45**, 1445–1462.

Narayandas G K, Alyokhin A V. 2006. Interplant movement of potato aphid (Homoptera: Aphididae) in response to environmental stimuli. *Environmental Entomology*, **35**, 733–739.

Pelletier Y, Nie X, Giguère M A, Nanayakkara U, Maw E, Foottit R. 2012. A new approach for the identification of aphid vectors (Hemiptera: Aphididae) of potato virus Y. *Journal of Economic Entomology*, **105**, 1909–1914.

Piron P G M. 1986. New aphid vectors of potato virus Y^{N}. *Netherlands Journal of Plant Pathology*, **92**, 223–229.

Qu H C, Drummond F A. 2018. Simulation-based modeling of wild blueberry pollination. *Computers and Electronics in Agriculture*, **144**, 94–101.

Qu H C, Seifan T, Seifan M. 2017. Effects of plant and pollinator traits on the maintenance of a food deceptive species within a plant community. *Oikos*, **126**, 1815–1826.

R Core Team. 2016. *R: A Language Environment for Statistical Computing*. The R Foundation for Statistical Computing, Vienna, Austria.

Radcliffe E B, Ragsdale D W. 2002. Aphid-transmitted potato viruses: The importance of understanding vector biology. *American Journal of Potato Research*, **79**, 353–386.

Scholthof K B G, Adkins S, Czosnek H, Palukaitis P, Jacquot E, Hohn T, Hohn B, Saunders K, Candresse T, Ahlquist P, Hemenway C, Foster G D. 2011. Top 10 plant viruses in molecular plant pathology. *Molecular Plant Pathology*, **12**, 938–954.

Steinger T, Goy G, Gilliand H, Hebeisen T, Derron J. 2015. Forecasting virus disease in seed potatoes using flight activity data of aphid vectors. *Annals of Applied Biology*, **166**, 410–419.

Systat Software. 2002. *TableCurve 2D*. Systat Software, San Jose, California.

Wang R Y, Pirone T P. 1996. Mineral oil interferes with retention of tobacco etch potyvirus in the stylets of *Myzus persicae*. *Phytopathology*, **86**, 820–823.

Wróbel S. 2007. Effect of a mineral oil on *Myzus persicae* capability to spread of PVY and PVM to successive potato plants. *Journal of Plant Protection Research*, **47**, 383–390.

Executive Editor-in-Chief WAN Fang-hao
Managing editor SUN Lu-juan