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Soybean vein necrosis virus: an emerging virus in North America

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Abstract
Few diseases have emerged in such a short period of time as soybean vein necrosis. The disease is present in all major producing areas in North America, affecting one of the major row field instead of row crops for the United States. Because of the significance of soybean in the agricultural economy and the widespread presence of the disease, the causal agent, soybean vein necrosis virus has been studied by several research groups. Research in the past 10 years has focused on virus epidemiology, management, and effects on yield and seed quality. This communication provides a review of the current knowledge on the virus and the disease.

Keywords Orthotospovirus · Soybean · Neohydatothrips variabilis

Introduction

Soybean vein necrosis virus (SVNV) is an orthotospovirus naturally infecting soybean. The virus is transmitted primarily by the soybean thrips (*Neohydatothrips variabilis*, Beach) in a persistent and propagative manner and causes localized infections on soybean leaves. As a distinct member of the genus *Orthotospovirus*, family *Tospoviridae*, SVNV shares minimal similarity with all established species in the genus and represents a new clade in the genus evolution [1, 2].

SVNV was first reported in Arkansas and Tennessee, US, in 2008 associated with symptoms that initiated with vein clearing followed by lesions and necrosis [3]. In subsequent years, symptoms were observed in the two aforementioned states but also several other soybean-growing areas including Illinois, Wisconsin, Michigan, Ohio, Pennsylvania, Delaware, Kansas, Oklahoma, Kentucky, Missouri, Mississippi, Louisiana, and Alabama [1, 4–11]. Nowadays, the virus has been confirmed in at least 22 states across the U.S. as well as Canada and Egypt [12] and vein necrosis has become the most prevalent virus disease in North America [2].

The rapid spread of SVNV raised concerns about its economic impact; providing impetus to gain a better understanding of the fundamental aspects of the virus and the disease, and to develop appropriate control strategies. This review highlights our knowledge on the biology and epidemiology of the virus as well as diagnostics and control strategies for the disease.

Genome organization

The genome of SVNV has a typical orthotospovirus organization which consists of three single-stranded RNA segments that are designated as L-, M-, and S- RNAs. The pleomorphic virions of orthotospoviruses range in size from 80 to 120 nm. The polymerase and nucleoprotein of orthotospoviruses are enclosed within a host-derived lipid membrane with the two viral glycoproteins—Gn and Gc projecting from the surface [13, 14]. In-silico analysis of the SVNV genome revealed classic features of members in the *Tospoviridae*, such as all three RNA segments have the highly conserved 5′ terminal sequence ($AGAGCA_{1-6}$) predicted to be critical in replication and transcription signal [15]. Many other, atypical, attributes of the genome will be discussed here in more detail.

SVNV L RNA is 9010 nucleotides (nt) in length and contains a single open-reading frame (ORF) in the negative orientation. The 19 nt of 5′- and 3′- ends are complementary to each other putatively leading to the circularization

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of the molecule forming a panhandle structure, similar to other orthotospoviruses [15]. The region between nt 8980-185 codes for a polyprotein of 336 kDa with five motifs [A ($DxxKWS_{539–544}$), B ($QGxxxxxSS_{527–535}$), C ($SDD_{665–667}$), D (K_{712}), and E ($EFxSE_{721–725}$)] present in RNA-dependent RNA polymerases (RdRp). Those motifs alongside motif F ($Kx_{451–452}$, $KxQR_{459–462}$, and $TxxDRxIY_{463–470}$), present only in some orthotospoviruses, are part of the "U-shaped" crevice formed by typical RdRp domains [16, 17]. The RNA and the polyprotein it encodes have distinct properties when compared with other members of the genus including size—the longest one alongside bean necrotic mosaic virus (BNeMV), the closest-related virus to SVNV. Additionally, a Lysine-rich extension ($TSSSGSK_{2900–2906}$ and $KWSKPKKKKKPKAKPKKSKKKHNK_{2908–2931}$) with unknown function is identified at the carboxy-terminal of the protein [1, 18].

The M RNA has 4955 nt with the first and last 27 showing almost perfect complementarity and potentially forming a panhandle structure. This RNA codes two ORFs which are separated by a 267-nt A/U-rich intergenic region (IGR). $ORF1_{58–1008}$, codes for a 35-kDa non-structural protein (NSm). The presence of highly conserved $LxDx_{40}G$ motif of the 30K movement protein superfamily suggests that the protein is involved in cell-to-cell movement [19–21]; however, the Leu residue has been substituted by an Ile at the beginning of the motif. The ''P/D-L-X motif'' and phospholipase A2 catalytic sites, present in some orthotospovirus orthologs such as tomato spotted virus (TSWV) and groundnut bud necrosis virus (GBNV) are absent from the SVNV counterpart [21, 22]. $ORF2_{4863–1276}$ codes for the precursor of the virion glycoproteins (Gn/Gc). Signal cleavage between Cys_{378} and Ser_{379} yields two proteins: Gn (43 kDa) and Gc (91 kDa). The Gn protein contains several signature motifs present in orthotospovirus orthologs including a $RGD_{29–31}$ domain, which is crucial in virion–cell receptor attachment [20, 21] as well as several *N*-Glycosylation sites ($N_{25,229,343}$) and transmembrane domains ($aa_{6–28,317–339,349–371}$). The SVNV Gc protein has a series of highly conversed sequences present in orthotospovirus orthologs including Lys_{702}, a $T\text{-}X\text{-}T_{714–716}$, $CTGxC_{730–734}$, and $TSxWGCEExxCXAxxxGxxxGxC_{754–776}$ [23], whereas *N*-Glycosylation sites transmembrane domains are present at $N_{5,20,171}$ and $aa_{77–99}$, respectively. SVNV has the largest glycoproteins among all the members in the genus with a long amino acid tail on the C-terminus.

S RNA is 2603 nt long and contains two ORFs in opposite orientations. The first and last six nucleotides of this segment are complementary, similar to the other two RNAs. The untranslated region is highly structured with 5′- and 3′- UTRs being 58 and 70 nt long, respectively. The $ORF1_{59–1381}$ encodes NSs, a 50-kDa non-structural protein predicted to be an RNAi suppressor [24]. Conserved $GK_{178–179}$ and $DExx_{148–151}$ comprise the Walker A and B motifs which interact with ATP/ADP phosphates and coordinate/bind Mg^{2+} ions during ATP hydrolysis [25, 26]. The remaining $ORF_{2533–1700}$ codes for a putative nucleoprotein (N) of 31 kDa. The protein has an RNA-binding Lysine-rich motif $KKDGKGKKSK_{264–273}$, as well as several discrete RNA-interacting amino acids ($PSN_{7–9}$, $RK_{51–52}$, $RY_{54–55}$, and $KK_{73–74}$), domains that probably allow nucleoprotein to participate in RNA synthesis together with the RNA L polyprotein as shown for members of the *Bunyavirales* [27–31]. The two ORFs are separated by a 318-nt A/U-rich IGR, one of the smallest among members in the genus [32].

Phylogenetic analyses based on all coding regions of the genome indicate that SVNV and BNeMV belong to a distinct clade that shares almost equidistance between American and Eurasian lineages [1, 18, 32, 33; Fig. 1]. Serological relationship between SVNV and other orthotospoviruses species representing existing serogroups or distinct serotypes within the genus verified SVNV having a distinct serotype [34], corroborating with its unique phylogenetic placement. The genomic divergence of SVNV-BNeMV clade from the other orthotospovirus groups and the unique features discussed here suggest that SVNV is the type member of a novel evolutionary lineage of *Fabaceae*-infecting orthotospoviruses.

Symptomology and host range

The early symptoms of SVNV infection include clearing along the main veins, sometimes with small light-green to yellow patches distributed between veins. Affected areas expand and become chlorotic and eventually necrotic as leaves mature [1, 3]. The distinction between infected and non-infected areas may be blurring on fully developed leaflets in the field and sometimes could be mistaken for other disorders (Fig. 2); such distinction becomes more clear as disease progresses (Fig. 2). Unlike diseases such as frogeye leaf spot or bacterial blight which also cause foliar lesions, SVNV infection-caused lesions expand through leaf veins to the surrounding areas and are rarely surrounded with halos. They are irregularly shaped and tend to unevenly distribute on leaf blades which probably mirror the vector-preferred feeding areas (Fig. 2). Later in the season, lesions coalesce leading to scotched appearance or leaf death (Fig. 2). Symptom intensities seem to vary in both greenhouse and field conditions (Zhou, personal observation). Mild symptoms exhibit as thread-shaped lesions along the main vein or other irregular shapes of yellow patches which take up minimal areas of the leaf blade (Fig. 2); whereas more aggressive symptoms display as yellow, or reddish-brown to dark brown lesions covering the major portion of the blade (Fig. 2). Such symptom variations may be correlated to different host genotypes as suggested by Anderson [33] which could represent

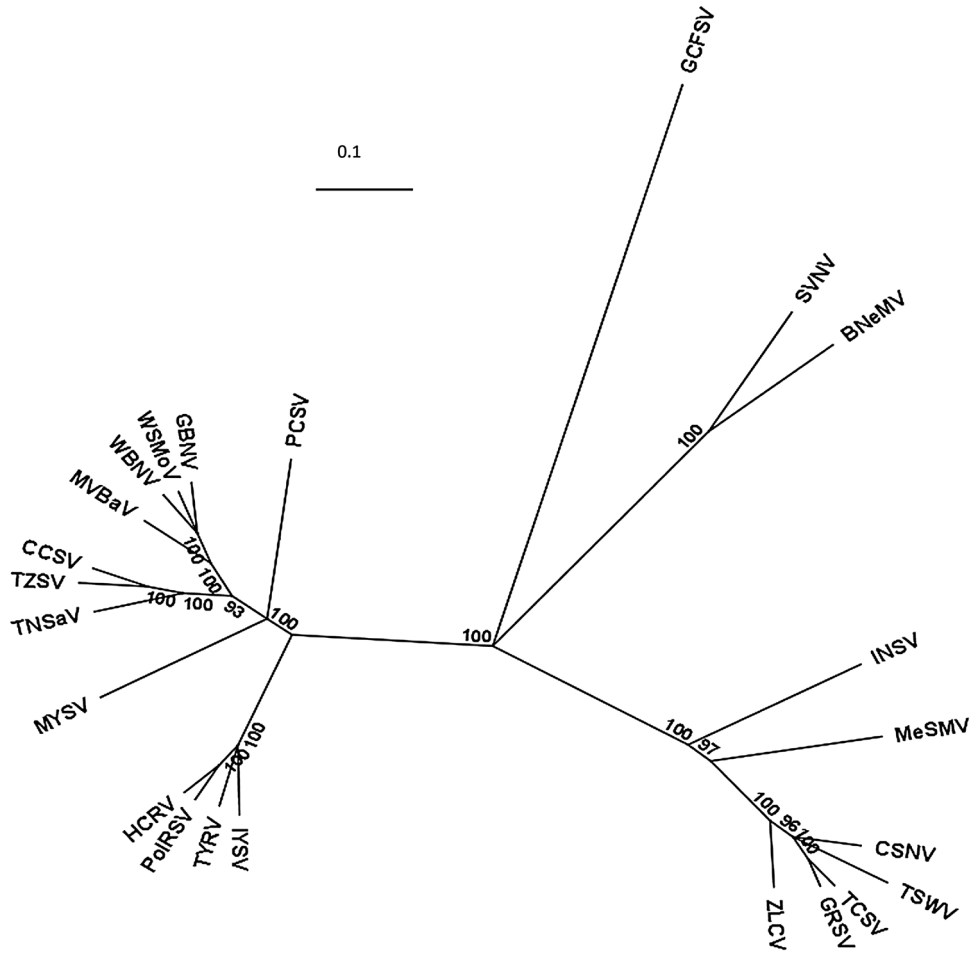

Fig. 1 Phylogenetic analysis based on alignment of all orthotospovirus RdRp amino acid sequences available in the National Center for Biotechnology Information (NCBI) Genbank as of September 2018. The dendrogram was produced in CLC Genomics Workbench 11.0.1 using the Neighbor-Joining algorithm with 1000 bootstrap replicates. Only bootstrap values > 90% are shown. The bar represents p-distance of 0.1. Virus acronyms used include GBNV (groundnut bud necrosis virus; NP 619688), GRSV (groundnut ringspot virus; AST36116), INSV (impatiens necrotic spot virus; NP 619710), IYSV (iris yellow spot virus; YP 009241381), PolRSV (polygonum ringspot virus; AOO95317), TCSV (tomato chlorotic spot virus; YP 009408637), TSWV (tomato spotted wilt virus; NP 049362), WBNV (watermelon bud necrosis virus; YP 009505544), WSMoV (watermelon sliver mottle virus; AAW56420), ZLCV (zucchini lethal chlorosis virus; YP 009316178), BNeMV (bean necrotic mosaic virus; AEF56575), CCSV (calla lily chlorotic spot virus; YP 009449454), CSNV (chrysanthemum stem necrosis virus; AII20576), HCRV (hippeastrum chlorotic ringspot virus; CDJ79757), MeSMV (melon severe mosaic virus; YP 009346017); MYSV (melon yellow spot virus; YP 717933), PCSV (pepper chlorotic spot virus; YP 009345145); SVNV (soybean vein necrosis virus; ADX01591), TNSaV (tomato necrotic spot associated virus; AMY62790); TYRV (tomato yellow ring virus; AEX09314); TZSV (tomato zonate spot virus; YP 001740047), GCFSV (groundnut chlorotic fan-spot virus; AJT59689), MVBaV (mulberry vein banding associated virus; YP 009126736)

as tolerant versus susceptible cultivars to either the virus or thrips feeding, or it could result from the fact that virus infection occurs at different growth stages of soybeans which causes different levels of damage to the plant. The timing of first appearance of disease symptoms in different soybean-growing regions varies from June to October [36–39]; on the other hand, the timing also varies between years depending on weather patterns. In general terms, in hotter and drier conditions, disease emerges early in the season, possibly due to higher vector populations (Zhou, Tzanetakis, personal observations). Disease symptoms are usually first observed on the lower canopy moving upwards as newly emerged leaves are the preferential feeding sites for thrips [37, 39].

Apart from soybean, SVNV has been reported to naturally infect another leguminous crop—yard-long bean (*Vigna unguiculata* spp. *sesquipedalis*) [6]. Infected yard-long beans exhibit vein yellowing and chlorotic spots surrounded by necrosis on leaflets. The virus is only detected on symptomatic but not on asymptomatic samples; how the virus moves on the plant (locally or systemically), however, has not been determined yet (Valverde, personal communication). Several studies have been performed to investigate

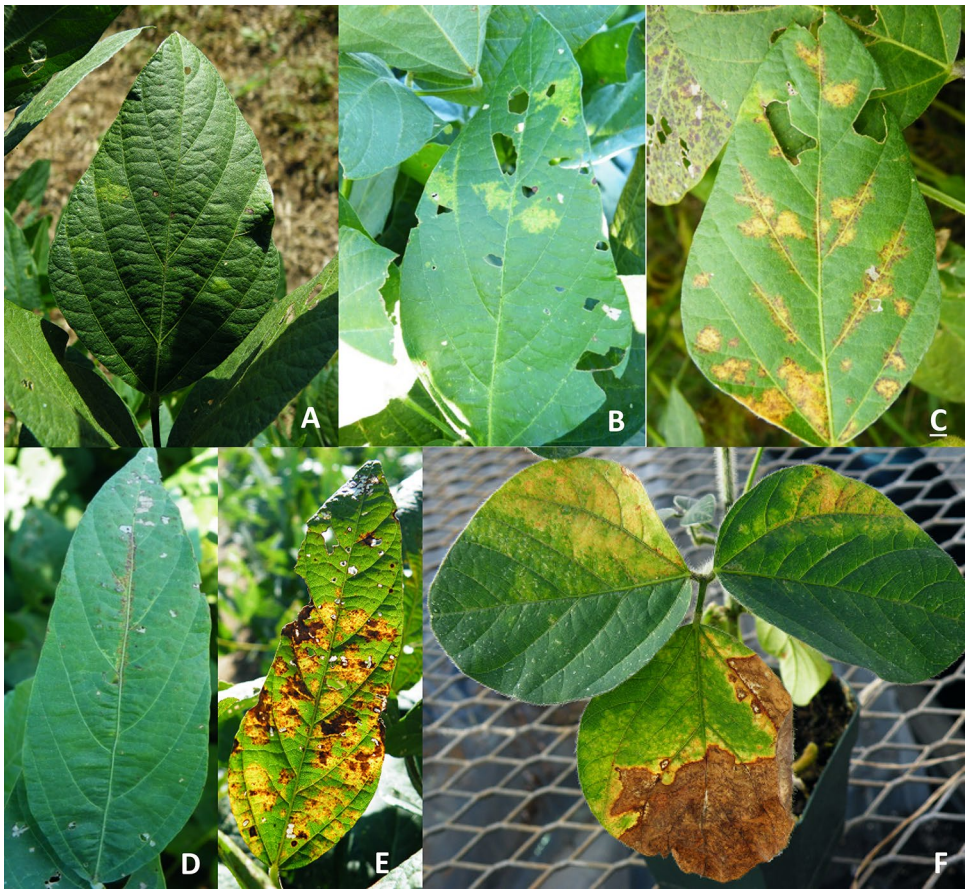

Fig. 2 Disease symptoms caused by SVNV infection on soybean. **a** Indistinguishable early symptom; **b** Distinct early symptom; **c** Uneven distribution of lesions on leaf blade; **d** Mild disease symptom; **e** Aggressive disease symptom; **f** Scotched leaf blade due to virus infection

the role of indigenous weeds in disease epidemiology. So far, natural SVNV infection has been reported on ivyleaf morning glory (*Ipomoea hederacea* Jacq.), entireleaf morning glory (*Ipomoea hederacea* var. *integriuscula*), and kudzu (*Pueraria montana*) [2, 39, 40]. The high incidence of morning glory species in soybean fields and the fact that ivyleaf morning glory is a natural host of SVNV stimulated researchers to further explore the role of weeds in virus dissemination. Both ivyleaf morning glory and pitted morning glory (*Ipomoea lacunose* L.) sustain virus replication in green house experiment whereas a 3-year field survey in Alabama detected low infection rate of SVNV infection on entireleaf morning glory but not on pitted morning glory [35, 40]. A recent study revealed kudzu—a weed species in the Fabaceae as an asymptomatic, systemic host of SVNV [41]. Given that kudzu is a perennial weed which presents in millions of acres in Southeastern U.S. and has the overlapping geographic range with many major soybean-producing states, it is possible that this plant species may serve as the major reservoir for SVNV; providing overwintering or early season population growth habitats for viruliferous thrips prior to moving to soybean.

All characterized SVNV isolates cause localized infections on soybean where the virus is restricted in and around the clearing or lesion areas [2, 36, 42]. Symptoms observed in soybean fields have been reproduced in green house studies using either mechanical or vector inoculation [2, 35, 43].

Several plant species were tested as alternative hosts in greenhouse studies, most of which produce local lesions indicating hypersensitive reactions to the virus. SVNV infection causes similar symptoms on legume species including cowpea (*Vigna unguiculate*), mungbean (*Vigna radiata*), medicago (*Medicago truncatula*), and pigeon pea (*Cajanus cajan*) (Fig. 3). Typical symptoms on these species include chlorotic lesions which become either necrotic or coalesce resulting in senescence or even death of inoculated leaves. Disease symptoms on non-leguminous hosts, however, vary. On *Nicotiana benthamiana*, SVNV produces necrosis on inoculated leaflets which expands to newly emerged leaves and the systemic movement of the virus leads to stem collapse and plant death [2]. On buckwheat (*Fagopyrum esculentum*), another systemic host, virus infection displays chlorosis to necrosis, whereas on melon (*Cucumis melo*), only small sunken gray lesions were observed [43]. In addition, few species including chrysanthemum (*Dendranthema grandiflorum*) and pumpkin (*Cucurbita pepo* L.) were proved as asymptomatic hosts [2]. According to Anderson [35], Palmer amaranth (*Amaranthus palmeri* S. Wats.) and

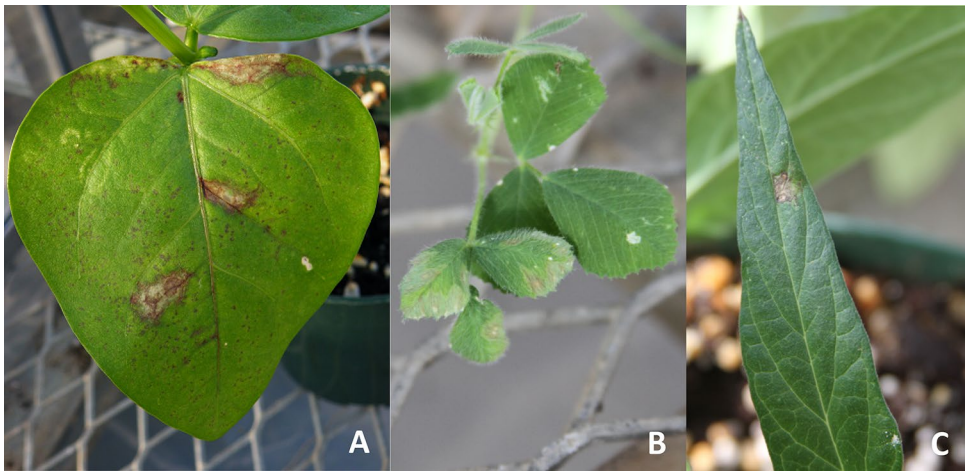

Fig. 3 Local lesions caused by SVNV on **a** Cowpea; **b** Medicago and **c** Pigeon pea

Redroot pigweed (*Amaranthus retroflexus* L.) were tested positive in thrips inoculation experiment; however, plants were not observed for a prolonged time period to determine whether they are symptomatic or asymptomatic hosts.

Disease diagnosis

Typical disease symptoms caused by the infection of orthotospoviruses include chlorotic lesions and necrosis, independent of localized or systemic hosts [14]. In the case of SVNV, the virus remains localized on soybean and exhibits symptoms as discussed above, which could aid the diagnosis of the disease. Confirmation of virus infection on verified hosts and diagnosis on new hosts, however, requires accurate and sensitive detection methods. There are currently two types of assays routinely used for SVNV detection: immunological- and PCR-based. For immunodiagnostics, polyclonal antibodies were generated against the recombinant *E. coli*-expressed nucleocapsid protein of the virus [42] enabling to detect the virus using dot blot or enzyme-linked immunosorbent assay (ELISA). A dilution of 1:2000 of this antiserum could detect SVNV in sap extract from naturally infected soybean leaf tissues diluted up to 1:512, according to Khatabi and co-workers. Apart from that, several ELISA kits are available in the market. Our previous studies showed DAS-ELISA using 1:200 dilution of polyclonal antibody generated against SVNV N protein from one commercial vendor is capable of detecting the virus from leaf tissues grinded in buffer at the ratio of 1/20 (w/v) but not in any further sap dilutions [11]. Likewise, other studies also showed inconsistent results when ELISA was used for SVNV detection: the virus was detected using RT-PCR in plant tissues that were tested as negative with ELISA [35, 44]. These results suggest the efficacy of immunological-based diagnosis varies; probably due to the quality of the antibodies used.

In terms of nucleic acid-based detection techniques, several PCR detection assays have been developed and successfully applied in virus diagnosis [1, 4–11]. Most assays were designed using the N gene as the target for amplification given that (1) the population structure analysis based on N protein of SVNV revealed a minimal diversity across a wide geographic area [2], which makes it ideal in designing assays that can detect even diverse virus isolates; (2) N gene is highly expressed allowing for sensitive detection. In Zhou and Tzanetakis [2], the assay could detect the virus in 4 pg of RNA extracted from naturally infected soybean tissues in 30 PCR cycles or 40 pg of the same RNA sample in 20 cycles. It is generally accepted that real-time PCR (qPCR) has higher sensitivity than conventional PCR in virus detection. However, the development of qPCR assays with lower detection limit than conventional PCR is challenging for SVNV. Only one qPCR assay has been published for SVNV by Keough et al. [45] used to determine copy numbers of the virion within individual *N. variabilis*. The efficiency of this assay, however, was not mentioned.

Transmission

The unique transmission properties of SVNV concur with the phylogenetic studies distinguishing the virus from other well-characterized members of the genus. There are more than 5000 thrips species described to date and only 17, belonging to the genera *Frankliniella, Thrips, Ceratothripoides, Scirtothrips, Dictyothrips, Neohydatothrips*, and *Taeniothrips* have been confirmed as vectors of orthotospoviruses (2, 46, 47; Table 1). The primary vector of SVNV, *Neohydatothrips variabilis* (Beach) (Fig. 4) is the only vector species belonging to the subfamily Sericothripinae (Thysanoptera: Thripidae); all the other genera belong to subfamily Thripinae (Thysanoptera: Thripidae). *N. variabilis* is a common pest for soybean and cotton in the U.S [44]

Table 1 Summary of thrips-orthotospovirus interactions confirmed by transmission studies

Thrips genera	Thrips species	Orthotospovirus vectored
Frankliniella	*Frankliniella occidentalis*	Chrysanthemum stem necrosis virus *Groundnut ringspot virus* *Impatiens necrotic spot virus* *Tomato chlorotic spot virus* *Tomato spotted wilt virus* Alstroemeria necrotic streak virus [65]
	Frankliniella schultzei	Chrysanthemum stem necrosis virus *Groundnut ringspot virus* *Groundnut bud necrosis virus* *Tomato chlorotic spot virus* *Tomato spotted wilt virus*
	Frankliniella intonsa	*Groundnut ringspot virus* *Impatiens necrotic spot virus* *Tomato chlorotic spot virus* *Tomato spotted wilt virus*
	Frankliniella fusca	*Tomato spotted wilt virus* *Impatiens necrotic spot virus* Soybean vein necrosis virus [45]
	Frankliniella gemina	*Tomato spotted wilt virus* *Groundnut ringspot virus*
	Frankliniella bispinosa	*Tomato spotted wilt virus*
	Frankliniella zucchini	*Zucchini lethal chlorosis virus*
	Frankliniella cephalica	*Tomato spotted wilt virus*
	Frankliniella tritici	*Soybean vein necrosis virus* [45]
Thrips	*Thrips palmi*	Calla lily chlorotic spot virus *Groundnut bud necrosis virus* Melon yellow spot virus *Watermelon silver mottle virus* Tomato necrotic ringspot virus [66]
	Thrips tabaci	*Iris yellow spot virus* *Tomato spotted wilt virus* Tomato yellow fruit ring virus
	Thrips setosus	*Tomato spotted wilt virus*
Scirtothrips	*Scirtothrips dorsalis*	*Groundnut bud necrosis virus* Groundnut chlorotic fan-spot virus *Groundnut yellow spot virus*
Ceratothripoides	*Ceratothripoides claratris*	Capsicum chlorosis virus Tomato necrotic ringspot virus [66]
Dictyothrips	*Dictyothrips betae*	*Polygonum ringspot virus*
Neohydatothrips	*Neohydatothrips variabilis*	Soybean vein necrosis virus [2, 45]
Taeniothrips	*Taeniothrips. eucharii*	Hippeastrum chlorotic ringspot virus [47]

Italics: assigned and; plain text: unassigned members of the genus *Orthotospovirus*

Numbers in the parenthesis indicate additional references to Reference [46]

and the phylogenetic placement of *N. variabilis* mirrors the phylogenetic space of SVNV as an orthotospovirus and may reflect the co-evolution of orthotospoviruses with their vectors [48]. Recent studies have reported two other common thrips species *Frankliniella tritici* (Fitch) (eastern flower thrips) and *Frankliniella fusca* (Hinds) (tobacco thrips) as vectors of SVNV. Their transmission efficiencies, however, are much lower to that of *N. variabilis* suggesting that the latter has coevolved with the virus and acts as its primary vector in the field [36]. Another important orthotospovirus vector—*Frankliniella occidentalis* (western flower thrips) is unable to transmit SVNV [39]. According to Keough et al. [45], SVNV-infected *N. variabilis* prefer to feed on non-infected leaflets and viruliferous females produce more offspring compared with their non-viruliferous counterparts. These attributes may have contributed the rapid spread of SVNV in a short time span. Similar to other orthotospoviruses, SVNV is considered to be transmitted in a propagative and persistent manner, and the acquisition of the virus by its vectors is a life stage-dependent process [13].

Seed transmission has always been a major concern for virus diseases, especially for seed-propagated crops like

Fig. 4 Different life stages of *Neohydatothrips variabilis* (Beach). **a** First instar larvae; **b**. Early second instar larvae; **c**. Late second instar larvae; **d**. Prepupa; **e**. Pupa, **f**. Adult

soybean, as it can act as the major route for long-distance dissemination of viruses [36, 49–51]. Investigations on whether SVNV is a seed-transmissible virus have been conducted by different researchers in recent years with contradictory results. Groves and co-workers [52] reported a 6% seed-transmission rate. In this study, a random seed sample obtained from a seed lot of a commercial soybean variety was planted under controlled conditions. Leaf samples collected from their seedlings were tested positive for SVNV using RT-PCR, but not ELISA. Additional testing using arbitrarily selected plants from initial testing and repeated experiments confirmed the presence of SVNV genome segments using RT-PCR and RNA-seq analysis. The authors therefore concluded that this is due to an asymptomatic, seed-transmissible SVNV isolate that is transmitted by soybean seeds at high rate. A study performed by Hajimorad et al. [36] on two soybean cultivars using over 2000 seeds derived from 20 SVNV-infected individual mother plants failed to detect the presence of SVNV using ELISA. They analyzed the genetic variation among SVNV isolate from infected mother plants and found the existence of a distinct isolate which has a unique amino acid mutation and branches separately from all other isolates indicating a relatively diverse virus population in the study. Considering the non-systemic movement of SVNV on soybean, the self-pollinating feature of soybean and the fact that SVNV infection occurs in the late-growth stage, Hajimorad et al. concluded that it is very unlikely SVNV is transmitted by seed. The Hajimorad et al. results are in agreement with our studies in which SVNV was not detected in over 600 seedlings germinated from seed collected from SVNV-infected mother plants of different cultivars growing in the field (Zhou and Tzanetakis, unpublished data).

Disease management

Management of orthotospovirus-caused diseases has always presented a major challenge [14, 53, 54]. There is limited knowledge on many aspects of the biology and epidemiology of SVNV which are crucial for developing effective strategies for virus control and disease management. Since the primary and secondary vector species were well documented and more data became available for potential alternative hosts of the virus [2, 6, 40, 43, 45], the current management options for soybean vein necrosis have focused on reducing

the impact of thrips on soybeans and seeking potential virus reservoirs in the field. Several studies have been conducted to determine the composition of thrips species and population dynamics of SVNV vectors, especially for *N. variabilis* over seasons in different geographic areas. Data show *N. variabilis* is the most abundant vector species in Northern, Midwestern, and Southern U.S. states [37, 38, 55]. The fact that distinct seasonal trends of thrips migration were not detected based on location in northern states suggests that virus vectors may not migrate from areas outside the region, instead, they may colonize other host plants, especially perennial species to overwinter during the absence of soybean and early in the growing season before moving to soybean [38, 55] which highlights the importance of finding and eliminating local virus reservoirs. Considering the peak activity for the primary vector is either at or prior to the occurrence of vein necrosis symptoms [37, 38], it is possible to reduce disease incidence by managing the planting system or planting date, as suggested by Kleczewski [56]. No SVNV-resistant soybean cultivar has been identified at this moment although one study does show a differentiation in symptom intensities among cultivars [35]. Apart from the resistance directly to the pathogen, cultivars that have resistance to virus vector could also reduce disease incidence. Such resistance may result from physical or biochemical features of particular cultivars or the combination of the two factors. To search for potential thrips resistance, we screened soybean cultivars with differential levels of leaf pubescence. Our study demonstrates the feeding damage caused by *N. variabilis* differs among selected cultivars and is correlated to their pubescence levels [39]. The effectiveness of chemical product on disease incidence including insecticide and seed treatment has not be evaluated to date [38].

Summary

The continuous reports of SVNV during the past decade in major soybean-producing areas in North America have drawn attention from the scientific community. Research has been conducted on different aspects of the virus and the disease it causes in order to better understand its biology, epidemiology, and estimate its impact on soybean yield. As a relatively new member of the genus *Orthotospovirus*, SVNV represents a distinct evolutionary linage that has many atypical molecular and biological characteristics. The inefficient movement of SVNV in soybean and the homogeneous population structure across a wide geographic range indicate the virus is most likely to be introduced from another host recently and have not adapted well to soybean [2, 36, 42]. On the other hand, the majority of alternative hosts characterized for SVNV to date belong to the Fabaceae family (Zhou and Tzanetakis, unpublished data); however, peanut (*Arachis hypogaea*)—a host of several orthotospoviruses including GBNV, GRSV, GYSV, and GCFSV is probably not a host of SVNV [2], although more cultivars need to be screened. Collectively, these characteristics are in agreement with the orthotospovirus classification proposed by Inoue and Sakurai [57] which takes into consideration host and vector specificities, suggesting a host-related adaptation of the genus *Orthotospovirus* toward members of the Fabaceae family in the case of SVNV-BNeMV clade. Future investigations on the function of viral proteins and host components may shed light on the special characteristics of this virus–host–vector pathosystem and the evolutionary pathway of orthotospoviruses. A lack of a reliable assay based on mechanical inoculation for SVNV infection of soybean is a bottleneck to perform any study that requires a uniformed disease pressure, such as estimate of the response of different soybean cultivars to virus infection and investigation on the synergistic interactions between SVNV and other viruses prevalent in soybean. On the other hand, virus inoculation using viruliferous thrips as inoculum may be more effective in the identification of alternative hosts given that it could differentiate host preference of virus vectors.

It was reported that SVNV infection on soybean reduces oil content of seeds but have minimal impact on the yield [58]. The case of being able to detect the virus from seedlings derived from seeds collected from SVNV-positive mother plants has raised the profile of SVNV to a seed-transmissible virus [52]. However, the presence of *F. tritici*—a SVNV virus vector in the greenhouse where the study took place–and the lack of genetic information of the unique SVNV isolate that leads to asymptomatic and systemic infection mentioned do not allow for the further study of the mechanisms of virus seed invasion. Seed transmission is a complicated biological phenomenon which involves host genotype, physiological and developmental stage of the host, virus replication, and movement as well as environmental conditions [49–51, 59]. Likewise, the impact of virus on yield and seed quality can also be affected by compounding factors including but not limited to cultivar genotype and timing of virus infection [60–64]. The fact that SVNV infection mostly occurs at the end of vegetative growth stages and the beginning of reproductive stages may mask its true impact on soybean. For those reasons, additional studies are needed to investigate the effects of infection timing on disease symptoms intensity, yield, seed quality, and potential of seed transmission.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Research involving human or animal participants This article does not contain any research involving human or animal participants.

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