Trends in Entomology
Vol. 15, 2019

Review

Invasive cereal aphids of North America: Biotypes, genetic variation, management, and lessons learned

Karen Harris-Shultz[1,*], Scott Armstrong[2] and Alana Jacobson[3]

[1]USDA-ARS, Crop Genetics and Breeding Research Unit, Tifton, GA 31793;
[2]USDA-ARS, Wheat, Peanut and Other Field Crops Research Unit, Stillwater, OK 74075;
[3]Auburn University, Department of Entomology and Plant Pathology, Auburn, AL 36849, USA.

ABSTRACT

Introductions of greenbug [*Schizaphis graminum* (Rondani)], Russian wheat aphid [*Diuraphis noxia*, (Mordvilko)], and sugarcane aphid [*Melanaphis sacchari* (Zehntner)] into the U.S. has disrupted the production of barley (*Hordeum vulgare* L.), sorghum [*Sorghum bicolor* (L.) Moench], wheat (*Triticum* spp. L.) and other small grain crops and has caused great economic losses. In this review article, information is given about each cereal aphid, its biotypic variation, genetic variability, as well as its management. Although multiple biotypes have been identified for the greenbug, Russian wheat aphid, and sugarcane aphid, a limited number of biotypes are of agronomic importance. For the greenbug and Russian wheat aphid, the aphid biotypes of agronomic importance are highly genetically similar. The sugarcane aphid biotype that has spread on sorghum and Johnsongrass [*S. halepense* (L.) Pers.] in all sorghum-growing regions is largely one 'super-clone'. Lessons learned from the past invasions of the greenbug and Russian wheat aphid directly apply to the current sugarcane aphid outbreak. The use of insecticides with multiple modes of action and the use of sorghum hybrids with multiple resistance genes may delay or prevent new sugarcane aphid biotypes from developing. Lastly since the use of classical biological control for management of the greenbug and Russian wheat aphid outbreaks had limited success, classical biological control is not recommended for the management of sugarcane aphids.

KEYWORDS: sugarcane aphid, Russian wheat aphid, greenbug, review.

ABBREVIATIONS

AFLP, amplified fragment length polymorphisms; COI, cytochrome c oxidase subunit I; DAP, days after planting; DAT, days after treatment; MLG, multilocus genotype; RAPD, random amplified polymorphic DNA; MLL, multilocus lineage; NPGS, National Plant Germplasm System; *RMES1*, Resistance to *Melanaphis sacchari*; RWA, Russian wheat aphid; SNP, single nucleotide polymorphism; SSR, simple sequence repeat; US-SCA, predominant sugarcane aphid genotype found on sorghum from 2015-2018.

TABLE OF CONTENTS

*Email id: Karen.Harris@ars.usda.gov

1. Introduction

1.1. Invasive cereal aphids of North America

Aphids (Hemiptera: Aphididae) are major pests of world agriculture and damage plants not only by serving as a vector to numerous plant viruses but they also remove photoassimilates by inserting their salivary stylets into the sieve elements of the phloem [1, 2]. Although an aphid is small in size, aphid populations can become extremely large due to their short generation times and high reproductive rates [3]. Cereal aphids thrive in extensive monoculture agricultural landscapes and can be serious pests to many species within Poaceae [4].

Introductions of greenbug, Russian wheat aphid, and sugarcane aphid into the U.S. have disrupted production of barley, sorghum, wheat and other small grain crops, and necessitated changes in insect pest management programs to reduce economic losses caused by these pests [5-8]. In this article we focus on the greenbug, Russian wheat aphid, and sugarcane aphid biology, biotypes, genetic variation, and management. We also address lessons learned from the greenbug and Russian wheat aphid outbreaks to address the current sugarcane aphid invasion.

1.2. Biotype vs. Genotype

The terms “biotype” and “genotype” are often used synonymously but their meanings are quite different. Biotype, in cereal aphids, is the ability to damage different plant genotypes. Entomologists often use a set of “host differentials”, or a collection of host plants with known resistance or susceptibility to a known biotype, to determine the biotype of an aphid clone [9]. A biotype can be comprised of an indefinite number of genotypes sharing similar virulence genes [10]. The number of aphid biotypes, potentially 2^n, is determined by the number of host resistance genes, where n is the number of non-allelic host resistance genes [11].

The term “genotype” is the genetic constitution of an organism. Over the years genetic variation has been assessed using protein-based markers such as allozymes and for the last forty-five years, DNA-based markers are predominantly used. The use of DNA markers began in 1974 with hybridization-based markers called restriction fragment length polymorphisms (RFLP) that were often codominant and able to identify a unique locus [12]. The generation of RFLP markers was time consuming, required a large amount of DNA (5-20 µg), and used radioactivity to visualize the alleles [13]. To avoid these challenges, PCR-based DNA markers were developed which amplified many regions of the genome, were primarily dominant in nature, but required very small amounts of DNA. These included amplified fragment length polymorphisms (AFLP) [14], random amplified

polymorphic DNA (RAPD) [15], and sequence-related amplified polymorphic (SRAP) markers [16]. Currently, PCR-based markers that amplify specific genomic sites are used. These markers include simple sequence repeat markers (SSR) and single nucleotide polymorphisms (SNPs). SSR markers, also called microsatellites, amplify a tandemly repeated short nucleotide motif of 1-6 bases and are codominant and reproducible [13]. SNPs are polymorphisms at a single nucleotide and advances in next generation sequencing technologies have allowed the identification of large numbers of SNPs.

2. Greenbug

2.1. Greenbug introduction

The greenbug, *Schizaphis graminum* (Rondani) (formerly *Toxoptera graminum*), infests about 70 grass species including wheat, barley, oats (*Avena sativa* L.), rye (*Secale cereale* L.), and sorghum in the southern Great Plains as well as many other parts of the world [17, 18]. Reports of damage caused by the greenbug began in the U.S. in 1884 [19]. In the U.S., greenbug is holocyclic above the 35th parallel and anholocyclic below the 35th parallel [20]. Northern locations are re-infested by alates using low-level jet winds [21, 22]. Annual losses for the Great Plains region ranged from approximately $10-250 million depending on the year [23, 24].

Greenbug feeding can damage sorghum and winter wheat [25, 26]. Infestations may occur annually on sorghum and wheat in the southern Great Plains, but large-scale outbreaks are rare, occurring every 5-10 years [27, 23]. Greenbugs extract large amounts of plant sap, depriving the plant of water and nutrients [28, 22]. These insects also inject enzymes during the feeding process that cause cell wall destruction and tissue necrosis [29]. The greenbug is a carrier of viruses including maize dwarf mosaic virus and barley yellow dwarf virus [30, 31]. Feeding by greenbugs on sorghum has been shown to cause plants to be predisposed to disease such as charcoal rot (*Macrophomina phaseolina*) [32]. Feeding by these aphids inhibits plant growth, may kill plants, and cause less yield and economic return [33, 26, 34].

2.2. Greenbug biotypes

The number of described greenbug biotypes in the U.S. is extensive. Most are considered “lab strains”, and only a few are of agricultural importance. The large number of non-agricultural biotypes is likely due to its ability to reproduce sexually and its wide range of non-cultivated grass hosts [35]. The biotypes were first given letter names and then were given state of collection names (ex. WY1). Greenbug biotype A is avirulent to the hexaploid wheat ‘Dickinson Selection 28A’ (DS 28A). In 1961 Biotype B was identified from greenhouse cultures that was virulent to wheat DS 28A [36]. In 1968 a severe outbreak of biotype C occurred on sorghum in the Midwest and Southwest [25]. It was the first U.S. biotype that was a major pest on sorghum and subsequently became the predominant greenbug biotype on sorghum and wheat [37]. Biotype C attacked sorghum in the summer and wheat in the winter and was warm-temperature tolerant. Biotype C was lighter in color and had little or no black on its cornicles compared to greenbugs on wheat (Biotype B) [25].

Poor greenbug control using disulfoton was reported in 1973 on wheat in the Texas High Plains but was attributed to the weather [38]. Similarly, in 1974 poor greenbug control using granular disulfoton was seen on grain sorghum in the same area and again was attributed to weather. In 1975 biotype D was identified on grain sorghum in the Texas High Plains that is resistant to disulfoton, an organophosphorous insecticide that was used at the time [38]. However, biotype D does not match the definition of a biotype as it is not based on insect-plant resistance relationships [35].

In 1980, biotype E was identified based on its ability to damage biotype C resistant sorghum and wheat [39]. Biotype E was originally collected on wheat in Bushland, Texas in 1979 and had spread 75 miles north of Bushland by May 1980. By 1981, biotype E replaced biotype C greenbugs over most of Texas, Kansas, Oklahoma, and Nebraska [40]. In 1984-1985 biotype C was the dominant greenbug biotype present in Arkansas wheat but biotype E was also present in the county samples (0-59%) [41]. In 1986 biotypes E and B were the predominant biotypes in Oklahoma in the spring (83% and 11% respectively) on wheat, biotypes E and C were

predominant on sorghum in the summer (94% and 6%, respectively), and biotypes E and C were predominant in the fall (97% and 2%, respectively) [42]. Biotype E preferred sorghum more than barley, oats, or wheat [43].

In 1986 biotype F was found in Ohio on Kentucky bluegrass (*Poa pratensis* L.) [44]. Biotype F can kill Canada bluegrass (*P. compressa* L.) and morphologically has no dorsal stripe. In 1988, biotype G was identified from Oklahoma that can damage all known resistant sources of wheat [43]. In the same study, biotype H was identified from Texas that can damage 'Post' barley but was avirulent on all sorghum lines tested. Both biotypes G and H lack the middorsal dark green abdominal stripe and have the general appearance of biotype F [43]. Biotypes F, G, and H preferred the small grains significantly more than sorghum [43].

In 1990, biotype I was identified on severely damaged sorghum in Kansas that was resistant to biotype E [45]. Fortunately, Biotype I was not virulent on greenbug biotype E-resistant wheat, barley, oat, and rye. In 1991-1993 greenbug biotypes E and I were collected from Kansas, Texas, Nebraska, Colorado, and Oklahoma from sorghum and wheat fields [37]. In 1994, biotype J was identified that was collected from wheat in Idaho in an area where barley is the major small grain crop [46]. Biotype J did not cause necrotic lesions in any of the wheat or barley entries tested and it was the only biotype tested that was able to kill 'Post' barley.

Biotype K was collected in 1992 in Kansas and damaged sorghum plants that were resistant to biotype I [47]. Fortunately, all small grain genotypes that had resistance to biotype I also had resistance to biotype K. The authors expressed concern that the widespread use of sorghum hybrids derived from PI 550610 could suppress the development of greenbug biotypes E and I but increase the amount of biotype K [47]. Many more biotypes have been identified [48] but only C, E, and I have caused significant economic losses to sorghum [47]. Over the years there has been a shift in the prevailing biotypes of C to E and then E to I [48]. Currently E, I, and K are impacting sorghum and wheat [49-51].

Other biotypes namely NY, FL1, SC, and KS1 have been identified using the host plant differentials listed in Table 1. The NY biotype was found to be susceptible to the sorghum differentials with the exception of PI 550607. The FL biotype was found on seashore paspalum (*Paspalum vaginatum* Swartz) turfgrass in November of 2003 at Belle Glade, FL [52] but is susceptible to all sorghum differentials and thus far not observed to be a pest of sorghum, wheat or barley within the US. The SC biotype has not been evaluated for sorghum but was discovered to be a different biotype based on the wheat, rye, and barley evaluations. KS1 is noted for being resistant to all four sorghum differentials but again, has not been identified as of this date to be found in sorghum. Subsequent collections of five new greenbug biotypes have been made from Wyoming, namely WY10 MC, WY81, WY10 B, WY12 MC and WY86 but these were found to be important to barley [53] and not listed in Table 1.

2.3. Greenbug genetic variability

Genetic diversity has been examined for the agronomically important greenbug biotypes as well as the other biotypes. In all the studies, the biotypes of agronomic importance C, E, I, and K were consistently grouped together. This suggests that using resistant cultivars exerts selection pressure on biotypes that may be selecting for mutants with greater virulence. Zhu-Saltzman *et al*. [54] used1775 AFLP markers to examine the genetic diversity of the biotypes of agronomic importance as well as four other greenbug biotypes. They found biotypes C, E, I, and K were between 92-98% genetically similar. Weng *et al*. [55] used 67 SSR markers, of which many were developed from other aphid species, to genotype greenbug biotypes C, E, I as well as three isolates from Wyoming. They found the biotypes C, E, and I grouped together. Later, 31 SSR markers were used to assess the genetic diversity of clonal greenbugs collected from Colorado and Wyoming as well as the biotypes E, G, H, I, and K [18]. In agreement with Zhu-Saltzman [54], the agriculturally important biotypes E, I, and K were genetically similar. Furthermore, biotypes E, I, and K were most similar to greenbugs collected from Colorado. The greenbugs that Weng *et al.* [48] collected were grouped by the place of collection. Kharrat *et al.* [56] used RAPD markers to examine genetic diversity of greenbugs collected from Tunisia and the biotypes C, E, I, K, F, G, and H. They too found that the agronomically important

Table 1. Response of wheat, rye, barley, and sorghum to multiple greenbug biotypes.

	Wheat								Rye		Barley			Sorghum			
Colony	**Custer**	**DS 28A**	**Amigo**	**Largo**	**CI 17959**	**CI 17882**	**GRS 1201**	**W 7984**	**Elbon**	**Insave**	**Wintermalt**	**Post 90**	**PI 426756**	**Tx 7000**	**Tx 2737**	**Tx 2783**	**PI 550607**
Resistance Gene	-	*gb1*	*Gb2*	*Gb3*	*Gb4*	*Gb5*	*Gb6*	*Gb7*	-	*Gb2, Gb6*	-	*Rsg1a*	*Rsg2b*	-	?	?	?
B	S	S	R	S	S	S	R	S	S	R	S	R	R			S	R
C	S	S	R	R	R	R	R	R	S	R	S	R	R	S	R	R	R
E	S	S	S	R	R	R	R	R	S	R	S	R	R	S	S	R	R
F	S	R	S	S	S	S	S	S	S	S	S	R	R			S	S
G	S	S	S	S	S	S	R	S	S	R	R	R	R	S	S	S	R
H	S	S	S	R	S	S	S	S	S	S	S	S	S				R
I	S	S	S	R	R	R	R	R	S	R	S	R	R	S	S	S	R
K	S	S	S	R	R	R	R		S	R	S	R	R	S	S	S	S
NY	S	R	R	S	S	S	S		S	R	S	R	R	S	S	S	R
FL1	S	R	R	S	S	S	S	S	S	R	S	R	R	S	S	S	S
SC	S	S	R	S	S	S	S		S	R	S	R	R				
KS1	S	R	R	S	S	S	S		S	R	S	R	R	R	R	S	R

Data was taken from references [9, 36, 39, 43-47, 49, 52, and 67]. R = resistant, S = susceptible reactions. Custer, Elbon, Wintermalt, and Tx7000 were used as susceptible checks for their respective species. '?' indicate the gene has not been identified from these sources to date. Blank cells indicate data is not available.

biotypes C, E, I, and K were grouped together and had similarity to samples collected from Tunisia. Similarly, Shufran *et al*. [57] sequenced a 1.4 kb fragment of the cytochrome c oxidase subunit I (COI) gene from 12 greenbug biotypes. The COI fragment is commonly sequenced to determine genetic diversity and phylogenetic relationships among samples as well as for species identification. They too found the agricultural biotypes C, E, I, and K grouped together, as well as biotype J that is non-virulent to wheat. Genetic distances among this clade ranged from 0.08-0.61%. Thus, with this amount of similarity between biotypes C, E, I, K, and J it is very unlikely that these agricultural biotypes are products of sexual reproduction and are more likely a collection of mutants in a common genetic background.

Genetic diversity within greenbug biotypes has been examined [9]. From 1995-2000 greenbugs were collected from a wide range of hosts in Oklahoma, Kansas, Colorado, South Carolina, and Syria. Clonal colonies were established, biotypes were determined, and a 1 kb region of the COI gene from each colony was sequenced. Three clades were identified of which Clade 1 (which had only 1.1% sequence divergence among samples) consisted of most of the samples classified as biotype I, all of the samples of biotype E, and a sample of biotypes K, J, C, SC, and G. Clade 2 consisted of most of the samples of biotype G and a sample of biotypes F, NY, I, and K. Clade 3 consisted of a sample from a previous study collected in Europe, a sample collected from Canada wild rye (*Elymus canadensis* L.), a sample classified as biotype B, and three samples classified as biotype I. A correlation existed between host genus of collection and clade which supports that these clades could have diverged on separate hosts. Greenbug samples collected from *Sorghum* or *Triticum* spp. were grouped in Clades 1 and 3 whereas samples collected from wheatgrass (*Agropyron* spp.) were primarily grouped in Clade 2.

2.4. Greenbug management

2.4.1. Insecticide use: insecticides and seed treatments

For small grains, parathion was used for greenbug control from 1948-1968 with no reports of aphid resistance to the insecticide [38]. After 1968, when biotype C became a major pest on sorghum, disulfoton was used to control the greenbug on grain sorghum and small grains with parathion being largely replaced. Thus, the greenbug population in some areas of the Texas High Plains was subjected to multiple applications of disulfoton in the winter on small grains and then multiple applications of the same chemical in the summer on sorghum [38]. Furthermore, many producers were using insecticide rates higher than the recommended rates and not monitoring greenbug population densities [38]. This led to the development of Biotype D, which are greenbugs resistant to organophosphates [38].

Systemic insecticides applied to winter wheat as seed treatments were also evaluated to control biotype C greenbug [58]. Disulfoton at 1.25, carbofuran at 3-5, and UC 21865 at 20-40 g AI/kg seed protected wheat from greenbug feeding, with limited phytotoxicity, for approximately 60 days after planting (DAP).

In 1993-1994, Gaucho (imidacloprid) was used as a seed treatment at two rates for three different sorghum hybrids in Kansas [59]. Both the 2 and 4 oz rates (per 100 lbs. seed) were effective at reducing greenbug numbers on seedling plants less than 14 d old. Only the 4 oz rate of Gaucho reduced greenbug numbers for 70-80 DAP on the greenbug-susceptible hybrid. Thus the 4 oz formulation was recommended for early and late season greenbug control.

2.4.2. Cultural practices: reduce tillage and adjust planting times

Cultural practices can be altered to control insect pests. Burton and Krenzer [60] found that greenbug populations were reduced in wheat plots where surface residues were moderate to high as compared to conventionally tilled plots. They later performed a similar experiment using sorghum [61] and found that reduced tillage and/or crop residues on the soil surface decreased the number of greenbugs and plant damage.

Harvey *et al*. [62] trapped greenbugs from 1974-1979 at Hays, Kansas and found two distinct flight periods. The first flight peaked during the first week of June and the second during the last week of July. They suggested altering the planting date of wheat and sorghum to avoid these peak flight times.

The time of planting has also been seen to influence greenbug abundance in winter wheat. Earlier planted

wheat supports early colonization of the greenbug and greater numbers of aphids as compared to later plantings [63]. Furthermore, incidence of Barley yellow dwarf virus declined with later plantings [63].

The use of a crop also has an impact on greenbug abundance. The use of winter wheat for grazing stocker cattle can reduce greenbug abundance as much as 98% as compared to non-grazed plots [64]. Another study found that grazing reduced aphid abundance as much as 87% and Barley yellow dwarf virus levels as much as 70% but often promoted greater abundance of grassy weeds [65]. They also found that grazing was correlated with reduced yields when aphids were not present.

2.4.3. Development of resistant cultivars

2.4.3.1. Wheat, rye, and barley

In the U.S., the start of breeding for resistance to greenbugs began with the selection of DS 28A from wheat that was resistant to greenbug biotype A [66]. The resistance from DS 28A was caused by a single recessive gene later named *gb1* [66, 67] (Table 1) and the resistance mechanism was described as tolerance to greenbug toxins resulting in less chlorophyll loss and reduced greenbug reproduction [68, 69]. For rye, 'Insave' rye was developed that was resistant to biotypes B, C, E, G, I, J, and K [49]. Greenbug resistance was transferred from 'Insave' rye to 'Amigo' wheat [70] to confer resistance to biotypes B and C. This greenbug source of resistance is conferred by a single dominant gene later named *Gb2* [71, 67] (Table 1) and the source of resistance in 'Amigo' was transferred to the red winter wheat cultivar 'TAM107'. 'TAM107' was first made available to growers in 1984 [72]. Similarly, the wheat-rye hybrid cultivar 'Gaucho' has resistance to biotype C and its resistance is also derived from the rye cultivar 'Insave F.A.' [73].

The wheat line 'Largo' was selected from a cross between 'Langdon' durum (*Triticum turgidum* L.) and the resistant source PI 268210 (*Aegilops tauschii* Coss.) [74, 75]. 'Largo' has resistance to biotypes C, E, H, I, J, and K [49, 76]. This resistance was found to be conferred by a single dominant gene later named *Gb3* [74, 67] (Table 1). CI 17959 and CI 17882 were found to have resistance to biotypes C, E, I, J, and K [49, 77, 78]. The resistance was conferred to CI 17959 and CI 17882 by single dominant genes named *Gb4* and *Gb5*, respectively [67] (Table 1). Biotype B, C, E, G, I, J, and K [49, 76] resistance was identified in wheat-rye translocation germplasm 'GRS-1201'. 'GRS-1201' has resistance from a single dominant gene named *Gb6* [76] (Table 1). Later W7984, a synthetic hexaploid wheat line, was found to have resistance to greenbug biotypes C, E, and I [79] (Table 1). This resistance was conferred by a single dominant gene and was designated *Gb7*.

Currently there are two known greenbug resistance genes in barley. *Rsg1a*, previously called *Grb*, is a single dominant resistance gene in PI 87181 ('Omugi') and 'Post 90' [80-82]. *Rsg2b* is a single dominant resistance gene from PI 426756 [81]. Both resistance genes confer resistance to a wide range of greenbug biotypes except biotype H [83, 84] (Table 1).

2.4.3.2. Sorghum

Sorghum breeders obtained biotype C resistance by using tunis grass [*Sorghum virgatum* (Hackel) Stapf] [85]. Resistance was reported in 'KS30' (a tunis grass × 'Combine Kafir-60') and 'Shallu'. Both sources of resistance appeared to be derived from tunis grass [86]. KS30 was released in 1969, resistance is simply inherited, and the mechanism of resistance is primarily tolerance [85].

In the 1970s, the Texas Agricultural Experiment Station released a series of biotype C-resistant hybrids with SA 7536-1, PI 264453, 'KS30', and IS 809 as the sources of resistance [35]. These lines were used to develop sorghum hybrids for the Southern Plains and the first biotype C-resistant hybrid sorghum was reported in 1975 [87]. Use of the biotype C-resistant hybrids increased and in 1979 50-60% of hybrids in the Southern Plains were biotype C-resistant [35]. By 1980, at least 90% of the sorghum acreage was planted with biotype C-resistant hybrids [88].

After the discovery of biotype E in 1980, commercial grain sorghum lines that were resistant to biotype C were found to be susceptible. The sorghum lines PI 220248, PI 264453, 'Capbam', and a bloomless mutant were found to be resistant to biotype E [89]. Dixon *et al*. [88] found that PI 264453 and PI 220248 had resistance to biotype E that was complexly inherited, and PI 264453 had a major factor in its cytoplasm that was controlling resistance.

More than 23,000 sorghum accessions were screened during the 1980s for resistance to greenbug biotype E and only six sources were resistant [45]. In 1981, Tx2783 was released with resistance to biotypes C and E [90]. The resistance was reported to be derived from 'Capbam'. Sorghum hybrids with resistance to greenbug biotype E were available starting in 1982 and by 1986, 38% of the total sorghum seed sold in Oklahoma was resistant to biotypes E and C, and 53% of seed sold had only biotype C resistance [42]. By 1990 biotype E-resistant hybrids were grown on 40-50% of the acreage in the Southern Plains [35].

Greenbug biotype I was collected in 1990 and sorghum lines that were resistant to biotype I were identified [91] and were utilized in public and private breeding programs. A tetraploid Johnsongrass line PI 266965, a commercial sorghum hybrid Cargill 607E, and a sorghum line from Syria, PI 550610, were found to have resistance to biotype I [91]. After biotype K was discovered in 1995 [47], which was virulent on biotype I-resistant sorghum, 115 hybrids were tested for resistance to greenbug biotypes E, I, and K [92]. Of these, 75 were resistant to biotype E but only Cargill 607E was resistant to biotype K [92, 47].

2.4.4. Conservation of natural enemies

Natural enemies can reduce greenbug density and prevent them from reaching their reproductive potentials [22]. Natural enemies of greenbugs include lady beetles (Coleoptera: Coccinellidae), lacewings (Neuroptera: Chrysopidae), spiders (Araneae), syrphid flies (Diptera: Syrphidae), parasitoid wasps (Hymenoptera: Aphelinidae, Aphidiidae, Braconidae) [93], damsel bugs (Hemiptera: Nabidae), ground beetles (Coleoptera: Carabidae), and entomopathogenic fungi (Entomophthorales and Hypocreales) [94, 8]. Coccinellids often have the strongest impact of all aphidophagous insects [95] and regulation of greenbugs is largely due to coccinellids in sorghum and wheat [96, 97]. Hymenopterous parasitoids, and to a large degree *Lysiphlebus testaceipes*, a generalist aphid parasite, are known to suppress greenbugs in wheat and sorghum [98, 99].

After the outbreak of biotype C greenbug on sorghum planted in the Great Plains, eleven species of hymenopterous parasitoids were imported into the United States from Europe, Iran, Pakistan, and Chile [17]. Six species of wasps, *Aphelinus asychis* (Walker), *Aphelinus varipes* (Forester), *Aphidius matricariae* Haliday, *Diaeretiella rapae* (McIntosh, *Ephedrus plagiator* (Nees), and *Praon pakistanum* Kirkland were released in Oklahoma, Texas, and other states in the Great Plains [100, 17]. Follow-up surveys did not identify permanent establishment of any of the released species except for those species that were already present in the areas prior to the release [17].

3. Russian wheat aphid (RWA)

3.1. RWA introduction

The Russian wheat aphid (RWA), *Diuraphis noxia*, (Mordvilko) (Hemiptera: Aphididae) was first reported by Mokrzecki in 1900 from fields of barley in Eupatoria, Crimea [101] and, since then, has dispersed globally to every major cereal production area. It is pale green in color, about 2 mm long, and feeds on the base of newly formed leaves as well as the inflorescence of barley and wheat [102]. Its native range is thought to be the area between the Caucasus Mountains and the Tian Shan (Mountains of Heaven) [103]. The RWA has a host range of at least 47 cool-season grasses and 18 warm-season grasses [104]. Russian wheat aphids use non-cultivated hosts such as crested wheatgrass (*Agropyron cristatum* (L.) Gaertner), Canada wild rye, and volunteer wheat and barley during the summer months when cultivated grains are not grown [105]. Invasive clones traveled from Turkey to South Africa in 1978 [106] and to Mexico in 1980 [107]. From Mexico the invasive clones moved to the U.S. (Texas) in 1986 [108] and to Chile in 1987 [109]. By 1988 the RWA moved across the western U.S. and Canada [103] leading to direct and indirect loss of $893 million mainly on barley and wheat in the western U.S. between 1987-1993 [110]. The RWA was a severe pest from the mid-1980s through the mid-1990s in the southern and central-western edges of the Great Plains [17]. The RWA population has declined since the mid-1990s [111].

Russian wheat aphid feeding results in reduced chlorophyll levels and a decrease in the capacity and efficiency of photosystem II in susceptible wheat and barley [112]. Russian wheat aphid further causes leaf rolling, plant stunting, purple

discoloration, prostrate growth, and longitudinal white leaf streaking [113, 114]. In addition, new leaves of infested susceptible plants do not unroll causing trapped awns and deformed spikes which leads to reduced grain yield [102]. The RWA is a poor vector of plant viruses despite earlier reports [103]. The RWA is primarily anholocyclic in North America but holocyclic populations occur in higher elevations of the Colorado Plateau [115].

3.2. RWA biotypes

Many biotypes of the RWA have been identified in the U.S. and have been monitored through the years to determine their prevalence. The biotype that first arrived in 1986 was later named RWA1. As the years passed, minor biotypic variation was found which included differences in damage to susceptible wheat [116], clones that differ in life cycle [117], and a clone that had a different cuticular hydrocarbon profile [118]. In 1994-1995, clones were collected from five western states and no new biotypes were identified [119]. In 2003, Prairie Red, a RWA-resistant hard red winter wheat line containing the *Dn4* resistance gene [120], was found infested with RWA in multiple areas in southeastern Colorado [121]. This new biotype, RWA2, induces greater injury on resistant and susceptible wheat cultivars. Only one accession tested, 94M370 (*Dn7*), had resistance to RWA2 but the *Dn7* gene exists in a region of the genome that has often been shown to have adverse quality effects on leavened bread products [122, 123].

Three new biotypes (RWA3-5) were identified from collections on wheat and barley from Kansas, Nebraska, Texas, and Wyoming from 2002-2003 [124]. The new biotypes were found from collections made in Texas on wheat (RWA3-4) and one from Wyoming on barley (RWA5). In 2005 collections were made from 98 fields of wheat and barley in Oklahoma, Texas, New Mexico, Colorado, Kansas, Nebraska, and Wyoming to determine the distribution and abundance of biotypes RWA1-5 [125]. Only RWA1 and RWA2 were identified and the biotype composition across all collection sites was 27% RWA1 and 73% RWA2. Thus, RWA1 and RWA2 were the biotypes of agricultural importance in 2005 and RWA2 predominated in most states. In 2008, three new biotypes were found (RWA6-8) from Colorado collections and RWA7-8 were found on non-grass hosts [126]. In 2007 a breakthrough was made with the finding that the RWA can reproduce sexually in the U.S. [115]. Newborn aphids were found from eggs in western Colorado. Interestingly, fundatrices (stem mothers) could be separated from alate vivipara (females produced parthenogenetically) by the number of segments in the antennae (5 verses 6 segments, respectively). Thirty-five new biotypes were identified on wild grasses and wheat; yet the authors did not name these, preferring to name only those with agricultural significance. Puterka *et al*. [127] combined RWA3, RWA4, RWA5, and RWA7 into one biotype renamed RWA3/7. This reclassification reduced the number of RWA biotypes to five namely RWA1, RWA2, RWA3/7, RWA6, and RWA8. Biotypes were reexamined in 2011 and 2013 in the Colorado Plateau and the Central Great Plains Regions. For both years the biotype that was mainly represented was RWA6 which is avirulent to *Dn4*-containing wheat. RWA1, RWA2, RWA3/7, and RWA8 were also detected in these regions but at lower levels than RWA6. The shifting of biotypes coincides with the decreased use of resistant wheat containing the *Dn4* gene after 2003.

3.3. RWA genetic variability

Russian wheat aphid genetic variability has been monitored in the U.S. and is low. Puterka *et al*. [128] genotyped 36 RWA collections from the U.S., Canada, France, Mexico, South Africa, Syria, Turkey, and Ukraine using seven RAPD markers and seven alloyzmes. The aphids were collected from wheat, oat, barley, and wheatgrass. Similarity was found among the Turkey, France, Canada, Mexico, South Africa, and U.S. samples suggesting a common origin. Robinson *et al*. [129] used 18 RAPD primers on a collection from Mexico, Chile, Syria, South Africa, France, and Canada. The RWA clones from Mexico, Chile, the U.S., Canada, France, and South Africa were highly genetically similar. Shufran *et al*. [119] used seven RAPD primers on a collection of aphids sampled in 1994-1995 from Kansas, Nebraska, Colorado, Wyoming, and Washington on cultivated and non-cultivated hosts. Only minor genotypic differences were found. Shufran and Payton [130] genotyped RWA1-5 with 58 RAPD primers, eight simple sequence repeat (SSR) markers, and used COI sequencing. No COI sequence variation or SSR polymorphism was seen between

samples. Two RAPD markers displayed polymorphism among the individual clones. These results suggest that the RWA1-5 are highly related. Liu *et al*. [131] used AFLP markers to genotype populations of RWA from Chile, Czech Republic, Ethiopia, Hungary, Iran, Kenya, Mexico, Syria, Spain, and the U.S. (including RWA1-5). They found RWA1-2 and RWA3-5 belong to the Middle East African Clade and European Clade, respectively. RWA1-2 were most similar to the RWA sample from Mexico. They suggest at least two invasions of RWAs occurred in the U.S. and perhaps three. The samples from the Middle East African Clade, which include RWA1-2, coincide with the documented history of spread of the RWA from the Middle East to South Africa, to Mexico, and to the U.S. This is in agreement with Shufran *et al*. [132] who suggested that RWA1 and RWA2 are likely the by-product of a single invasion.

3.4. RWA management

3.4.1. Cultural practices

To reduce RWA numbers, hosts of the RWA should not be allowed to grow during the non-crop season [106]. Host plants such as wheat, barley, triticale (*Triticum* × *Secale*), and Agrotricum (*Agropyron* × *Triticum*) should not be used as pasture grasses [106]. During the non-crop season, volunteer and weedy hosts should be eliminated to prevent increasing RWA pest numbers.

For RWA control, agronomic practices should be considered [106]. The planting date has been shown to impact RWA infestation. Russian wheat aphid infestation levels are usually higher in early planted wheat in Montana [133], northeastern Colorado [134], western Colorado [135], and Canada [136]. A two-year study in 1992-1993 was conducted in western Colorado on winter wheat examining the effects of planting date on RWA infestation and viral diseases [137]. Using five planting dates from September until late October at two-week intervals, the highest yield occurred on the third planting date (Oct. 2-3). In agreement with the previous studies, the highest RWA numbers were seen at the first planting date when no insecticides were used.

The use of irrigation during periods of low precipitation is recommended to reduce RWA populations in wheat [138]. Indeed, well-watered field-grown wheat had lower RWA densities than non-irrigated wheat and aphid density was not impacted by fertilizer rate [138].

3.4.2. Host resistance and insecticide use

Russian wheat aphid was managed in the U.S. by the use of aphid-resistant barley and wheat cultivars. Prior to 1990, all barley cultivars grown in the U.S. were susceptible to RWA1 [139]. The USDA-ARS in Stillwater, OK screened over 24,000 barley accessions in a greenhouse from 1990-1993. They identified 109 accessions with some level of resistance and selections were made to produce homogenous RWA1-resistant lines from each of the 109 accessions [139]. Two germplasm lines, STARS-9301B and STARS-9577B were publicly released [140, 141]. The sources of resistance in STARS-9301B and STARS-9577B are from PI 366450 and CIho 4165, respectively, both of which were collected from Afghanistan [140, 141]. Additionally, Smith *et al*. [142] identified sources of RWA1 resistance from Iran and the Soviet Union.

More than 25,000 wheat accessions were screened for RWA resistance by the Western Coordinating Committee No. 66, a group of scientists committed to the development of cultivated cereals with host plant resistance to the RWA [143]. At least 86 accessions were found to have reproducible resistance to RWA, and accessions collected from the ancestral RWA boundaries of Central Asia had the highest frequency of resistance [143]. The USDA-ARS released two RWA-resistant wheat lines, STARS-9302W and STARS-9303W in 1993 [144]. The source of resistance in both lines was from PI 149898, a line donated to the National Plant Germplasm System (NPGS) from the Saratov Russian Federation. ‘Halt’ was released as a red winter wheat variety in 1994 and was the first RWA-resistant wheat cultivar that possessed *Dn4* resistance developed in the U.S. [145]. Field evaluations from three different locations in Colorado concluded that a sister line of ‘Halt’ reduced RWA reproduction and damage potential when compared to the standard susceptible ‘TAM 107’ [146]. ‘Prairie Red’ was released in 1998 [120]. The source of RWA resistance in ‘Halt’ and ‘Prairie Red’ is from PI 372129, an accession from Turkmenistan [147, 145].

By 2003-2004 only approximately 25% of the Colorado winter wheat acreage was planted with

aphid-resistant cultivars [121] as for many areas the aphid had no longer been a persistent problem [125]. Wheat varieties that contained the *Dn4* gene were able to manage the pest in the Great Plains from 1995-2003.

In North America, farmers applied insecticides and delayed plantings of their crops to control the RWA in barley [148, 142] and wheat prior to the use of resistant cultivars starting in 1994 [125]. In 1989, 916,000 ha of wheat and barley were treated with insecticides at a cost of $21 million [102]. The use of RWA-resistant cultivars was an economical solution for wheat and barley growers to the RWA until resistance was overcome in wheat in 2003. Fortunately, RWA1 to RWA5 do not severely damage the primary sources of resistance in barley, STARS-9301B and STARS-9577B [140-141, 149].

3.4.3. Classical biological control and endophyte-infected grasses

The combination of plant resistance with natural biological control would be the ideal strategy for the management of the RWA. Biological control alone is unlikely to be an efficient management strategy [102]. Naturally occurring insect enemies of the RWA include several species of coccinellids, syrphids, chrysopids, and parasitoid wasps [102].

From 1988-1994, USDA-ARS and university scientists made 62 collection trips to 17 countries to collect predators and parasitoids of *D. noxia* [150]. Twenty-nine species of predators and parasitoids as well as six species of fungal pathogens were collected. Federal and state scientists shipped mummified aphids containing the parasitoids *Aphelinus albipodus* Hayat & Fatima, *A. asychis* Walker (Hymenoptera: Aphelinidae), *Diaeretiella rapae* (M'Intosh), *Aphidius matricariae* Haliday, *Aphidius colemani* (Viereck), *Aphidius picipes* (Nees), *Aphidius rhopalosiphi* DeStefani-Perez, *Ephedrus plagiator* (Ness), and *Praon gallicum* Stary (Hymenoptera: Braconidae) for rearing and release [151]. These parasitoids and predators were released in 16 states. Efforts to establish predators were reported as unsuccessful [151] but three species of parasitoids, *A. albipodus*, *A. asychis*, and *D. rapae* spread throughout the wheat production region in Wyoming within five years of release [111]. Likewise, after the release of seven exotic hymenopterous parasitoids in eastern Colorado from 1991-1993, *A. asychis* and *A. albipodus* were recovered in Colorado one year later from the releases [152]. Similarly, in Washington and Idaho, seven species of parasitoids were released and after six years three species, *A. albipodus*, *A. uzbekistanicus* Luzhetzhi, and *Praon gallicum* Stary, were found that could be attributed to the release program.

Additionally, the RWA is susceptible to endophyte-infected grasses. In replicated field plots, the endophyte-infected perennial ryegrass, *Lolium perenne* L. had less RWA than endophyte-free ryegrass [153]. This may be due to the fungal production of alkaloids in infected plants that are toxic to insects and livestock [154].

4. Sugarcane aphid

4.1. Sugarcane aphid introduction

The sugarcane aphid, *Melanaphis sacchari* (Zehntner) (Hemiptera: Aphididae), is a pest on sorghum and sugarcane (*Saccharum* spp.) in Africa, Asia, Australia, and parts of Central and South America [155]. Its distribution follows the cultivation of sorghum and sugarcane and its host range includes the genera *Cynodon*, *Miscanthus*, *Oryza*, *Panicum*, *Paspalum*, *Pennisetum*, *Saccharum*, *Setaria*, *Sorghum,* and *Zea* [155]. The sugarcane aphid is largely anholocyclic but sexual oviparae have been observed in India [156].

Sugarcane aphids have become a serious, perennial pest on grain, forage, and sweet sorghum in the United States since its discovery on sorghum near Beaumont, TX in 2013 [5, 157]. Since then it has spread in all directions and currently has spread to 25 states as well as Mexico, thus infesting all sorghum-production regions of the U.S. and Mexico [158-160]. The ability to rapidly move from state to state is attributed to high alate production and alates traveling by wind-aided movement [5]. The host range of the invasive sugarcane aphid in the U.S. includes *Sorghum* spp. such as the pervasive weed Johnsongrass (*S. halepense* [L.] Pers.), Sudan grass (*S. bicolor* subsp. *drummondii* [Nees ex Steud.] de Wet & Harlan, giant miscanthus (*Miscanthus sinensis* × *M. sacchariflorus* Greef & Deuter ex Hodkinson & Renvoize) and Columbus grass (*S. almum* Parodi) as well as sugarcane and energycane (*Saccharum* hyb.) [161-163]. Sugarcane

aphids have been previously reported in Florida and Louisiana feeding on sugarcane [164, 163] and they were also documented feeding on sorghum in Florida prior to the recent outbreak but were not considered of economic importance at that time [165].

Sugarcane aphids are tan, orange, lemon-yellow, or gray with prominent black cornicles and dark feet [157, 155]. The gray body color is seen during cool conditions such as in winter or fall, and the winter phenotype can survive several successive overnight freezes [5, 166]. These aphids have predominantly asexual reproduction [155]. The mean longevity of apterae on sorghum was 28 d and a single aphid can produce on average 86-96 nymphs depending on the age of the plant [167]. The sugarcane aphid has one of the fastest reproductive rates of aphids on sorghum and doubling time values on susceptible hybrids ranged from 3.9-7.9 d [168-171]. No sexual forms have been observed in the U.S., but they have been found in Mexico [172].

Sugarcane aphids feed on sap from phloem tissue of the leaves, stem, and the panicle of the plant [168]. Initial colonies begin on the lowest leaves of the plant and as the colonies grow and the leaves die, the aphids move up the plant [157]. Populations exceeding 10,000 aphids on a single plant have been recorded [5]. Aphid feeding causes yellow to red or brown pigmentation on the leaves, followed by leaf chlorosis, leaf necrosis, stunted growth, increased plant water stress, poor plant vigor, delay or prevention of head emergence, and can result in plant death [173, 159, 155, 157]. Plant damage occurs from a loss of plant nutrients and sugars and a reduction in photosynthetic efficiency due to sooty mold buildup that grows from the honeydew deposited on the plants by the aphids [155, 5]. Damage caused by aphid feeding decreases sorghum yield, reduces seed weight, and lowers grain quality [5]. In addition to the damage that the sugarcane aphid has on sorghum plants, honeydew also covers the plant creating a problem during harvest, as combines become clogged [161]. Yield decline on susceptible hybrids ranged from 50-100% in infested fields [168, 157].

The sugarcane aphid is a common and efficient vector of the Sugarcane yellow leaf virus on sugarcane [174]. Indeed, sugarcane is widely infected in the United States and many other countries. This *Polerovirus* causes leaf yellowing symptoms, known as sugarcane yellow leaf disease, of which some sugarcane cultivars are resistant to virus infection [174]. For susceptible sugarcane cultivars, yield losses of up to 20% have been reported [175]. Sugarcane yellow leaf virus was detected in Columbus grass [176] and grain sorghum collected in Florida [175]. None of the grain sorghum plants infected showed yellow leaf symptoms [175].

4.2. Sugarcane aphid genetic variability

Genetic diversity was examined in sugarcane aphid samples collected worldwide from 2002-2009 [177]. Genotyping conducted using ten SSR markers and the sequencing of a fragment of the COI gene identified five multilocus lineages (MLL) whose distributions were structured geographically. Multilocus genotypes (MLG), where a MLG is a combination of alleles found at two or more loci in a single individual, that differed slightly due to mutation or scoring errors belong to the same MLL. Sugarcane aphids that were classified as MLL-A were collected from Africa, those classified as MLL-B were found in Australia, those classified as MLL-C were from South America, the Caribbean, the Indian Ocean, and East Africa, those classified as MLL-D were from the U.S., and those sugarcane aphids classified as MLL-E were collected from China [177]. Sequencing of the COI fragment identified three haplotypes (three different sequences; identical sequences were assigned the same haplotype) with no association between haplotype and the host plant. Similarly, there was no association between MLL and host plant. Thus, no molecular evidence was found for the separation of sugarcane aphids into *M. sacchari* and *M. sorghi* [177]. Furthermore, there was evidence that these populations have been reproducing clonally for a long time (high heterozygote excess, low genetic diversity).

Host plant specialization was seen for the sugarcane aphid in Reunion Island [178]. Sugarcane is the dominant crop while wild sorghum (*S. bicolor* (L.) Moench subsp. *verticilliflorum* (Steud.) de Wet ex. Wiersema & J. Dahlb) is a common weed on Reunion Island. Multilocus genotypes Ms11 and Ms16, which belong to MLL-C, were observed more frequently on sugarcane while Ms15 was

collected from colonies on wild sorghum. Use of lab transfer experiments found that Ms11 clones performed better on sugarcane than sorghum whereas the Ms15 lineage developed poorly on sugarcane. In contrast the Ms16 lineage had no difference in performance between sorghum and sugarcane. This study supports the existence of host plant specialization within a MLL of the sugarcane aphid despite low genetic differentiation [178].

Sugarcane aphid diversity was examined from collections made on sorghum from seven U.S. states and one territory in 2015, two years after the invasion of the sugarcane aphid on grain sorghum [179]. Genotyping of 46 collected samples using 52 SSR markers found that a single sugarcane aphid genotype predominates (45/46 samples) over a large geographic area on sorghum and supports that the sugarcane aphid is reproducing asexually in the U.S. [179]. Furthermore, sugarcane aphid samples collected from multiple states in 2016 from sorghum or in 2015 and 2016 from Johnsongrass were primarily the same predominant genotype found on sorghum in 2015 [180]. This predominant genotype was named the US-SCA genotype. This study confirmed that the US-SCA genotype uses Johnsongrass as an alternative host [180].

To determine if the US-SCA genotype is a new genotype or a genotype previously identified, Nibouche *et al.* [181] performed a follow-up study comparing sugarcane aphid samples collected from 2013-2017 in North America with the worldwide collected samples from 2002- 2009 [177]. They found that the 2013-2017 samples consist of a new dominant lineage named MLL-F with a predominant MLG named Ms50 representing 90% of the MLL-F samples [181]. Consistent with the results of Harris-Shultz *et al*. [179, 180], Ms50 is a 'super-clone' and is the dominant MLG from the continental U.S., Mexico, Puerto Rico, and Haiti. This is different from their past study where samples from the U.S collected from 2007-2009 were only MLL-D. Of interest, in the 2013-2017 samples, sugarcane aphids that were MLL-D were only observed on sugarcane while sugarcane aphids that were assigned to MLL-F were found on *Sorghum* spp. and sugarcane.

4.3. Sugarcane aphid biotypes

Only one study currently addresses sugarcane aphid biotypes in the U.S. In this study, two host-specific sugarcane aphid biotypes have been identified in the U.S. [182]. One biotype was collected on sugarcane near Bell Grade, Florida and belonged to MLL-D whereas the other biotype included a sample that was collected near Beaumont, Texas on sorghum as well as a sample collected from Columbus grass from Florida; these last two samples were identical, using a set of SSR markers, and both samples belonged to the MLL-F lineage. When the MLL-D genotype from sugarcane and the MLL-F genotype from sorghum were phenotyped using host plant differentials such as a resistant sorghum AG1201, a susceptible sorghum KS 585, Johnsongrass and Columbus grass, the intrinsic rate of increase was significantly higher for the MLL-F genotype on sorghum, Johnsongrass, and Columbus grass, than the MLL-D genotype on these hosts. The primary host for the MLL-D genotype was sugarcane and, to a lesser degree, Johnsongrass.

4.4. Species debate

The taxonomic name of the sugarcane aphid has changed with time and was formerly *Aphis sacchari* Zehntner, *Longiunguis sacchari* (Zehntner), and *Melanaphis pyrarius* (Passerini) [167]. Blackman and Eastop [183] suggested that *Melanaphis* aphids found feeding on sorghum or sugarcane were distinct taxa and should be referred to as *M. sorghi* or *M. sacchari*, respectively. In contrast, Remaudiere and Remaudiere (1997) [184] considered *M. sorghi* as a synonym for *M. sacchari*. Nibouche *et al.* [177] performed genotyping of sugarcane aphids collected worldwide using SSR markers and by sequencing the COI fragment and found no molecular evidence for the separation of sugarcane aphids into *M. sacchari* and *M. sorghi.*

4.5. Sugarcane aphid management

4.5.1. Insecticide use

Insecticidal seed treatments are used to protect sorghum from sugarcane aphids and provide 3-4 weeks of protection. The products Cruiser (thiamethoxam), Poncho (clothianidin), Nipsit Inside (clothianidin), and Gaucho (imidacloprid) had significantly less sugarcane aphids than the untreated control until 33 DAP [185].

Sorghum fields should be scouted weekly for sugarcane aphids and then twice weekly once aphids are detected [186]. For sorghum, the use of

insecticides is recommended at 50-125 aphids per leaf [187]. The insecticides Transform (Dow AgroSciences, Indianapolis, IN, 50% sulfoxaflor), Sivanto (Bayer CropScience, Leverkusen, Germany, 17.09% flupyradifurone), and Centric (Syngenta, 40% thiamethoxam) reduced sugarcane aphid numbers at 14 days after treatment (DAT) [188]. Currently two products, Transform and Sivanto, are used in the U.S. to control the sugarcane aphid on sorghum for up to 21 DAT with minimal impact on natural enemies [5]. Both chemicals penetrate leaves through translaminar movement [166]. Additionally, in Mexico, foliar-applied imidacloprid is used for grain sorghum [5].

4.5.2. Cultural practices

Elimination of volunteer plants and the destruction of weedy hosts could aid in the reduction of the sugarcane aphid population during the non-growing season [155]. This is because sugarcane aphid overwinters on Johnsongrass and volunteer sorghum in southern regions and more northern locations are infested by winged aphids carried from southern regions [166]. Furthermore, mulching with rice (*Oryza sativa* L.) or wheat straw is effective in reducing colonization of aphids [155].

Planting as early as agronomically possible is recommended to allow plant growth and maturity before aphids arrive on field crops [189, 166]. One may need to consider latitude with these recommendations. In Tifton, GA a 3-year planting date study was conducted, and 23 sweet sorghum cultivars were planted each year in April, May, and June with three replicates [190]. For all three plantings and for all three years the sugarcane aphid peak occurred on July 13-15 and the sugarcane aphid population crash, due to an unknown fungal entomopathogen, occurred on July 21-29. Plant damage was less in the June planting as compared to the April and May plantings. Furthermore, mean juice Brix (an estimate of sugar concentration) was higher in the June planting as compared to the earlier planting dates as the June planting reaped the benefits of the aphid population crash in the adjacent plots. Thus, early planting is not recommended in Tifton, GA but planting dates can be staggered to take advantage of fungal entomopathogens.

4.5.3. Host resistance and genetic mapping

Host resistance can have a large impact on sugarcane aphid population dynamics [186]. Host resistance to the predominant U.S. sugarcane aphid genotype has been identified from sorghum lines Tx2783 [90], 0L2042, and SP7715 [171]. 0L2042 and SP7715 are owned and were developed by Chromatin Inc. (Chicago, IL, USA). Tx2783, originally released in 1984, has resistance to greenbug biotypes C and E, and sugarcane aphids in Botswana and Zimbabwe in the mid-1980s [90, 159]. Armstrong *et al*. (2015) found that it also has resistance to U.S. sugarcane aphid, with expression of tolerance and antibiosis. Tx2783 has a complicated pedigree but includes Capbam, of Russian origin, and SC110, which has sugarcane aphid resistance [161]. Additionally, lines B11055, SC170, Ent62/SADC, R.11143, R.11259 and (SV1*Sima/S23250)-LG15 as well as hybrids A11055/RTx436 and A11055/RTx437 displayed sugarcane aphid resistance [161, 191, 170]. Furthermore, a set of forage and grain sorghums were evaluated for sugarcane aphid resistance and many were highly resistant [191, 159].

The commercial hybrid DKS 37-07 which is resistant to greenbug biotypes C and E is also resistant to the sugarcane aphid [170]. It has been planted widely since the sugarcane aphid invasion, as it possesses good yield potential and has lower aphid population densities when sugarcane aphids are present [186]. A list of commercially available sorghum hybrids that are tolerant to the sugarcane aphid has been compiled and consists of hybrids that have been confirmed to be tolerant by university or federal researchers [192]. Furthermore, international research examining sugarcane aphid resistance in sorghum lines and hybrids and the mechanism of resistance has been compiled [155].

Mapping and identification of sugarcane aphid resistance genes has been limited. A dominant gene named Resistance to *Melanaphis sacchari* (*RMES1*) was identified from grain sorghum variety Henong 16 and was mapped to the short arm of chromosome 6 using a BTx623 × Henong16 population [193]. The *RMES1* locus was delimited to a region of about 126 kb which contains five predicted genes of which three are leucine-rich repeat containing proteins.

4.5.4. Natural enemies

Sugarcane aphid infestations on sorghum recruited natural enemies known to occur on other cereals,

including Coccinellidae, Syrphidae, Chrysophidae, Hemerobiidae, and Anthocoridae [168, 5, 169, 186]. Additionally, the predominant parasitoid that used sugarcane aphid as a host was *Aphelinus* sp. *varipes* group (Hymenoptera: Aphelinidae), while *Lysiphlebus testaceipes* (Cresson) (Hymenoptera: Braconidae) was rarely seen [5, 168]. These natural enemies were not able to decrease the aphid populations below economic threshold. Of interest, biological control was improved significantly on a resistant sorghum hybrid as compared to a susceptible hybrid [186].

Aphid "crashes" i.e., when aphids build to extremely large populations and then suddenly all die, have been observed for the sugarcane aphid. Aphid cadavers on sorghum leaves were collected from two locations in Texas and three locations in Georgia [194] and the sporulating fungus was identified as *Lecanicillium lecanii* (Zimmerman) Zare & Games, a natural pathogen of aphids and scales. It is thought that the epizootics of the fungus originates from fungal spores residing in the crop soil that are splashed onto the crop foliage [195]. Successful infection and epizootic initiation require a temperature between 18-31 °C (temperature is isolate dependent) and *V. lecanii* spores require the humidity to be near saturation in the microhabitat for successful germination [195].

Entomopathogenic fungi infecting sugarcane aphids in Mexico have also been identified [196]. The species associated with sugarcane aphids collected from South of Tamaulipas were *Lecanicillium longisporum*, *Beauveria bassiana*, and *Isaria javanica*. In Guanajuato, the species associated with the sugarcane aphid were *L. longisporum* and *B. bassiana*. Commercial formulations of entomopathogens exist but often the high levels of mortality seen for aphids in lab bioassays are frequently not seen when entomopathogens are applied to field environments [197].

5. Lessons learned from past invasions for the current sugarcane aphid invasion

5.1. A limited number of agriculturally important aphid genotypes have been dispersed by long range aerial dispersal

Invasive aphids with genotypes that are superior as agricultural pests have successfully spread throughout the U.S. and neighboring countries. It is believed that prevailing wind currents moved the RWA from Mexico to Texas and once there, these winds were responsible for dispersing the aphid to the other U.S. states [108]. Similarly, greenbug biotype C (discovered in 1968) that is warm-temperature-tolerant and feeds on sorghum and wheat, spread to Arizona, Colorado, Kansas, Nebraska, Oklahoma, South Dakota, and Texas in a single year [25]. Likewise, the sugarcane aphid first appeared feeding on grain sorghum near Beaumont, TX in 2013 and by 2019 reached 25 states and thus covers all sorghum-production regions of the U.S. and Mexico [158, 159].

Indeed, aphids are among the fastest colonizers in the animal kingdom [103]. Wellings [198] used data from RWA and the yellow clover aphid [*Therioaphis trifolii* (Monell)] and found the colonization front moved at a rate of 229 and 235 km/year, respectively. They further suggested that the rate of colonization of holocyclic species could be up to an order of magnitude less than anholocyclic species. Generally, newly colonized areas of RWA often experience high plant yield losses for the first few years, after which the RWA becomes a sporadic pest [103]. It is thought that some type of ecological balance occurs after a few years.

Another theme seen with invasive cereal aphids is that a few 'superclones' or their derivatives cause economic problems. Despite many biotypes of the RWA being found in the U.S., only RWA1 and RWA2 were economically important and are also genetically similar [131, 132]. Similarly, despite many biotypes of the greenbug being identified in the U.S. only biotype C, E, I, and K have economic importance and this set of biotypes are also genetically similar [54]. Likewise, one major superclone of the sugarcane aphid has spread throughout the U.S. feeding primarily on sorghum and Johnsongrass [179-181].

5.2. The sugarcane aphid may develop insecticide resistance

Pesticide resistance has been documented for 27 species of aphids [199]. Pesticide resistance in insects occurs by the evolution of two mechanisms. The first mechanism is the enhanced production of metabolic enzymes which sequester or detoxify the pesticide [200]. These detoxifying enzymes

include esterases, glutathione δ-transferases, and cytochrome P450 monooxygenases, and the enzyme production is enhanced by duplication or amplification of the corresponding genes or through mutations in the regulating loci of these genes [201, 200]. Alternatively, mutations can occur in the genes encoding the detoxifying enzymes which cause an enhanced ability to metabolize the pesticide [200]. The second mechanism for insect pesticide resistance is by the mutation of the target protein of the pesticide allowing the insect to be more tolerant of the pesticide or by the amplification of the insecticidal target gene [200].

Sivanto and Transform are used to control the sugarcane aphid and are in Insecticide Resistance Action Committee (IRAC) subgroup 4D (butenolides) and 4C (sulfoximines) insecticides, respectively [5]. Sivanto has a longer residual than Transform [202, 166] and both pesticides now have a U.S. Environmental Protection Agency Fungicide and Rodenticide Act (FIFRA) full Section 3 federal registration. Furthermore, all the seed treatments used for sugarcane aphid control are in IRAC subgroup 4A (neonicotinoids).

The application of two insecticides in the same IRAC group used over a wide area yearly (for more northern states) or continually (for more southern states) may select for aphids with pesticide resistance. Both Sivanto and Transform act as post-synaptic nicotinic acetylcholine receptor agonists [203]. Multiple applications of these two chemicals over large areas with a single clonal genotype present may cause the selection of aphid genotypes with resistance to Sivanto or Transform. For greenbug, biotype C resistance to organophosphorous insecticides was first seen in 1973, five years after the 1968 outbreak [25, 38].

5.3. Host resistance: use of hybrids with single-gene resistance may be risky

The use of mainly one or two sources of resistance to the major biotype over a large geographical area can cause a loss of host resistance when a new biotype develops. In 1980 at least 90% of the sorghum acreage in the U.S. was planted with hybrids with greenbug biotype C resistance derived from 'Shallu Grain' and 'KS30' [89]. These lines were thought to share *S. virgatum* as a source of resistance and the mechanism of resistance was tolerance [85]. This selection pressure may have resulted in the development of highly related biotypes *via* the mutation process. Failure to use multiple resistance genes often shortens the lifespan of host resistance. Starks *et al*. [89] expressed the outcome of the failure to use multiple resistance genes when they stated, "Years of painstaking research have been nullified by the development of the new biotype, and effective commercial use of resistance that has been widespread in sorghum and barley has been largely eliminated."

The mechanism of host resistance is thought to impact the evolution of pathogen/insect resistance to the host. Cultivars with tolerance resistance are often more stable than cultivars with antibiosis resistance [204]. Inheritance of aphid resistance can be monogenic or polygenic, and most host resistance genes that have major qualitative effects encode a class of proteins that are nucleotide binding and contain leucine-rich repeats [3]. Resistance genes in plants to aphids are often located in the same plant chromosomal region which is also the case for pathogen resistance genes [3]. The use of monogenic resistance genes in cultivars is not recommended, as for some aphid species with time and wide deployment, there has been a loss of host resistance when the host contains a major resistance gene [3]. For more durable resistance, cultivars should have multiple resistance genes in a single cultivar, which is often called gene pyramiding, or cultivars should have resistance that is controlled by multiple quantitative trait loci [3, 205].

5.4. Agronomically important biotypes may change with time

From the RWA and greenbug invasions of the U.S. the agronomic biotypes are genetically related, and the biotypes change with time. The RWA1 and RWA2 are related as are greenbug biotypes C, E, I, and K. Thus, with one predominant genotype in the U.S. of the sugarcane aphid, one can expect that mutation and selection will be acting upon this clonal population resulting in new biotypes.

5.5. Classical biological control is not recommended

Classical biological control, the introduction of exotic natural enemies to suppress the populations of an invasive pest species, is not recommended for sugarcane aphid management as it is uncertain if the classical biological control efforts against

RWA and greenbug contributed substantially to their control [17]. Although the classical biological control approach has easy marketability with the public, as it avoids pesticide use and suggests that lasting agricultural benefits are possible, there can be undesirable side effects to the release of exotic natural enemies [206]. Colares *et al*. [169] noted that the preadaptations of many resident predators and parasitoids to utilize a new prey/host may require a period of evolutionary adaptation to be biological control agents. Ecosystems can resist and assimilate invasive pests [206]. Indeed, natural enemies including Coccinellidae, Syrphidae, Chrysophidae, and Anthocoridae were all recruited to sorghum that was infested with sugarcane aphids [186].

6. Conclusion

From the previous aphid invasions of greenbug and RWA in the U.S., several themes become apparent that directly apply to the recent sugarcane aphid infestation. 'Superclones' (and in some cases their mutants) of all three aphid species that thrive in agricultural landscapes spread by aerial dispersal in the U.S. causing devastating impacts on many cereal crops. Furthermore, the use of multiple insecticides with varying modes of action and the use of hybrids with multiple resistance genes may prevent or increase the time for new biotypes of the sugarcane aphid to develop. Lastly since classical biological control had limited success for the greenbug or RWA it is not recommended for sugarcane aphid management.

ACKNOWLEDGEMENTS

The authors would like to thank Mike Brewer (Texas A&M University) for inspiring us to write this review article. We also thank Duncan McClusky, University of Georgia librarian, for obtaining many of the hard-to-find references and Joseph Knoll (USDA-ARS) and Somashekhar Punnuri (Fort Valley State) for reviewing this article.

DISCLAIMER

Mention of trade names or commercial products in this article is solely for the purpose of providing specific information and does not imply recommendation or endorsement by the U.S. Department of Agriculture. USDA is an equal opportunity provider and employer.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

REFERENCES

1. Farquharson, K. L. 2017, Plant Cell, 29, 2309-2310.
2. Smith, C. M. and Boyko, E. V. 2007, Entomol. Exp. Appl., 122,1-16.
3. Dogimont, C., Bendahmane, A., Chovelon, V. and Boissot, N. 2010, C. R. Biol., 333, 566-573.
4. Elliott, N. C., Kieckhefer, R. W., Lee, J.-H. and French, B. W. 1998, Landscape Ecol., 14, 239-252.
5. Bowling, R. D., Brewer, M. J., Kerns, D. L., Gordy, J., Seiter, N., Elliott, N. E., Buntin, G. D., Way, M. O., Royer, T. A., Biles, S. and Maxson, E. 2016a, J. Integr. Pest Manag., 7, 12.
6. Michels, G. J. and Burd, J. D. 2007, IPM Case Studies: Sorghum. In: H.F. van Emden and R. Harrington (Eds.) Aphids as Crop Pests. CAB International, Wallingford, United Kingdom, 627-637.
7. Quisenberry, S. S. and Peairs, F. B. Eds. 1998, Proceedings of Response Model for an Introduced Pest-The Russian Wheat Aphid. Lanham, MD: Entomol. Soc. Am.
8. Royer, T. A., Pendleton, B. R., Elliott, N. C. and Giles, K. L. 2015, J. Integr. Pest Manag., 6, 19.
9. Anstead, J. A., Burd, J. D. and Shufran, K. A. 2002, Bull. Entomol. Res., 92, 17-24.
10. Puterka, G. J. and Peters, D. C. 1990, Sexual reproduction and inheritance of virulence in the greenbug *Schizaphis graminum* (Rondani), *In* R. K. Campbell and R. D. Eikenbary (Eds.), Aphid-plant genotype interactions. Elsevier, Amsterdam, 289-318.
11. Diehl, S. R. and Bush, G. L. 1984, Annu. Rev. Entomol., 29, 471-504.
12. Bostein, D., White, R. L., Skolnick, M. and Davis, R. W. 1980, Am. J. Hum. Genet., 32, 314-331.
13. Grover, A. and Sharma, P. C. 2016, Crit. Rev. Biotechnol., 36, 290-302.
14. Vos, P., Hogers, R., Bleeker, M., Reijans, M., van de Lee, T., Hornes, M., Friters, A., Pot, J., Paleman, J., Kuiper, M. and Zabeau, M. 1995, Nuclei Acids Res., 23, 4407-4414.

15. Williams, J. G. K., Kubeik, A. R., Livak, K. J., Rafalski, J. A. and Tingey, S. V. 1990, Nucleic Acids Res., 18, 6531-6535.
16. Robarts, D. W. and Wolfe, A. D. 2014, Appl. Plant Sci., 2, apps.1400017.
17. Brewer, M. J. and Elliott, N. C. 2004, Annu. Rev. Entomol., 49, 219-242.
18. Weng, Y., Perumal, A., Burd, J. D. and Rudd, J. C. 2010, J. Econ. Entomol., 103, 1454-1463.
19. Hunter, S. J. 1909, Univ. Kansas Bull., 9, 1-163.
20. Webster, F. M. and Phillips, W. J. 1912, U.S. Dep. Agric. Bur. Entomol. Bull., 110, 153.
21. Irwin, M. E. and Thresh, J. M. 1988, Philos. T. Roy. Soc. B 321, 421-446.
22. Starks, K. J. and Mayo, Z. B. 1985, International Crops Research Institute for the Semi-Arid Tropics. Proceedings of the International Sorghum Entomology Workshop, 15-21 July 1984, Texas A&M University, College Station, TX USA
23. Starks, K. J. and Burton, R. L. 1977, Preventing greenbug outbreaks. Rep. No. 309, USDA Sci. Educ. Admin. Leaflet. Washington, D.C.
24. Webster, J. A. 1995, Economic impact of the greenbug in the western United States: 1992-1993. Rep. Vol. Publ. No. 155, Great Plains Agric. Counc., Stillwater, Okla.
25. Harvey, T. L. and Hackerott, H. L. 1969, J. Econ. Entomol., 62, 776-779.
26. Kindler, S. D., Elliott, N. C., Giles, K. L., Royer, T. A., Fuentes-Granados, R. and Tao, F. 2002, J. Econ. Entomol., 95, 89-95.
27. Giles, K. L., Jones, D. B., Royer, T. A., Elliott, N. C. and Kindler, S. D. 2003, J. Econ. Entomol., 96, 975-982.
28. Sandstrom, J., Telang, A. and Moran, N. A. 2000, J. Insect Physiol., 46, 33-40.
29. Ma, R., Reese, J. C., Black, W. C. and Bramel-Cox, P. 1990, J. Insect Physiol. 36, 507-512.
30. Daniels, N. E. and Toler, R. W. 1969, Plant Dis. Rep., 53, 59-61.
31. Wallin, J. R. and Loonan, D. V. 1971, Phytopath., 61, 1068-1070.
32. Teetes, G. L., Rosenow, D. T., Frederiksen, R. A. and Johnson, J. W. 1973, The predisposing influence of greenbugs on charcoal rot of sorghum. Texas Agricultural Experiment Station, College Station, Texas USA. PR-3173, 6.
33. Burton, R. L., Morrison, R. D., Starks, K. J. and Simmon, D. D. 1985, J. Econ. Entomol., 78, 395-401.
34. Royer, T. A., Giles, K. L., Kindler, S. D. and Elliott, N. C. 2004, Cereal aphid expert system and *Glance 'n Go* sampling for greenbugs: Questions and answers. CR-7191. Oklahoma Cooperative Extension Service, Stillwater, OK.
35. Porter, D. R., Burd, J. D., Shufran, K. A., Webster, J. A. and Teetes, G. L. 1997, J. Econ. Entomol., 90, 1055-1065.
36. Wood, E. A. 1961, J. Econ. Entomol., 54, 1171-1173.
37. Bowling, R., Wilde, G., Harvey, T., Sloderbeck, P., Bell, K. O., Morrison, W. P. and Brooks, H. L. 1994, J. Econ. Entomol., 87, 1696-1700.
38. Teetes, G. L., Schaefer, C. A., Gipson, J. R., McIntyre, R. C. and Latham, E. E. 1975, J. Econ. Entomol., 68, 214-216.
39. Porter, K. B., Peterson, G. L. and Vise, O. 1982, Crop Sci., 22, 847-850.
40. Kindler, S. D., Spomer, S. M. and Harvey, T. L. 1984, J. Kansas Entomol. Soc., 57, 155-158.
41. Dumas, B. A. and Mueller, A. J. 1986, J. Entomol. Sci., 21, 38-42.
42. Kerns, D. L., Peters, D. C. and Puterka, G. J. 1987, Southwest. Entomol., 12, 237-243.
43. Puterka, G. J., Peters, D. C., Kerns, D. L., Slosser, J. E., Bush, L., Worrall, D. W. and McNew, R. W. 1988, J. Econ. Entomol., 81, 1754-1759.
44. Kindler, S. D. and Spomer, S. M. 1986, Environ. Entomol., 15, 567-572.
45. Harvey, T. L., Kofoid, K. D., Martin, T. J. and Sloderbeck, P. E. 1991, Crop Sci., 31, 1689-1691.
46. Beregovoy, V. H. and Peters, D. C. 1994, J. Kansas Entomol., Soc., 67, 248-252.
47. Harvey, T. L., Wilde, G. E. and Kofoid, K. D. 1997, Crop Sci., 37, 989-991.
48. Weng, Y., Perumal, A., Burd, J. D. and Rudd, J. C. 2010, J. Econ. Entomol., 103,1454-1463.

49. Burd, J. D. and Porter, D. R. 2006, J. Econ. Entomol., 99, 959-965.
50. Wu, Y. Q., Huang, Y., Porter, D. R., Tauer, C. G. and Hollaway, L. 2007, J. Econ. Entomol., 100, 1672-1678.
51. Zorrilla, H. 2019, Managing greenbug biotype and insecticide resistant changes. https://www.pioneer.com/us/agronomy/insecticide.html
52. Nuessly, G., Nagata, R., Burd, J., Hentz, M., Carroll, A. and Halbert, S. 2008, Environ. Entomol., 37, 586-91.
53. Armstrong, J. S., Mornhinweg, D. W., Payton, M. E. and Puterka, G. J. 2016, J. Econ. Entomol., 109, 434-438.
54. Zhu-Saltzman, K., Li, H., Klein, P. E., Gorena, R. L. and Saltzman, R. A. 2003, Agr. Forest Entomol., 5, 311-315.
55. Weng, Y., Azhaguvel, P., Michels, G. J. and Rudd, J. C. 2007, Insect Mol. Biol., 16, 613-622.
56. Kharrat, I., Bouktila, D., Mezghani-Khemakhem, M., Makni, H. and Makni, M. 2012, Rev. Colomb. Entomol., 38, 87-90.
57. Shufran, K. A., Burd, J. D., Anstead, J. A. and Lushai, G. 2000, Insect Mol. Biol., 9, 179-184.
58. Pike, K. S. 1978, J. Econ. Entomol., 71, 827-832.
59. Sloderbeck, P., Witt, M. and Buschman, L. 1995, Report of Progress- Kansas Agricultural Experiment Station, 739, 18-22.
60. Burton, R. L. and Krenzer, E. G. 1985, J. Econ. Entomol., 78, 390-394.
61. Burton, R. L., Jones, O. R., Burd, J. D., Wicks, G. A. and Krenzer, E. C. 1987, J. Econ. Entomol., 80, 792-798.
62. Harvey, T. L., Hackerott, H. L. and Martin, T. J. 1982, J. Econ. Entomol., 75, 36-39.
63. Hesler, L. S., Riedell, W. E., Langham, M. A. C. and Osborne, S. L. 2005, J. Econ. Entomol., 98, 2020-2027.
64. Arnold, D. C. 1981, J. Kansas Entomol. Soc., 54, 571-577.
65. Ismail, E. A., Giles, K. L., Coburn, L., Royer, T. A., Hunter, R. M., Verchot, J., Horn, G. W., Krenzer, E. G., Peeper, T. F., Payton, M. E., Michels, G. J. and Owings, D. A. 2003, Southwest. Entomol., 28, 121-130.
66. Curtis, B. C., Schlehuber, A. M. and Wood, E. A. 1960, Agron. J., 52, 599-602.
67. Tyler, J. M., Webster, J. A. and Merkle, O. G. 1987, Crop Sci., 27, 526-527.
68. Girma, M., Kofoid, K. D. and Reese, J. C. 1998, J. Kansas Entomol. Soc., 71, 108-115.
69. Reese, J. C, Schwenke, J. R., Lamont, P. S. and Zehr, D. D. 1994, J. Agric. Entomol., 11, 255-270.
70. Sebesta, E. E. and Wood, E. A. 1978, Agron. Abstr. Am. Soc. Agron. 61-62.
71. Hollenhorst, M. M. and Joppa, L. R. 1983, Crop Sci., 23, 91-93.
72. Porter, K. B., Worrall, W. D., Gardenhire, J. H., Gilmore, E. C., McDaniel, M. E. and Tuleen, N. A. 1987, Crop Sci., 27, 818-819.
73. Wood, E. A., Sebesta, E. E. and Starks, K. J. 1974, Environ. Entomol., 3, 720-721.
74. Joppa, L. R., Timian, R. G. and Williams, N. D. 1980, Crop Sci., 20, 343-344.
75. Joppa, L. R. and Williams, N. D. 1982, Crop Sci., 22, 901-902.
76. Porter, D. R., Webster, J. A. and Friebe, B. 1994, Crop Sci., 34, 625-628.
77. Martin, T. J., Harvey, T. L. and Hatchett, J. H. 1982, Crop Sci., 22, 1089.
78. Tyler, J. M., Webster, J. A. and Smith, E. L. 1985, Crop Sci., 25, 686-688
79. Weng, Y., Li, W., Devkota, R. N. and Rudd, J. C. 2005, Theor. Appl. Genet., 110, 462-469.
80. Gardenhire, J. H. and Chada, H. L. 1961, Crop Sci., 1, 349-352.
81. Merkle, O. G., Webster, J. A. and Morgan, G. H. 1987, Crop Sci., 27, 241-243.
82. Mornhinweg, D. W., Edwards, L. H., Smith, E. L., Morgan, G. H., Webster, J. A., Porter, D. R. and Carver, B. F. 2004, Crop Sci., 44, 2263.
83. Anstead, J. A., Burd, J. D. and Shufran, K. A. 2003, Environ. Entomol., 32, 662-667.
84. Porter, D. R. and Mornhinweg, D. W. 2004, Crop Sci., 44, 1245-1247.
85. Hackerott, H. L., Harvey, T. L. and Ross, W. M. 1969, Crop Sci., 9, 656-658.
86. Hackerott, H. L., Harvey, T. L. and Ross, W. M. 1972, Crop Sci., 12, 719.
87. Follmer, G. and Maunder, B. 1976, Sorghum Newsl., 19, 6-7.
88. Dixon, A. G., Bramel-Cox, P. J. and Harvey, T. L. 1991, Theor. Appl. Genet., 81, 105-110.

89. Starks, K. J., Burton, R. L. and Merkle, O. G. 1983, J. Econ. Entomol., 76, 877-880.
90. Peterson, G. C., Johnson, J. W., Teetes, G. L. and Rosenow, D. T. 1984, Crop Sci., 24, 390.
91. Andrews, D. J., Bramel-Cox, P. J. and Wilde, G. E. 1993, Crop Sci., 33, 198-199.
92. Brooks, H. L. 1996. Greenbug ratings on commercial sorghum hybrids. Kansas State University Extension Entomology. HB No. 147.
93. Walker, A. L., Bottrell, D. G. and Cate, J. R. 1973, Ann. Entomol. Soc. Am., 66, 173-176.
94. Feng, M. G., Johnson, J. B. and Kish, L. P. 1990, Environ. Entomol., 19, 1534-1542.
95. Hodek, I. 1970, Bioscience, 20, 543-552.
96. Kring, T. J., Gilstrap, F. E. and Michels, G. J. Jr. 1985, J. Econ. Entomol., 78, 269-273.
97. Rice, M. E. and Wilde, G. E. 1988, Environ. Entomol., 17, 836-841.
98. Fernandes, O. A. Wright, R. J. and Mayo, Z. B. 1998, J. Econ. Entomol., 91, 1315-1319.
99. Jones, D. B. 2001, Natural enemy thresholds for greenbug, *Schizaphis graminum* Rondani, on winter wheat. M.S. thesis, Oklahoma State University, Stillwater, OK.
100. Jackson, H. B., Rogers, C. E. and Eikenbary, R. D. 1971, J. Econ. Entomol., 64, 1435-1438.
101. Grossheim, N. A. 1915, Rev. Appl. Entomol. Ser. A, 3, 307-308.
102. Robinson, J. 1994. Identification and characterization of resistance to the Russian wheat aphid in small-grain cereals: Investigations at CIMMYT, 1990-92. CIMMYT Research Report No.3. Mexico, D.E: CIMMYT.
103. Halbert, S. E. and Stoetzel, M. B. 1998. Historical overview of the Russian wheat aphid (Homoptera: Aphididae),. *In* S.S. Quisenberry and F.B. Peairs [Eds.], A response model for an introduced pest-the Russian wheat aphid. Thomas Say Publications in Entomology. Entomological Society of America, Lanham, MD, 13-30.
104. Kindler, S. D. and Springer, T. L. 1989, J. Econ. Entomol., 82, 1358-1362.
105. Armstrong, J. S., Porter, M. R. and Peairs, F. B. 1991, J. Econ. Entomol., 84, 1691-1694.
106. Walters, M. C., Penn, F., Du Toit, F., Botha, T. C., Aalbersberg, K., Hewitt, P. H. and Broodryk, S. W. 1980, The Russian wheat aphid, farming in South Africa. Leafl. Series, Wheat C3, Government Printer, 6. Pflanzenschutzdienstes, South Africa.
107. Gilchrist, L. I., Rodriguez, R. and Burnett, P. A. 1984, The extent of freestate streak and *Diuraphis noxia* in Mexico, *In* P. A. Burnett and E. Cuellar [Eds.], Barley yellow dwarf. Proceedings of the Workshop, 6-8 December 1983, Mexico City, Mexico. CIMMYT, El Batan, Mexico, 157-163.
108. Stoetzel, M. B. 1987. J. Econ. Entomol., 80, 696-704.
109. Zerene, Z. M., Caglevic, D. M. and Ramirez, A. I. 1988, Agric. Tec., (Santiago) 48, 60-61.
110. Morrison W. P and Peairs, F. B.1998, Response model concept and economic impact. In: Quisenberry S. S, Peairs F. B, (Eds). Response model for an introduced pest-the Russian wheat aphid. Lanham, MD: Entomological Society of America.
111. Brewer, M. J., Nelson, D. J., Ahern, R. G., Donahue, J. D. and Prokrym, D. R. 2001, Environ. Entomol., 30, 578-88.
112. Burd, J. D. and Elliott, N. C. 1996, J. Econ. Entomol., 89, 1332-1337.
113. Hewitt, P. H., van Niekerk, G. J. J., Walters, M. C., Kriel, C. F. and Fouche, A. 1984. Aspects of the ecology of the Russian wheat aphid, *Diuraphis noxia*, in the Bloemfontein district. I. The colonization and infestation of sown wheat, identification of summer hosts and cause of infestation symptoms, *In* M.C. Walters [ed.], Progress in Russian wheat aphid (*Diuraphis noxia* Mordv.) research in the Republic of South Africa. Technical Communication No. 191, Department of Agriculture, Republic of South Africa, 3-13.
114. Burd, J. D. and Burton, R. L. 1992, J. Econ. Entomol., 85, 2017-2022.
115. Puterka, G. J., Hammon, R. W., Burd, J. D., Peairs, F. B., Randolph, T. L. and Cooper, W. R. 2012, J. Econ. Entomol., 105, 1057-1068.
116. Bush, L., Slosser, J. E. and Worrall, W. D. 1989, J. Econ. Entomol., 82, 466-471.
117. Kiriac, I., Gruber, F., Poprawski, T., Halbert, S. and Elberson, L. 1990, Proc. Entomol. Soc. Wash., 92, 544-547.
118. Bergman, D. K., Dillwith, J. W., Campbell, R. K. and Eikenbary, R. D. 1990, Southwest. Entomol., 15, 91-100.

119. Shufran, K. A., Burd, J. D. and Webster, J. A. 1997, J. Econ. Entomol., 90, 1684-1689.
120. Quick, J. S., Stromberger, J. A., Clayshulte, S., Clifford, B., Johnson, J. J., Peairs, F. B., Rudolph, J. B. and Lorenz, K. 2001, Crop Sci., 41, 1362-1363.
121. Haley, S. D., Peairs, F. B., Walker, C. B., Rudolph, J. B. and Randolph, R. L. 2004, Crop Sci., 44, 1589-1592.
122. Marais, G. F., Horn, M. and Du Toit, F. 1994, Plant Breed., 113, 265-271.
123. Zhao, C., Cui, F., Wang, X., Shan, S., Li, X., Bao, Y. and Wang H. 2012, Field Crops Res., 127, 79-84.
124. Burd, J. D., Porter, D. R., Puterka, G. J., Haley, S. D. and Peairs, F. 2006, J. Econ. Entomol., 99, 1862-1866.
125. Puterka, G. J., Burd, J. D., Porter, D., Shufran, K., Baker, C., Bowling, B. and Patrick, C. 2007, J. Econ. Entomol., 100, 1679-1684.
126. Weiland, A. A., Peairs, F. B., Randolph, T. L., Rudolph, J. B., Haley, S. D. and Puterka, G. J. 2008, J. Econ. Entomol., 101, 569-574.
127. Puterka, G. J., Nicholson, S. J., Brown, M. J., Cooper, W. R., Peairs, F. B. and Randolph, T. L. 2014, J. Econ. Entomol., 107, 1274-1283.
128. Puterka, G. J., Black, W. C., Steiner, W. M. and Burton, R. L. 1993, Heredity, 70, 604-618.
129. Robinson, J., Fisher, M. and Hoisington, D. 1993, Southwest. Entomol., 18, 121-127.
130. Shufran, K. A. and Payton, T. L. 2009, J. Econ. Entomol., 102, 440-445.
131. Liu, X., Marshall, J. L., Stary, P., Edwards, O., Puterka, G., Dolatti, L., El Bouhssini, M., Malinga, J., Lage, J. and Smith, C. M. 2010, J. Econ. Entomol., 103, 958-965.
132. Shufran, K. A., Kirkman, L. R. and Puterka, G. J. 2007, J. Kansas Entomol. Soc., 80, 319-326.
133. Kammerzell, K. J. and Johnson, G. D. 1991, Evaluation of different seeding dates to reduce Russian wheat aphid damage in winter wheat in Montana.. *In* G. D. Johnson (Ed)., Proc. 4th Russian What Aphid Workshop, 10-12 October 1990, Bozeman, Montana. Montana State University, Bozeman, Montana, 63-74.
134. Armstrong, S., Walker, C. B., Peairs, F. B. and Lister, R. 1992, The effect of planting date in eastern Colorado on Russian wheat aphid infestations in winter wheat. 109-115. In W. P. Morrison Ed., Proc. 5th Annu. Russian Wheat Aphid Conf., 26-28 January 1992, Fort Worth, Texas. Great Plains Agric. Counc. Publ. 142.
135. Hammon, R. W., Judson, F. M., Sanford, D. and Peairs, F. B. 1991, Date of planting and Russian wheat aphid populations. In G. D. Johnson Ed., Proc. 4th Russian Wheat Aphid Workshop, 10-12 October 1990, Bozeman, Montana. Montana State University, Bozeman, Montana, 49-53.
136. Butts, R. A. 1992, The influence of seeding dates on the impact of fall infestations of Russian wheat aphid in winter wheat. pp. 120-122. In W. P Morrison Ed., Proc. 5th Annu. Russian Wheat Aphid Conf., 26-28 January 1992, Fort Worth, Texas. Great Plains Agric. Counc. Publ. 142.
137. Hammon, R. W., Pearson, C. H. and Peairs, F.B. 1996, J. Kansas Entomol. Soc., 69, 302-309.
138. Archer, T. L. Bynum, E. D., Onken, A. B. and Wendt, C. W. 1995, Crop Prot. 14, 165-169.
139. Mornhinweg, D. W., Brewer, M. J. and Porter, D. R. 2006, Crop Sci., 46, 36-42.
140. Mornhinweg, D. W., Porter, D. R. and Webster, J. A. 1995, Crop Sci., 35, 602.
141. Mornhinweg, D. W., Porter, D. R. and Webster, J. A. 1999, Crop Sci., 39, 882-883.
142. Smith, C. M., Schotzko, D., Zemetra, R. S., Souza, E. J. and Schroeder-Teeter, S. 1991, J. Econ. Entomol., 84, 328-332.
143. Souza, E. J. 1998, Host plant resistance to the Russian Wheat Aphid (Homoptera: Aphididae) in wheat and barley, *In* S.S. Quisenberry and F.B. Peairs [Eds.], A response model for an introduced pest-the Russian wheat aphid. Thomas Say Publications in Entomology. Entomological Society of America, Lanham, MD, 13-30.
144. Baker, C. A., Porter, D. R. and Webster, J. A. 1994, Crop Sci., 34, 1135-1136.
145. Quick, J. S., Ellis, G. E., Normann, R. M., Stromberger, J. A., Shanahan, J. F., Peairs, F. B., Rudolph, J. B. and Lorenz, K. 1996, Crop Sci., 36, 210.

146. Randolph, T. L., Peairs, F. B., Kroening, M. K., Armstrong, J. S., Hammon, R. W., Walker, C. B. and Quick, J. S. 2003, J. Econ. Entomol., 96, 352-360.
147. Quick, J. S., Nkongolo, K. K., Meyer, W., Peairs, F. B. and Weaver, B. 1991, Crop Sci., 31, 50-53.
148. Webster, J. A., Baker, C. A. and Porter, D. R. 1991, J. Econ. Entomol., 84, 669-673.
149. Puterka, G. J., Burd, J. D, Mornhinweg, D. W., Haley, S. D. and Peairs, F. B. 2006, J. Econ. Entomol., 99, 2151-2155.
150. Hopper, K. R., Coutinot, D., Chen, K., Kazmer, D. J. and Mercadier, G. 1998, Exploration for natural enemies to control *Diuraphis noxia* (Homoptera: Aphididae) in the United States. S. S. Quisenberry, and F. B. Peairs, Eds. Proceedings of Response Model for an Introduced Pest-The Russian Wheat Aphid. Lanham, MD: Entomol. Soc. Am.
151. Prokrym, D. R., Pike, K. S. and Nelson, D. J. 1998, Biological control of *Diuraphis noxia* (Homoptera: Aphididae): implementation and evaluation of natural enemies, pp. 183-208. *In* S. S. Quisenberry and F. B. Peairs [Eds.], Response model for an introduced pest-the Russian wheat aphid. Thomas Say Publications in Entomology: Proceedings. Entomological Society of America, Lanham, MD.
152. Elliott, N. C., Burd, J. D., Armstrong, J. S., Walker, C. B., Reed, D. K. and Peairs, F. B. 1995, Southwest. Entomol., 20, 125-129.
153. Clement, S. L., Lester, D. G., Wilson, A. D. and Pike, K. S. 1992, J. Econ. Entomol., 85, 583-588.
154. Clay, K. 1988, Ecology, 69, 10-16.
155. Singh, B. U., Padmaja, P. G. and Seetharama, N. 2004, Crop Prot., 23, 739-755.
156. David, S. K. and Sandhu, G. S. 1976, Entomol. Rec., 88, 28-29.
157. Villanueva, R. T., Brewer, M., Way, M. O., Biles, S., Sekula-Ortiz, D., Bynum, E., Swart, J., Crumley, C., Knutson, A., Porter, P., Parker, R., Odvody, G., Allen, C., Ragsdale, D., Rooney, W., Peterson, G., Kerns, D., Royer, T. and Armstrong, S. 2014, Sugarcane aphid: A new pest of sorghum. Texas A&M AgriLife Extension, Ento-035. (http://denton.agrilife.org/files/2013/08/ENTO-035-The-Sugarcane-Aphid-2014.pdf).
158. EDDMapS. 2019, Early detection & distribution mapping system. The University of Georgia-Center for Invasive Species and Ecosystem Health. Available online at http://www.eddmaps.org/; last accessed October 8, 2019.
159. Peterson, G. C., Armstrong, J. S., Pendleton, B. B., Stelter, M. and Brewer, M. J. 2018, J. Plant Regist., 12, 391-398
160. Rodriguez del Bosque, L. A. and Teran, A. 2015, Southwest. Entomol., 40, 433-434.
161. Armstrong, J. S., Rooney, W. L., Peterson, G. C., Villenueva, R. T., Brewer, M. J. and Sekula-Ortiz, D. 2015, J. Econ. Entomol., 108, 576-582.
162. Armstrong, J. S., Harris-Shultz, K. R., Ni, X., Wang, H., Knoll, J. E. and Anderson, W. F. 2019, Trends Entomol., 15, 1-14.
163. White, W. H., Reagan, T. E. and Hall, D. G. 2001, Florida Entomol., 84, 435-436.
164. Mead, F. W. 1978, Cooperative Plant Pest Report, 3, 475.
165. Denmark, H. A. 1988, Sugarcane aphids in Florida. Florida Department of Agriculture and Consumer Services, Division of Plant Industry, Entomology Circular, 302.
166. Michaud, J. P., Whitworth, R. J., Schwarting, H, McCornack, B. and Zukoff, S. 2016, Sorghum insect management. Kansas State University Research and Extension. MF742, Manhattan, KS, 12 p. (http://entomology.k-state.edu/extension/publications/).
167. van Rensburg, N. J. 1973. J. Entomol. Soc. S. Afr., 36, 293-298.
168. Brewer, M. J., Gordy, J. W., Kerns, D. L., Woolley, J. B., Rooney, W. L. and Bowling, R. D. 2017, J. Econ. Entomol., 110, 2109-2118.
169. Colares, F., Michaud, J. P., Bain, C. L. and Torres, J. B. 2015, Biol. Control, 90, 16-24.
170. Limaje, A., Hayes, C., Armstrong, J. S., Hoback, W., Zarrabi, A., Paudyal, S. and Burke, J. 2018, J. Entomol. Sci., 53, 230-241.
171. Armstrong, J. S., Paudyal, S., Limaje, A., Elliott, N. and Hoback, W. 2018, J. Entomol. Sci., 53, 478-485.
172. Pena-Martinez, R., Lilia Munoz-Viveros, A., Bujanos-Muniz, R., Luevano-Borroel, J., Tamayo-Mejia, F. and Cortez-Mondaca, E. 2016, Southwest. Entomol., 41, 127-131.

173. Brewer, M. J., Bowling, R., Michaud, J. P. and Jacobson, A. L. 2016, Sugarcane aphid-a new pest in North America. ENTO-056. Texas A&M AgriLife Extension Service, College Station, TX. (http://ccag.tamu.edu/sorghum-insect-pests/).
174. Schenck, S. and Lehrer, A. T. 2000, Plant Dis., 84, 1085-1088.
175. Wei, C., Hincapie, M., Larsen, N., Nuessly, G. and Rott, P. 2016, Dis. Notes, 100, 1798.
176. Espinoza Delgado, H. V., Kaye, C., Hincapie, M., Boukari, W., Wei, C., Fernandez, J. V., Mollov, D., Comstock, J. C. and Rott, P. 2016, Plant Dis., 100, 1027.
177. Nibouche, S., Fartek, B., Mississipi, S., Delatte, H., Reynaud, B. and Costet, L. 2014, PLoS ONE, 9, e106067.
178. Nibouche, S., Mississipi, S., Fartek, B., Delatte, H., Reynaud, B. and Costet, L. 2015, PLoS ONE, 10, e0143704.
179. Harris-Shultz, K., Ni, X., Wadl, P. A., Wang, X., Wang, H., Huang, F., Flanders, K., Seiter, N., Kerns, D., Meagher, R., Xue, Q., Reisig, D., Buntin, D., Cuevas, H. E., Brewer, M. J. and Yang, X. 2017, Crop Sci., 57, 2064-2072.
180. Harris-Shultz, K., Brewer, M. J., Wadl, P. A., Ni, X. and Wang, H. 2018, Crop Sci., 58, 2533-2541.
181. Nibouche, S., Costet, L., Holt, J. R., Jacobson, A., Pekarcik, A., Sadeyen, J., Armstrong, J. S., Peterson, G. C., McLaren, N. and Medina, R. F. 2018, PLoS ONE, 13, e0196124.
182. Paudyal S., Armstrong, J. S., Harris-Shultz, K. R., Wang, H., Giles, K. L., Rott, P. C. and Payton, M. E. 2019, Trends Entomol., 15, 47-58.
183. Blackman, R. L. and Eastop, V. F. 2006, Aphids on the world's herbaceous plants and shrubs. Chichester, UK: John Wiley & Sons Ltd.
184. Remaudiere, G. and Remaudiere, M. 1997, Catalogue of the world's Aphididae. Paris, France: INRA.
185. Jones, N., Brown, S., Williams, S., Emfinger, K. and Kerns, D. 2015, Arthropod Manag. Tests, 40, 1-2.
186. Szczepaniec, A. 2018, Crop Prot.,109, 72-79.
187. Bowling R., Brewer, M., Knutson, A., Biles, S., Way, M. and Sekula-Ortiz, D. 2016b, Scouting sugarcane aphids in South, Central, and West Texas. NTO-43, Texas A&M AgriLife Extension Service, College Station, TX. (http://ccag.tamu.edu/sorghum-insect-pests/).
188. Buntin, G. D. and Roberts, P. M. 2016, Arthropod Manag. Tests, 41, 1-2.
189. Haar, P. J., Buntin, G. D., Jacobson, A., Pekarcik, A., Way, M. O. and Zarrabi, A. 2019, J. Econ. Entomol. In press
190. Knoll, J. E., Harris-Shultz, K. R. and Ni, X. 2018, Adv. Sugar Crop Proc. Conv., 2 (in press).
191. Armstrong, J. S., Mbulwe, L., Sekula-Ortiz, D., Villanueva, R. T. and Rooney, W. L. 2016, J. Econ. Entomol., 110, 259-265.
192. MyFields.info. Anonymous. Hybrids with some resistance to sugarcane aphid. https://www.myfields.info/pests/sugarcane-aphid
193. Wang, F., Zhao, S., Han, Y., Shao, Y., Dong, Z., Gao, Y., Zhang, K., X. Liu, D. Li, J. Chang, D. Wang. 2013, Mol. Breeding, 31, 777-784.
194. Haar, P. J., Bowling, R., Gardner, W. A. and Buntin, G. D. 2018, J. Ent. Sci, 53, 104-106.
195. Hall, R. A. 1981, Fungi: *Verticillium lecanii*, In Burges, H. D. [Ed.] Microbial Control of Pests and Plant Diseases 1970-1980. Academic Press, London, 483-498.
196. Zambrano-Gutierrez, J., Alatorre-Rosas, R., Carrillo-Benitez, M. G., Lomeli-Flores, J. R., Guzman-Plazola, R. A., Azuara-Dominguez, A. and Tera-Vargas, A. P. 2019, Adv. Microbiol., 9, 38-55.
197. James, R. R., Croft, B. A., Shaffer, B. T. and Lighthart, B. 1998, Environ. Entomol., 27, 1506-1513.
198. Wellings, P. W. 1994, Eur. J. Entomol., 91, 121-125.
199. Arthropod Pesticide Resistance Database. 2019, Available at https://www.pesticideresistance.org
200. Bass, C. and Field, L. M. 2011, Pest Manag. Sci., 67, 886-890.
201. Li, X. C., Schuler, M. A. and Berenbaum, M. R. 2007, Annu. Rev. Entomol., 52, 231-253.

202. Zarrabi, A.A., Alyousuf, A., Royer, T.A., Seuhs, S.K. and Giles, K.L. 2018, Arthropod Manag. Tests, 48, 1-2.
203. Colares, F., Michaud, J.P., Bain, C.L. and Torres, J.B. 2017, J. Econ. Entomol., 110, 52-58.
204. Smith, C.M. and Chuang, W.-P. 2014, Plant Manag. Sci., 70, 528-540.
205. Stam, R. and McDonald, B.A. 2018, Mol. Plant Pathol., 19, 521-524.
206. Michaud, J.P. 2002, Ann. Entomol. Soc. Am., 94, 531-540.