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Evolution of Soybean Aphid Biotypes: Understanding and Managing Virulence to Host-Plant Resistance

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1. Introduction

The soybean aphid (*Aphis glycines* Matsumura) has rapidly become one of the most significant insect pests of soybean (*Glycine max*) worldwide (Ragsdale et al., 2007). The rise of soybean aphid in importance is in large part due to the invasion of North America ca. 2000, presumably coming from its native range in Asia (Ragsdale et al., 2004). In the first few years of the North American invasion, the soybean aphid spread across much of the North-Central US and the provinces of Ontario and Québec. Its current distribution includes over 80% of the soybean growing region of the US and Canada (Vennette & Ragsdale, 2004). Worldwide, the distribution includes much of East Asia (China, Japan, The Philippines, South Korea, Indonesia, Malaysia, Thailand, Vietnam, and Russia), all of which likely represents the ancestral range (Footit et al., 2006).

Options for management and control of soybean aphid are limited. As an alternative to chemical insecticides, host-plant resistance is a common method of aphid control (Van Emden, 2007), which uses plant hosts with genetically inherited traits that enable the plant to withstand pest attack better than a plant lacking these traits (Smith, 2005). Although soybean aphid resistant varieties have been studied in China (Wu et al., 2004), new soybean varieties have been developed with resistance to the soybean aphid specifically for use in North America (Hill *et al.*, 2004, 2006a,b; Mian *et al.*, 2008; Zhang *et al.*, 2009; Hill *et al.*, 2010). Varieties for North American commercial use were available for the first time in 2010. However, the host-plant resistance strategy is complicated because of rapid evolution of soybean aphid populations (*i.e.* biotypes) that have overcome host-plant resistance (*i.e.* virulence). Biotypes can be defined as "populations within an arthropod species that differ in their ability to utilize a particular trait in a particular plant genotype" (Smith, 2005). The presence of soybean aphid biotypes with virulence to host-plant resistance varieties *before these varieties were commercially released* suggests that the soybean aphid can rapidly adapt to these new lines and thereby threaten the effectiveness and sustainability of the host-plant resistance strategy.

Fig. 1. Soybean aphid infestation on soybean

To fully understand the threat to host-plant resistance, research must focus not only on developing new varieties of soybean-aphid resistant soybean, but also on the genetic, genomic and evolutionary factors involved in adaptation and spread of soybean aphid biotypes. This chapter will review the current status of soybean aphid biotypes, highlight functional and population genetic and genomic studies that may provide clues to uncovering the basis for soybean aphid biotype adaptation, and outline future studies that will be necessary if resistant soybean varieties are to be used and preserved as a soybean aphid management strategy.

2. Soybean aphid biology

The soybean aphid is among the more than 450 named species within the genus *Aphis* (Blackman & Eastop 2007; Couer d'acier et al., 2007). However, despite the total number of named species, the evolution of the genus is difficult to interpret and remains largely unresolved, mostly due to a recent adaptive radiation (Couer d'acier et al. 2007; Kim & Lee 2007). This is especially true for the 3-4 species groups: *A. fabae, A. craccivora, A. gossypii* and *A. frangulae*, with the latter 2 groups sometimes lumped together as the *A. gossypii* group (Kim & Lee, 2008). Kim & Lee (2008) placed *A. glycines* relatively basal within the *A. gossypii* group and to *A. glycines* itself, based on mitochondrial and nuclear DNA. However, Wu et al. (2004) report that *A. glycines* and *A. gossypii* can produce viable offspring in the laboratory and in nature, as well as share an overwintering host, *Rhamnus davurica* Pal. The apparent disconnect between genetic and mating evidence points to the difficulty of establishing accurate species relationships within *Aphis*.

The original distribution of *A. glycines* is described as Oriental and East Asian (Footit et al. 2006), which overlaps with the original cultivation of soybean (Qiu & Chang 2010). Its initial spread was probably related to the expansion of soybean. There are at least two known recent introductions since 2000. The soybean aphid was reported to be found in Australia (Fletcher and Desborough, 2000), although any concern of achieving invasive or pest status appears to

be unfulfilled. The more serious North American invasion was also first detected in 2000 in Wisconsin (Ragsdale et al., 2004; 2007), within 250 km of the city of Chicago, a major international trading center by both sea and air travel. How the soybean aphid invaded, the size of the invasion, and where it came from is still unknown. Nonetheless, within 3 years of its arrival, the soybean aphid had spread to 21 US states and 3 Canadian provinces, placing an estimated 80% of the North American soybean crop at risk (Vennette and Ragsdale, 2004). The current distribution is widespread across North America, although the heaviest infestations and economic damage occurs within the Midwestern US, and the Great Lakes states and the provinces of Ontario and Québec, Canada.

The life cycle of the soybean aphid in North America is similar to what is known from its native range (Wang *et al.* 1994). It is a typical heteroecious, holocyclic species, alternating among sexual reproduction on primary hosts and asexual reproduction on secondary hosts (Ragsdale *et al.* 2004). The primary hosts are various buckthorn trees (*Rhamnus* spp., Voegtlin *et al.* 2005; Yoo et al. 2005). Buckthorn is a widely distributed herbaceous shrub in the flowering plant family Rosales. This shrub is commonly used for horticultural and landscaping, especially along hedges and fence rows. These characteristics led to the introduction of buckthorn into North America from Europe by the early 19th century. It quickly became invasive and led to an invasion meltdown involving 10 other Eurasian plant, animal and pathogen species (Hiempel et al. 2010). In North America, overwintering of the soybean aphid mainly occurs on *Rhamnus cathartica* (common buckthorn, Figure 3), but can also occur on *R. alnifolia* (alderleaf buckthorn), *R. lanceolata* (lanceleaf buckthorn) and *F. alnus* (glossy buckthorn) (Voegtlin et al. 2005; Yoo et al. 2005; Hill et al. 2010). Across its native Asian range, *R. davurica* and *R. japonica* are the main hosts (Yoo et al. 2005).

Fig. 2. Soybean aphids on buckthorn (*R. cathartica*). Note the presence of winged forms (most likley males (androparae) and females (gynoparae) and wingless females (oviparae). Picture taken on October 6, 2006 in Toledo, OH, USA. Photo Credit: R. B. Hammond, The Ohio State University.

The secondary host is largely restricted to soybean (Blackman & Eastop 2000; Ragsdale et al. 2004) although some additional hosts may play a minor role (Blackman & Eastop 2000). On soybean, populations can double every 6-7 days (Ragsdale *et al.* 2007), with close to 15 generations occurring within one growing season. Fully developed adults can produce more than 9 nymphs per day and total more than 60 nymphs in a lifetime under laboratory conditions (McCornack *et al.* 2004). During autumn, soybean aphids return to primary host, probably cued by soybean senescence and the typical environmental changes associated with the change of seasons.

Feeding of the soybean aphid on soybean affects various plant characteristics including seed quality and size, pod number, and plant height, and, most importantly, overall yield (Hill *et al.*, 2004; Ragsdale et al. 2007). Dramatic yield losses of up to 50% have been reported under heavy soybean aphid infestations (Wang *et al.*, 1994), and evidence suggests even small infestations can lead to disruptions in normal soybean physiology (Macedo *et al.* 2003). The soybean aphid also poses a potentially large threat to soybeans because of its ability to vector a variety of viruses such as *Potato Y virus, Alfalfa mosaic virus*, *Soybean mosaic virus*, *Cucumber mosaic virus* and possibly *Soybean dwarf virus* to soybean and other crops (Iwaki *et al.* 1980; Hill *et al.* 2001; Clark & Perry 2002), adding to the economic importance of this pest. Virus transmission is especially important in food grade, seed grade and organic soybean production, since known thresholds are only based to prevent yield loss. Economic losses from the soybean aphid have been calculated between US$2.4 and US$4.9 billion and annually (Song *et al.* 2006; Kim et al. 2008).

3. Soybean aphid management

There are few methods for controlling soybean aphid. Chemical control of soybean aphid is effective but requires frequent scouting by trained individuals and use of established thresholds (see Ragsdale et al. 2007). Although effective, control with insecticides may be environmentally hazardous and harm beneficial and natural enemies, leading to repeated applications and the increased risk of environmental degradation (Costamagna and Landis 2006). A safer and better strategy to combat the soybean aphid may be obtained by host-plant resistance.

A few historical studies have measured soybean aphid resistance in the soybean aphid (Wu et al. 2004). The North American invasion has necessitated a resurgence in screening soybean lines for resistance to the soybean aphid. In various studies, host-plant resistance in the form of both antibiosis (reduced survival/fecundity) and antixenosis (non-attractive/repellency of establishment) has been found. To date, 4 independent *Rag* (Resistance to *Aphis glycines*) genes have been described: *Rag*1 (Hill *et al.* 2004), *Rag*2 (Mian *et al.* 2008), *Rag*3, and *Rag*4 (Zhang *et al.* 2009). *Rag*I was first identified in the soybean cultivar Dowling and maps to soybean linkage group (LG) M (Hill et al. 2006a; Li et al. 2007). Similar resistance was found in the cv. Jackson, as was found to map at the same location as Dowling, suggesting a possible allelic difference (Hill et al. 2006b). *Rag*2 was discovered in two plant introductions (PIs): 243540 and 200538 and maps to LG F (Kang et al. 2008, Mian et al. 2008, Hill et al. 2009). Zhang et al. (2009) found 2 resistance genes in PI 567541B, *Rag*3 and *Rag*4. *Rag*3 maps close to *Rag*1 and *Rag*4 maps on LG F, but is not near *Rag*2. There are a few other promising PIs (e.g. 567301B, Mian et al. 2008) that may yet reveal additional major or minor effect *Rag* genes. The first North American commercially released soybean aphid resistant cultivar (starting in 2010) will contain *Rag*1.

The host-plant resistance strategy is complicated by soybean aphid biotypes. The resistant soybean lines with the *Rag*1 genes provided strong resistance against soybean aphids collected from Illinois (Hill *et al.* 2004, 2006a, b). However, when tested against soybean aphids collected in Ohio, these soybean lines were ineffective, and Ohio aphids were able to survive and reproduce on *Rag*1 as effectively as on susceptible plants (Kim *et al.* 2008). Mian *et al.* (2008a) noted that the same aphids that had survived on *Rag*1 did not survive on *Rag*2 (PI 243540). These biotypes, defined by their differential survivability on *Rag* genes, have been named biotype-1 and biotype-2 where biotype-1 is defined as avirulent to *Rag*1, and biotype-2 is defined as virulent to *Rag*1. Hill et al. documented the presence of biotype-3. This biotype was initially collected in 2007 overwintering on *Frangula alnus* in Indiana. Testing of biotype-3 on both *Rag*1 and *Rag2* varieties revealed that while *Rag*1 resistance was retained, there was no significant difference no significant difference in colonization, survivability and reproduction with biotype-3 on *Rag*2 when compared to susceptible varieties (Hill et al. 2010). Thus, biotype-3 is defined as avirulent to *Rag*1 but virulent to *Rag*2. Table 1 represents the current status and definitions of known biotypes—these initial data suggests there may be more biotypes present that have yet to be identified.

Resistance Gene or soybean PI	Biotype 1	Biotype 2	Biotype 3
*Rag*1	avirulent	**virulent**	avirulent
*Rag*2	avirulent	avirulent	**virulent**
PI 567301B[a]	avirulent	avirulent	ND[b]
PI 567541B	avirulent	avirulent	**moderately virulent**
PI 437696	avirulent	avirulent	avirulent

[a]From Mian et al. 2008
[b]Not determined

Table 1. Known soybean aphid biotypes and virulence relationships (Hill et al. 2010)

Given the presence of soybean aphid biotypes, questions have arisen on the sustainability and efficacy of incorporating host-plant resistance for soybean aphid management. The evolution of soybean aphid biotypes, and therefore the usefulness of host-plant resistance, depends on 1) the genetic variation for virulence, and 2) how this variation can spread locally, regionally and globally. Unfortunately, few molecular resources exist for the soybean aphid (but see Bai et al. 2010) which limits researchers' ability to infer the durability of soybean aphid resistant soybean. Further studies on aphid biotype evolution are therefore necessary if host-plant resistance is to be a viable aphid management strategy. The following sections will focus on some preliminary studies that may provide the foundation for future work in understanding biotype evolution.

4. Genetic basis for biotype evolution

Many species within the family Aphididae have biotypes in association with host-plant resistance (Smith 2005)—van Emden (2007) lists 14 species not including the soybean aphid. Three characteristics of the aphid life cycle make this group more amenable to biotype adaptation than other insect pest species. First, most aphids carry bacterial endosymbionts (e.g. *Buchnera*, *Hamiltonella, Rickettsia*, *Arsenophonus, Regiella, Serratia*) which provide essential amino acids and may be involved in biotype formation and aphid defense (Ruggle

and Gutierrez 1995; Birkle and Douglas 1999; Moran and Wernegreen 2000; Wille and Hartman 2009; Oliver *et al.* 2010). These endosymbionts have been associated with different biotypes or host-races of insects, presumably because of the different nutrients and amino acids afforded by different hosts (Simon *et al.* 2003; Chiel *et al.* 2007). For example, Ruggle and Gutierrez (1995) indicated virulence to Lucerne (alfalfa) varieties is symbiont based. Second, aphids feed exclusively on plant phloem. Typically, this type of feeding induces consistent responses within plants through interactions with aphid saliva (Mutti *et al.* 2008), and places great importance on the role of the salivary glands in biotype adaptation. Previous research suggests specific factors found in aphid saliva are important in biotype adaptation. For example, greater pectin methylase activity in saliva of *Schizaphis graminum* biotypes leads to resistance breakdown in sorghum (Dreyer and Campbell 1984), and saliva related proteins may be involved in *Diuraphis noxia* biotype adaptation against wheat (Lapitan et al 2007). Third, the rapidity with which aphids are able to adapt is no doubt aided by their complex life cycle. Most species are holocyclic (alternating between primary and secondary hosts) and heteroecious (undergoing sexual and asexual reproduction), although variations and phenotypic plasticity are common (Moran 1992; Blackman and Eastop 2000; 2007). Any adaptation that evolves during the asexual stage can quickly become common and spread, as the generation time can be as little as a few days. However, despite the frequency at which biotypes evolve in aphids, very little is known about the genetic mechanisms of biotype evolution in general and in the soybean aphid in particular. Only in few studies (Dreyer and Campbell 1984; Lapitan et al. 2007) have mechanisms been explained, but yet the gene(s) involved still remain elusive.

This lack of knowledge also extends to the genetic and physiological mechanisms that provide soybean aphid resistance to the plant. Although genetic and quantitative trait loci (QTL) mapping has located genomic regions responsible for resistance, the actual gene(s) conferring resistance at each locus has not been identified or cloned. Using cDNA microarrays and various stages of soybean aphid infestation, 140 genes showed differential expression in among resistant (cv. Dowling—*Rag*1) and susceptible varieties (Li et al. 2008). These genes were related to many molecular processes including cell wall, defense, metabolism, and signaling. Verification of 3 defense related genes using quantitative PCR showed a more rapid and prolonged induction in cv. Dowling versus susceptible and illustrated a possible earlier inducement of genes related to resistance. Interestingly, some of the genes shared a common mechanism with the pathogen *Pseudomonas syringae*.

In a recent study, Chiozza et al. (2010) compared amino acid profiles between soybean lines LD05-16060 (resistant, *Rag*1) and its susceptible isoline, SD01-76R. Various amino acids show both constitutive and induced concentration differences. Most amino acids showed a higher constitutive concentration in the susceptible line at the V6 stage, including α-aminobutyric acid, aspargine, glutamine, glutamic acid, histidine, proline and serine. However, – α-aminobutyric acid, glutamine, histidine, and tryptophan all had significantly higher quantity in resistant plants during R4 and R5 stage than susceptible plants. Chiozza et al. (2010) suggest that the decrease of aspargine and glutamic acid could have a nutruitive effect on the soybean aphid related to resistance. Indeed, performance of the soybean is lower when glutamic acid is not available (Wille & Hartman 2009). In addition, most aphids carry endosymbiotic bacteria (*Buchnera aphidocola*) which are involved in transport and synthesis of essential amino acids, including glutamic acid (Liadouze et al. 1995; other sources). If indeed a decrease in glutamic acid provides resistance, it is tempting to speculate that soybean aphids virulent to *Rag*1 may harbor a different *B. aphidicola* strain that is

efficient in metabolic pathways involving glutamic acid, thereby compensating for the low quantities available in *Rag*1. The resistance offered in other soybean aphid resistance varieties has not been investigated as much as *Rag*1.

Recent studies have now laid the groundwork for detailed investigations into the genetics and mechanisms of soybean aphid resistance and aphid biotype adaptation. First the whole genome sequence for soybean has been published (Schmutz et al. 2010), which will allow for better annotation of soybean aphid resistance QTLs and well as provide higher-resolution markers for fine-scale mapping. In addition, Bai et al. (2010) presented the first significant sequencing resource for the soybean aphid. Over 278 million base pairs were sequenced, resulting in 56,688 genomic sequences and 19,293 transcripts (i.e. expressed sequences). In addition, 39,822 bp of sequence shared similarity with the obligatory endosymiont *Buchnera aphidocola* and to the facultative endosymbiont *Hamiltonella defensa.*

5. Population biology issues: The distribution and spread of biotypes

The ability and potential of virulence to spread across the soybean growing region is critical to the increase in virulent biotype frequencies and the decrease in host-plant resistance durability. The insect resistance management (IRM) strategy for transgenic corn is a decent analogy to a possible solution for preserving susceptible soybean aphids. In the IRM system, a maximum percentage of insect-resistant corn can be planted (usually 80-95%) with the remainder reserved for planting a susceptible variety (refuge). The refuge provides a proportion of insects that do not encounter the intense selection pressure of resistant crops, and hence should maintain susceptible alleles in the insect population. These refuge insects are also available to mate with any potential resistant insects, thereby producing susceptible offspring (assuming resistance in insects is recessive). However, the soybean aphid is not a typical insect pest as explained above. The evolution of a specific biotype does not necessarily indicate the durability of a particular resistant variety will be impacted. There are various factors that can affect how quickly a biotype can increase in local frequency, spread regionally and globally, and transfer genetic variation for virulence into future generations: fitness depends both on survivability and mating success. Its obligatory asexual and sexual phase, as well as its reliance on a single plant for mating and overwintering, complicates the potential spread of virulence throughout populations. During the soybean aphid life cycle, there are three specific migration events that can all affect how virulence can spread. First is the summer, asexual generation migration among soybean fields, second is the autumn return from soybean to buckthorn, and third is the spring migration from buckthorn to soybean. During these migration events, soybean aphids are at the mercy of population genetic, demographic, landscape, ecological, environmental and evolutionary forces that can either promote or abate the spread of biotypes.

Summer Migration. Once soybean aphids colonize soybean, asexual reproduction can rapidly increase population sizes. Like many aphid species (Dixon 1988), temperature plays a large role in the reproductive rate for the soybean aphid. In laboratory conditions, development thresholds range from 9.5ºC to 34.9ºC, with 27.8ºC found to be the optimal temperature (Hirano et al. 1996; McCornack et al. 2004), with population doubling times ranging from 1.5 days (at 25ºC) to 1.9 days (at 20ºC and 30ºC) (McCornack et al. 2004). In the field, Ragsdale et al. (2007) found an average population doubling time of 6.8 days. Thus, just by temperature alone, the potential of a virulent biotype to rapidly increase in frequency is high. However, other important factors may play a role such as plant host quality and presence of natural enemies (Ragsdale et al. 2007; Costamagna a& Landis 2006).

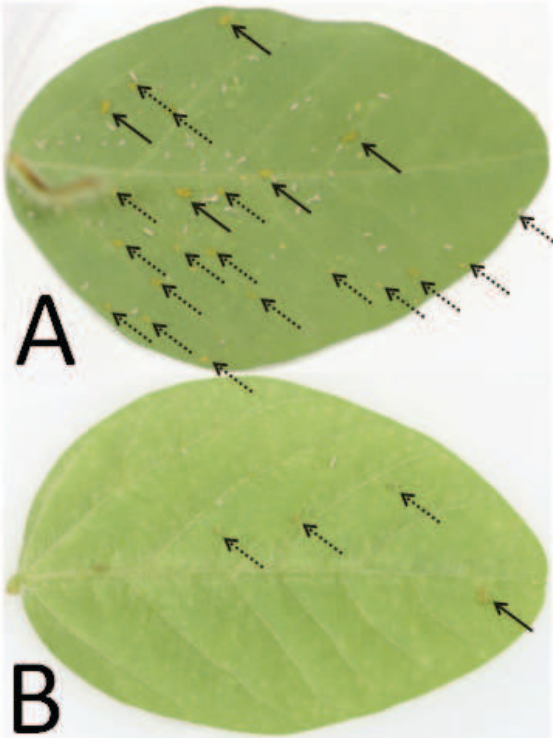

Fig. 3. Aphid reproduction on a detached cv. Jackson leaf. Each leaf started with 1 adult aphid, figure shows reproduction after 7 days. A) Biotype 2; B) Biotype 1. Solid arrows point to adults, broken arrows point to nymphs.

After the initial colonization of soybean, there is a slight delay before alates are produced that can migrate and infest other surrounding fields. During this phase, there are likely no restrictions to migration, and alates can spread across fairly large distances. Using microsatellite molecular markers, Michel et al. (2009), showed insignificant amounts of genetic differentiation over 1,500 km. This pattern was in contrast to populations collected earlier, i.e. before the late summer migration, where populations were more genetically distinct. However, this study could not rule out that the distribution of genetic variation was due to a more a step-wise manner of migration (geographic scales necessary for accurate isolation by distance assessment were not included). Nonetheless, there appears little to impede the spread of a virulent biotype among soybean fields. The rapid invasion during which the soybean aphid spread to over 20 US states and 3 Canadian provinces in 3 years also testaments to the large migratory ability.

If indeed there are few limits on migration, then estimating the likelihood of biotype spread depends on the initial frequency and distribution, and how quickly these frequencies can increase. However, research is limited in both areas. Recently, Michel et al. (2010) developed biotype determination assay using detached resistant soybean leaves (Figure 3). In cv. Jackson (*Rag*1-like, see above) and PI243540 (*Rag*2), similar levels of aphid resistance was found between detached leaves and whole plants, suggesting that resistance is retained.

This was further corroborated by comparing growth rates (i.e. net fecundity or the number of nymphs) of biotype 1 and biotype 2 on detached cv. Jackson, where the growth rate of biotype 2 was consistently more than double that of biotype 1 after the second day. The growth rate estimates for biotype 1 and biotype 2 can then be used as baselines in comparisons to unknown field samples. In 2009, aphids were collected in total of 8 fields (6 in Ohio, and 1 each in South Dakota Kansas) and reared on detached cv. Jackson leaves. Table 2 shows the frequency distribution of aphids with net fecundity matching biotype 1, between biotypes (moderately virulent to *Rag*1), biotype 2, or exceeding biotype 2 (A.P. Michel, unpublished).

First, the results show much variability, even among fields from the same state. The frequency of aphids that had similar growth rates as biotype 2 or greater ranged from 0 to 40%. This variation also persisted regionally—Ohio had a relatively higher rate of *Rag*1 virulent aphids, Kansas had a more moderate level (14%), and South Dakota had no aphids which matched or exceeded growth rates of biotype 2. Aphid colonies have been found on *Rag*1 bearing plants in South Dakota (K. Tilmon, South Dakota State University, personal communication), but whether this represents 23% of aphids moderately virulent to *Rag*1, or actual biotype 2 aphids undetected with the above data is unknown. In any case, the data illustrates that, at least for *Rag*1, virulence is widely distributed. It is also important to note, however, that these biotype 2 frequencies reflect natural genetic variation *without* the presence of a large selection pressure.

		Number of aphids that produced:				
Location	N[a]	<7[b] nymphs (biotype 1)	8-15[c] nymphs	15-19[d] nymphs (biotype 2)	>20 nymphs	Average
OH-1	25	14 (56%)	11(44%)	0	0	8.1
OH-2	40	12 (30%)	26 (65%)	1 (3%)	1 (3%)	9.1
OH-3	26	12 (46%)	10 (38%)	3 (12%)	1 (4%)	9.3
OH-4	40	10 (25%)	16 (40%)	7 (18%)	7 (18%)	12.1
OH-5	36	9 (25%)	13 (36%)	8 (22%)	6 (17%)	12.1
OH-6	35	6 (17%)	15 (43%)	7 (20%)	7 (20%)	13.9
SD	30	23 (77%)	7 (23%)	0	0	4.4
KS	21	9 (43%)	9 (43%)	3 (14%)	0	8.7
Overall [e]		11.9 (40%)	13.4 (40%)	3.6 (10%)	2.8 (10%)	

[a]Number of aphids tested
[b]7.12 is the upper 95% Confidence Interval (CI) for biotype 1
[c]15.54 is the lower 95% CI for biotype 2
[d]18.50 is the upper 95% CI for biotype 2
[e]among all locations

Table 2. Frequency and geographic distribution of soybean aphid virulence to cv. Jackson

Given the migration ability and biotype frequencies, researchers must next address two critical questions. First, is the comparative level of fitness between biotypes in different resistant plant

environments and the resulting different selection pressures. Initially, host-plant resistant lines may be of limited release and use. Susceptible hosts could then act as a sink for virulent biotypes, but only if they are less fit than avirulent biotypes. If virulent biotypes outcompete avirulent biotypes even in susceptible environments, then virulence will spread much more rapidly. Second, the increase and spread of virulent biotypes may also depend on the initial starting frequency. For example, how much more quickly will biotype 2 increase in frequency with a initial frequency of 2% when compared to 20%? More importantly, as soybean producers look to an integrated management approach with insecticide seed treatments, soybean aphid resistant genes, and chemical applications, the rate at which (or if) these frequencies reach economic threshold (Ragsdale et al. 2007) must be estimated. These estimations are crucial as the frequency of biotypes produced in soybean fields represent the overall potential population that is available for mating and transmitting genetic variation for virulence to the next generation, assuming soybean aphids can successfully colonize their primary host. Finally, and most importantly, is the level of selection pressure placed on soybean aphid populations. Increased adoption of a certain resistant soybean variety will rapidly increase virulent biotypes by increasing the likelihood of successful migration. For example, assuming migration is random among soybean fields, biotype 2 is likely to survive if adoption of *Rag*1 varieties is high (i.e. high selection pressure). But if *Rag*2 varieties are also widespread throughout the landscape, then a certain number of biotype 2 aphids will suffer mortality and virulence for *Rag*1 will be slower to increase in frequency (until, of course, a biotype arises with virulence for both *Rag*1 and *Rag*2).

Autumn Migration. As stated earlier, the primary or overwintering host of the soybean aphid is buckthorn. However, there is a drastic difference in distribution between the soybean aphid, especially during the peak summer months, and the presence of buckthorn. The soybean aphid has been found as far south as Alabama and Georgia in the USA (about 34ºN latitude), but buckthorn is mainly found in the North Central US and Great Lakes region of the US and Canada and rarely present or abundant at latitudes below 41ºN (Ragsdale et al. 2004). Thus, in some cases, soybean aphids present in southern US states would have to migrate long distances in order to find its primary host plant. It is untested whether or not these more southern aphids from soybean can successfully return to buckthorn, but previous population genetic studies suggest no biological restraint on long-distance migration (Michel et al. 2009). The relative contribution of these aphids to the mating population may be more important, i.e. if long-distance buckthorn colonization occurs but at lower success rates than for aphids where buckthorn is in closer proximity. Understanding possible differences in migration success from geographical populations is critical in estimating the durability of host-plant resistance. For example, if it is shown that soybean aphids from southern US states are less likely to colonize buckthorn, then selection pressures may be inconsequential in these areas because any new virulent biotype is less likely to transmit its genetic variation to the next generation. If biotypes cannot mate then this ultimately leads to an evolutionary dead-end. Alternatively, where success rates of buckthorn colonization are high, the deployment of host-plant resistant varieties, as well as biotype frequencies, must be more heavily managed, as genetic variation for virulence is more likely to persist.

There are other environmental and ecological factors to consider in addition to successful colonization. Once buckthorn is colonized, soybean aphids must survive long enough for oviposition to occur. Natural enemies can follow aphid migration on to buckthorn (Gray 2006) but to date no study has examined the level of biological control on buckthorn. McCornack et al. (2005) noted that alates may perish at 15-18ºC above the supercooling

point (calculated at -14.9ºC and-15.2ºC for oviparae and gynoparae, respectively). In an extremely cool autumn, significant mortality can occur before oviposition. One of the largest, if not the largest, migratory flight from soybean to buckthorn occurred in 2009 across North America (Gray 2009a). The large populations on buckthorn, combined with the cooler, wet conditions led to a fungal pathogen outbreak (Gray 2009, Issue No. 24, Article 5/November 6, 2009). Although the particular fungal pathogen causing mortality has not been identified, numerous fungal pathogens have been found from soybean aphid (Nielsen and Hajek 2005). Massive mortality ensued, and oviposition was minimal. Thus, even though migration and colonization was successful across a wide geographical area, few soybean aphids emerged the following spring (Hammond et al. 2010).

Spring Migration. Soybean aphid eggs are laid in late autumn near buds or on the bark. Egg hatch is most likely determined by temperature and largely coincides with bud swell of buckthorn (Bahlai et al. 2007). Bahlai et al. (2007) were able to develop a predictive model for egg hatch based on temperature alone, but the inclusion of heat units from solar radiation dramatically improved the accuracy of the model, suggesting a microclimate effect (see also Welsman et al. 2007). Fundatrices, or stem mothers, are the form of aphids that hatch from eggs. This behavioral form is usually plump and large, and begins asexual reproduction. At least 2-3 assexual generations occur on buckthorn, and growth rates can be dependent on spring temperatures, up to 1.5 generations per day at 24ºC (Bahlai et al. 2007).

Various ecological and environmental factors can significantly influence the spring population size and the number of alates that can eventually colonize soybean. McCornack et al. (2005) showed that successful egg overwintering is not likely to occur below -34ºC, which is the supercooling point of soybean aphid eggs (the supercooling point is the temperature at which freezing of tissue occurs). In addition, reproduction on buckthorn can be limited by temperature. In 2007, an extremely warm spring encouraged buckthorn bud break and soybean aphid egg hatch. Within the next few weeks, temperatures rapidly decreased and remained below freezing, which likely caused fundatrix and spring viviparae mortality (Hammond & Eisley 2007). Of the aphids that remain on buckthorn, alates are eventually formed, perhaps by a density dependant mechanism (Bahlai et al. 2007), and migrate to soybean fields.

The colonization of soybean from buckthorn remains complicated in both the timing and the influence of landscape, particularly the proximity to buckthorn (Ragsdale et al. 2004; Bahlai et al. 2010). Ragsdale et al. (2004) noted that colonization occurred in only 4 of 42 locations of soybean aphid infested buckthorn stands that contained potted soybean plants. This is in keeping with the observation that, in some locations, no local sources of soybean aphid on buckthorn can be found as soybean begins to emerge. In addition, observations from Minnesota, Illinois and Indiana showed that on occasion alates have been formed on buckthorn several weeks before soybean emergence, though there is no other known alternate host to support these early migrants. This "phenological disjunction" between the primary and secondary hosts of soybean and the aphid itself can lead to paucity of aphid genetic founder material for soybean colonization and influence biotype frequencies.

If a genetic bottleneck during this migration event occurs, then early populations of soybean aphids should contain less genetic diversity and fewer clones than populations collected later in the season. Using microsatellite molecular markers, Michel et al. (2009) compared clonal and genetic diversities among early collections from Michigan (July 7, 2008) and Ontario (July 22, 2008) to eight other U.S. populations collected after August 15, 2008. Populations from Michigan and Ontario had a significantly lower number clones per

population than mid-August collections (average genotypic diversity, *GD*: 0.47 and 0.82, for early and late, respectively, Mann-Whitney U = 16.0, $P = 0.02$), and significantly less clones (12.5 and 28.9, respectively, Mann-Whitney U = 16.0, $P = 0.02$), even when taking into account differences in population sample size. Both measures were significantly correlated with time (*GD*: $R = 0.53$, $P = 0.013$; clones: $R2 = 0.51$. $P = 0.02$). Later season populations contained higher levels of clones and *GD*, presumably due to the immigration of other clones from nearby fields. Thus, molecular evidence supports a genetic bottleneck during soybean colonization. In this case, initial population sizes of soybean aphids in soybean are low and consist of very few clones. Diversity is then a result of the interplay between genetic drift (including random mortality from environmental conditions or natural enemies), and selection through clonal amplification. Clonal amplification refers to the rapid increase in frequency in certain asexual clones through selection which is quite common in many aphid species during the asexual phase (Vorburger et al. 2003; Vialette et al. 2005), and likely occurs in soybean aphid (Michel et al. 2010). Currently, the use of soybean aphid resistant varieties is limited, therefore selective pressure and clonal amplification of specific virulent biotypes is not likely to occur. If adoption increases, however, the spread of a virulent biotype might depend more on its ability to colonize a particular resistant soybean variety because, if successful, clonal amplification can quickly increase its frequency.

Random mortality during the spring colonization of soybean suggests that colonization of soybean may be restricted or most important at small spatial scales, dependant on buckthorn abundance and proximity. Using population dynamic modeling and the Akaike's information criterion (which compares the likelihood of competing models), Bahlai et al. (2010) showed that, indeed, the presence or absence of buckthorn was highly related to soybean aphid colonization, and the estimated number of buckthorn shrubs best predicted aphid density, although the distances explored were less than 5km. As soybean aphids likely use both short and long distance dispersal (Zhang et al. 2008; Michel et al. 2009) expanding these models across larger distances would improve the ability of identifying buckthorn source populations for soybean colonization.

These migration and colonization events may have a profound effect on the level and distribution of biotypes, and ultimately how virulence evolves on a regional scale. If, for example, local soybean aphid populations can adapt to local fields of resistant varieties, then we might expect the sustainability of these varieties to be low, as each local population represents an opportunity for adaptation. Alternatively, if adaptation to resistant varieties depends on distinct virulent genotypes, then virulence evolution will be at the mercy of migration and colonization patterns and the stochastic demographic events associated with such movement. If substantial mortality occurs during colonizations and migrations, and if these events are geographically dependant (e.g. distribution of buckthorn) the likelihood of virulent genotypes spreading may be low. While the movement and migration patterns of the soybean aphid are becoming clearer (Michel et al. 2009; Bahlai et al. 2010), a lack of understanding on the genetics of virulence hinders the ability to accurately predict host-plant resistance sustainability.

6. Conclusion and future needs for biotype evolution

As the discovery of soybean aphid biotypes occurred relatively recently, (Kim et al. 2008; Hill et al. 2010), much information is still needed to determine their influence on resistant soybean varieties. Most critical are determining the relative frequencies and distributions of

biotypes, and further elucidating the role of the overwintering host in regards to population genetic issues. However, the largest obstacle remains individually diagnosing aphids to biotypes. Using detached leaves may help (Michel et al. 2010), but this is a laborious process (taking about 2 weeks) and may not work for all host-plant resistant varieties. For example, Michel et al. (2010) demonstrated that PI567301B, which shares similar qualities to *Rag*2 (PI243540, Mian et al. 2008), does not retain resistance when leaves are detached from whole plants. Ultimately, diagnosis should rely on molecular methods, but linking molecular markers to biotype designations may take many years and prove difficult in an aphid species where any such genetic markers may disassociate from the virulence trait through recombination in the obligatory sexual phase. Newly developed molecular resources for the soybean aphid should help in the search for diagnostic genetic markers.

The question remains, however, as to how durable host-plant resistant soybean varieties against the soybean aphid will remain effective. In other host-plant resistance systems with virulent aphid biotypes, resistant varieties can remain durable for extended periods of time. For almost 100 years, resistance in grapes (*Vitis vinifera*) to *Diuraphis noxia* has held. For *Schizaphis graminum*, host plant resistance in wheat has been tenuous at best (Puterka and Peters 199). However, studies have suggested that other hosts instead of wheat (wild grasses) were driving biotype adaptation instead (Anstead et al. 2003). Since the soybean aphid has no known significant secondary host other than soybean, research on *S. graminum* may not be a completely accurate comparison, but it may forewarn users and researchers about the utility of host-plant resistance.

The presence and evolution of soybean aphid biotypes places great importance on the constant development of new soybean aphid resistant soybean varieties (Hill et al. 2010). By having a wide array of potential soybean aphid resistant genes and varieties—including the possibility virulence can likely be managed and the durability of host-plant resistance will be preserved. In addition, the possibility of mixing or gene pyramiding (combining more than one resistance gene in a variety) and geographically varying *Rag* gene deployment may extend the life of host-plant resistance (Porter et al. 2000; Bush et al. 1991; Smith 2005). Furthermore it is important to keep in mind other potential control tactics including insecticidal seed treatments and mid or late season insecticide applications. Through a multi-year and multi-state research collaboration, an economic threshold exists for the soybean aphid (Ragsdale et al. 2007), which works for the majority of soybean fields. The integration of all tactics will be necessary to slow the evolution of soybean aphid biotypes and extend the durability of host-plant resistance in soybean.

7. Acknowledgements

We would like to acknowledge W. Zhang, L. Orantes, Y. Chen, N. Davila-Olivas, L. Cañas, J. Todd, and L. Wallace for soybean aphid rearing and detached leaf assay tests. T. Mendiola. K. Freewalt and T.-H. Jun assisted with soybean rearing and assessing host plant resistance. C. Hill (University of Illinois) provided biotype 1 individuals. Soybean aphid from South Dakota and Kansas were collected and sent by Dr. K. Tillmon (South Dakota State University) and Dr. B. McCornack (Kansas State University). Images were provided by Dr. R. Hammond (The Ohio State University). Dr. B. Bonning (Iowa State University) provided comments regarding soybean aphid genomics. Funding was provided by The Ohio Soybean Council, and the Ohio Agricultural Research and Development Center, The Ohio State University.

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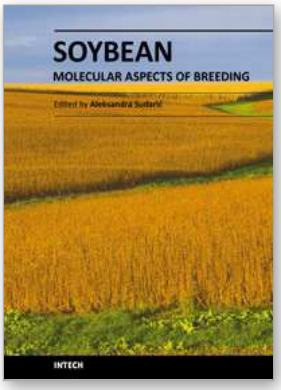

Soybean - Molecular Aspects of Breeding
Edited by Dr. Aleksandra Sudaric

ISBN 978-953-307-240-1
Hard cover, 514 pages
Publisher InTech
Published online 11, April, 2011
Published in print edition April, 2011

The book Soybean: Molecular Aspects of Breeding focuses on recent progress in our understanding of the genetics and molecular biology of soybean and provides a broad review of the subject, from genome diversity to transformation and integration of desired genes using current technologies. This book is divided into four parts (Molecular Biology and Biotechnology, Breeding for Abiotic Stress, Breeding for Biotic Stress, Recent Technology) and contains 22 chapters.

How to reference
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Andrew P. Michel, Omprakash Mittapalli and M. A. Rouf Mian (2011). Evolution of Soybean Aphid Biotypes: Understanding and Managing Virulence to Host-Plant Resistance, Soybean - Molecular Aspects of Breeding, Dr. Aleksandra Sudaric (Ed.), ISBN: 978-953-307-240-1, InTech, Available from: http://www.intechopen.com/books/soybean-molecular-aspects-of-breeding/evolution-of-soybean-aphid-biotypes-understanding-and-managing-virulence-to-host-plant-resistance

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