FINAL PROJECT REPORT

Project Title: Assessment of apple immune responses to wooly apple aphid saliva

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Total Project Funding: **$**164,987 **Year 1**: 58,710 **Year 2**: 49,079 **Year 3**: 57,198

Budget History:

Item	2016	2017	2018
Salaries	$32,836	$20,646	$32654
Benefits	$3,424	$16,463	$22044
Wages			
Benefits			
Equipment			
Supplies	$22,450	$11,970	$2,000
Travel			$500
Plot Fees			
Miscellaneous			
Total	$58,710	$49,079	$57,198

OBJECTIVES

All plants share networks of related genes and proteins that work together to generate immune responses to both insects and pathogens. The main goal of our project was to identify these networks in apple as they relate to aphid feeding, although resolving these immune networks will inform upon any biotic stress response imposed on apple in the future. A complementary goal was to examine how the aphids trigger these networks by characterizing insect genes. For both of these goals, we trained a MS student in current bioinformatics techniques and with expertise in apple-aphid interactions. Our approach combined transcriptomic information on the apple genes induced by aphid colonization with the genes active in aphids as a first hurdle in linking insects to plants. This fundamental research will help us to better understand resistance mechanisms in apple and how insect populations vary across growing regions. Our specific objectives were to:

1. Identify the WAA salivary proteins that alter plant form and function in roots and shoots.
When feeding, WAA discharges salivary proteins into plant tissues. These proteins play critical roles in reprogramming the physiological processes of infested plant tissues, i.e., roots and shoots. Because salivary proteins are secreted by salivary glands, we used a transcriptomic assessment of extracted salivary glands to identify all the genes that encode secretory proteins in WAA. We compared this to whole body extractions to rule out transcripts expressed in dissected tissue but not associated with salivary glands. To verify the gene products, we also collected salivary proteins for proteomic analysis. Initial proteome screens (2016-2017) revealed more insect material (within replicates and total number of samples) was necessary to increase replicates and detection given many of these proteins are low in abundance. However, recently published studies indicate the number of proteins found in insect saliva is much less than what should be produced from protein-encoding genes annotated in the transcriptome (Thorpe et al. 2016, Boulain et al. 2018). *Thus, transcriptome profiling of salivary glands was selected as a better approach to identify insect secretory proteins that antagonize plants compared to proteome collections*. Because of this we did not pursue more proteome studies. We secured extra funding to create a WAA genome to increase the ability to detect genes and their products related to colonization. This genome is assembled with annotation ongoing. Once completed, these data will represent the most comprehensive database of WAA genetics that will be publicly available at http://bipaa.genouest.org/is/.

2. Characterize the plant immune response in resistant and susceptible rootstocks.
Apple resistance to aphids is known to depend on at least four genes (Er 1-4). By assessing transcriptomes of apples that differ in susceptibility (Er-2 background) to aphid attack we can identify how genes interact to protect against aphids. We may also identify WAA-specific processes unrelated to typical resistance signaling to increase candidates or markers for resistance breeding. Sample collection was completed in Fall 2017 using the susceptible genotype G.935 and two commonly used genotypes with greater resistance G.16 and G.87 determined by performance trials. Sequencing data was returned Spring 2018 and analyses completed 2019. We also screened novel rootstocks from a resistance mapping population for performance with WAA in collaboration with G. Fazio during Summer and Fall 2017.

3. Identify functional plant traits that confer immunity to WAA.
Preliminary screens of commercial and unreleased rootstocks with known resistance genes showed variable colonization by WAA. We originally planned to phenotype the underlying biochemistry related to resistance using the transcriptome as a guide for which processes to assess. Given the analyses (objective 2) indicated RNA signaling, transcription, and post translational processing such as ubiquitination (degradation) of proteins were strongly upregulated, we chose not to screen for biochemical changes in phenolics, callose, or reactive oxygen species known to alter colonization (e.g., Zhou et al. 2013). Rather we diverted resources to annotate and compare effector genes in WAA populations from CA and WA, and evaluate apple response to the different populations. We

hypothesize that effector proteins from the aphid are targeting upstream genetic processes that regulate transcription and translation to ensure colonization. These pathways also regulate resistance in plants, and are co-expressed with nucleotide-binding site leucine-rich repeats (NBS-LRR) proteins that act to monitor effector targeting of plant processes by pathogens (McHale et al. 2006).

4. Map these traits to genes in apple to facilitate marker-assisted breeding.
Breeding-program specific DNA tests for high impact attributes are required to streamline cultivar development. Objectives 2 and 3 showed numerous genes across chromosomes play a role in resistance, making it difficult to identify single markers. However, several genes induced by aphid feeding showed both general regulation under aphid attack, and unique genotype and population-specific patterns. Motifs containing LRR, NB-ARC, TRR, R and other domains with known roles in resistance to disease were identified and linked to their chromosomal location for future marker development.

SIGNIFICANT FINDINGS
Woolly apple aphid

- >390 genes were identified as putative effectors from the de novo transcriptome. These include enzymes that detoxify compounds or otherwise mobilize nutrients for feeding, and protein-binding molecules that regulate protein signaling in apple.
- >60% of effector genes are unique to the WAA and do not occur in other insects, but some known to enhance insect performance do occur in WAA (Fig 1).
- At least one protein mimics a transmission protein necessary for successful infection of two families of plant viruses (the caulimoviruses and the potyviruses).
- CA and WA aphid populations may differ little in their genes (reanalysis with the complete genome will confirm this) but each population differentially alters apple gene expression.
- A high-quality genome assembly will serve the global apple community in understanding local and rootstock-specific resistance. With this information, virulence potential of aphid populations can be identified at local and regional levels, with regard to management (organic vs conventional), and specific to rootstocks.

Apple

- 10 unreleased genotypes were screened for resistance. One genotype prevented colonization that led to aphid dispersal/death in 5 days (Fig 6). Nine genotypes showed a range of survival between 15–40% (five shown in Fig 1). This evaluation identified rootstocks for aphid-apple transcriptomic evaluations in the future.
- Rescreening aphid performance on resistant (G.87/5087) and susceptible Geneva (16/G.16, 935/G.935) rootstocks showed similar survival (50%), indicating resistance exists beyond Er-2 for select aphid populations.
- Apple genotypes vary in constitutive expression of defense and immune-related genes, but other processes (e.g., photosynthesis, RNA processing, protein) emerge as determinants of successful colonization. Thus, a lack of Er resistance may still provide tolerance to aphids if other processes in the genotype function in an enhanced manner.
- In G.87, seven immune/effector recognition genes found on several chromosomes may contribute to aphid resistance because of elevated expression without aphids.
- Aphid feeding on apple plants remodeled the apple transcriptome more than other aphid studies at a similar time point.
- G.16 responded the greatest whereas G.935 and G.87 responded less to aphids. Comparisons among treatments revealed effector targets (genes suppressed by aphids) and effective immune response (genes induced by aphids).
- Immune signaling was differentially induced depending on the aphid population and overall CA aphids altered rootstocks more than WA aphids (Fig 5).

- Immune genes altered by aphids were identified specific to three genotypes, Er-2 resistance, aphid populations, and shared among all comparisons.

RESULTS & DISCUSSION

The woolly apple aphid has numerous plant-manipulating genes. Using a linked read technology (10x Genomics) we assembled large genomic DNA fragments from a lane of Hiseq 3000/4000, 150bp paired end reads at a coverage of 80x (Supernova 2.1.1). Reducing the total number of input reads produced an assembly of 300.82 Mbp with scaffold N50 of 3.37 Mbp. The analysis of gene content (BUSCO) resulted in 1580 complete genes, 1551 single copy, and 64 missing. This represented a high-quality assembly as a good starting point; however, we improved this genome using Dovetail Genomics, LLC and their Chicago-HiRise sequencing method. Here they constructed another short-read Chicago library of 150bp paired end reads from Illumina Miseq at a read coverage of ~80x. Using HiRise software the 10x assembly was scaffolded, increasing the N50 to 29.96kbp, and a slightly improved BUSCO with 1 more gene. This genome is currently being annotated using the Dovetail topologically associated domains method with other well annotated aphid genomes as reference. These final data will become available to the public in 2020.

Because effector characterization is becoming critical for understanding insect-induced plant responses, we performed differential expression analysis between salivary glands (SG) and whole-body samples and assessed SG-specific genes for potential to act as plant-manipulating genes (effectors). For this we de novo assembled a genome from a pool of all transcripts that showed similar statistics as the genome (e.g., 1598 complete genes by BUSCO) but will be improved once annotation is complete. Our analysis revealed 5,377 transcripts upregulated in SG at the 'isoform' level but only 390 genes that encode for secretory proteins. Known aphid effectors were found at both the isoform and gene level, indicating WAA interacts with plant signaling through processes similar to other aphids (**Fig. 1**), but 250 genes were found to be unique to WAA. Our experience annotating another aphid-like galling insect genome (grape phylloxera), leads us to predict the percentage of genes unique to WAA will remain high (~60%) and the overall effector count will increase. For example, the pea aphid encodes 3600 candidate effector genes, but only 740 are up regulated in salivary glands (Boulain et al., 2018).

We assigned tentative functions to 95 candidate effectors including various enzymes such as glycoside hydrolases (GHs) (8), peptidases (6), peroxidases (5), lipases (4), and several other enzymes. These enzymes are important to plant cell wall development, occur in other aphids, and may enable stylet penetration (Calderón-Cortés et al. 2012; Eyun et al., 2014; Wybouw et al. 2016). (Harmel et al., 2008; Miles 1999; Rao et al., 2013). Analogous to other organism antioxidant defenses, the salivary peroxidases we identified in WAA may function to counter ROS burst by scavenging H_2O_2. Differential expression analysis of WAA across different host genotypes resulted in few genes altered (26, 17 and 4 DE genes for the contrasts G.935 vs G.16, G.87 vs G.16, and G.935 vs G.87, respectively); however, we expect this

Gene	*A. pisum* gene (BIPAA)	WAA Trinity ID	Secretory	LogFC in salivary gland	Aphid Performance
C002	ACYPI008617	DN4506_c0_g1_i1	**Yes**	8.3	Increases fecundity & enables phloem feeding
		DN20770_c0_g2_i4	**Yes**	NS	
Me23	ACYPI002439	DN20770_c0_g2_i2	No	NS	Increases fecundity
		DN6812_c0_g1_i16	No	5.9	
		DN6812_c0_g1_i21	No	7.4	
		DN6812_c0_g1_i3	**Yes**	6.5	
		DN6812_c0_g1_i7	No	6	
Shp	ACYPI009881	DN6812_c0_g1_i13	No	6.4	Increases fecundity
		DN4653_c0_g3_i2	**Yes**	-2.86	
		DN11304_c0_g1_i1	**Yes**	NS	
		DN4660_c0_g2_i3	**Yes**	NS	
Mp10	ACYPI000097	DN3464_c1_g1_i1	**Yes**	NS	Increases fecundity
		DN2575_c7_g1_i1	No	-3.27	
		DN2761_c0_g1_i3	**Yes**	-2.32	
Mif1	ACYPI002465	DN2761_c0_g1_i5	No	-2.31	Increases fecundity
Armet	ACYPI008001	DN5107_c1_g1_i1	**Yes**	1.7	Increases survival

Fig. 1. Effectors known from other aphids to alter plants are present in WAA. At least 390 more effectors were detected with the majority (250) found only in WAA and not in other aphids.

number to increase with the completed annotation and re-analysis. Based on other studies, transcriptional plasticity largely determines host-specific performance of aphids (Boulain et al., 2019) but broader population assessment may reveal genotype/biotype specific genes retained or lost in distinct geographic regions.

INDUSTRY BENEFIT

- High quality genome for future WAA population and genotype-specific assessment
- Gene expression profiles for populations that vary in plant resistance responses
- Markers (effector genes) for understanding rootstock x aphid interactions

FUTURE DIRECTIONS

- Investigate population genetics of WAA to better link genotypes to growing regions and virulence on rootstocks, especially in areas where rootstock resistance failed/is failing or management costs/methods are increasing.
- Assess plant phenotypes/responses in native WAA-host (elm, hawthorn, cotoneaster) interactions to understand mechanisms for tolerance.
- Develop a genome-based genotyping protocol for virulence (effector) prediction of new genotypes that arise/invade.

Aphids used in omics analyses perform along expectations for rootstocks G.935, G.16, G.87, and G.202. A log-rank Kaplan-Meier survival analysis revealed aphids declined initially on G.935 then remained constant, indicating tolerance to or a lack of inducible defenses. Aphids declined to ~50% survival on G.16 and G.87, indicating a stronger defense response (**Fig. 2**). For all four genotypes, there were no visible signs of a hypersensitive response (i.e. necrosis), and no aphid mortality was observed. This suggests antixenotic factors determine early defense responses for these genotypes. Previous characterizations of WAA performance on apple genotypes derived from 'Robusta 5' and *M. floribunda* genetic backgrounds showed similar WAA performance/feeding behaviors (Sandanayaka et al., 2003; Sandanayaka et al., 2005).

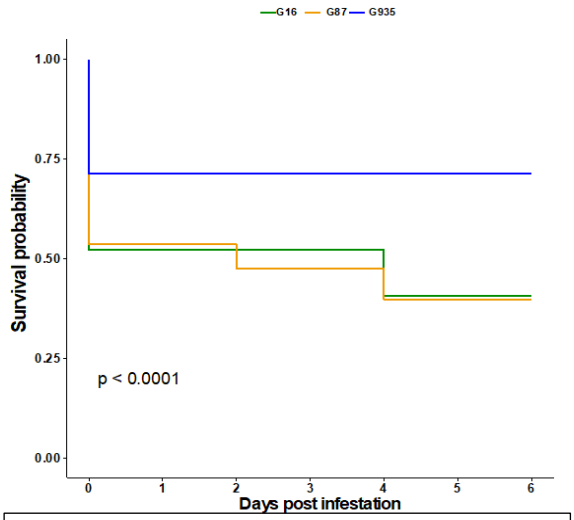

Fig. 2: Aphids showed high survivorship on susceptible G.935 but equivalent survivorship on Er2-resistant G.87 and susceptible G.16. No insects survived on G.202 (data not shown).

G.87 and G.935 are from crosses between 'Ottawa 3' and 'Robusta 5', and thus share a similar genetic background compared to G.16 ('Ottawa 3' x *M. floribunda*), yet G.87 is characterized as resistant whereas G.16 and G.935 are not. Because we found aphids performed similarly on genotypes differing in characterized resistance, we profiled their transcriptomes to understand what contributes to aphid survival in the first 2-3d of feeding.

Apple genotypes vary in constitutive expression of defense-related genes. Contrasts between **uninfested** G.935 vs G.16, G.935 vs G.87, and G.16 vs G.87 resulted in 2294, 178, and 2005 uniquely expressed genes, respectively. Notably, the expression profile for G.16 is highly dissimilar to G.935 and G.87, whereas G.87 and G.935 expression patterns are similar: a confirmation of their genetic backgrounds. Of the genes different between G.935 and G.87, there was 1 enriched bin (external stimulus response). Of the genes different between G.16 and G.87 or G.16 and G.935, bins

enriched with greater expression included photosynthesis, protein biosynthesis, RNA processing, and coenzyme modification whereas bins enriched with lesser expression were in cell wall organization and protein modification. **These profiles (Fig 3) indicate G.16 has significant gene activity relative to G.935 and G.87, which likely contributes to the reduced aphid performance**.

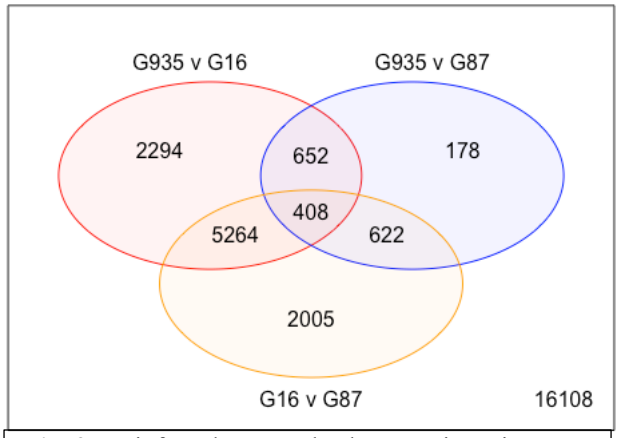

Fig. 3: Uninfested rootstocks that vary in resistance show different expression levels of many genes. Notably Er-based resistance (G.87) and enhanced gene activity (G.16) independently contribute to aphid resistance in the first days of colonization.

Of the 178 (91:up, 87:down) genes that are unique in G.87, seven immune/effector recognition (LRR/disease resistance proteins) are up and found on several chromosomes (3, 11, 2 adjacent on 13 and 3 on 15). Of the 5264 unique to G.16, **14 immune related genes are expressed more than other genotypes and further induced by aphids.** These include two disease resistance (LRR and NB-ARC proteins) found on different chromosomes (1 and 3), indicating potential loci for ER independent resistance. Furthermore, of the 210 genes that are up in G.16 but *suppressed* by aphids, 8 are related to stress response and function in protein-protein interactions. These genes may be targeted by aphid effectors to enable colonization, given how the proteasome is emerging as a novel target of galling and non-galling insects to manipulate plant function (Nabity 2016, Miao et al., 2018, Zhao et al., 2019).

G.935 and G.87 are relatively similar in gene expression, but G.935 lacks resistance and G.87 shows the Er-2 signature (Fazio & Beers 2010). This suggests the Er-based resistance may be linked to relatively few genes working together. Without these genes, however, a baseline of gene expression such as was found in G.16 is enough to reduce aphid colonization success.

Apple transcriptome undergoes remodeling shortly after colonization. Aphid feeding on apple plants remodeled the host transcriptome with a total of 1474 genes differentially expressed between all infested and control plants. This is nearly double other studies that found 637 altered genes in tomato after potato aphid feeding (Coppola et al. 2013), and ~650 DE genes in maize after corn leaf aphid feeding (Tzin et al. 2015) after 48h. In our study enriched bins included photosynthesis (85 genes), cell wall organization (85 genes), and cytoskeleton organization (23 genes), indicating these processes are perturbed more than expected compared to other processes during the first phase of colonization.

For individual genotype comparisons, G.16 responded the greatest (1858:up, 1685:down) whereas G.935 (2:up, 1:down) and G.87 (103:up, 41:down) responded less to aphids. A closer look at

the two genotypes that reduced aphid survival showed 15 genes were expressed similarly when aphids attacked (**Fig. 4**). Three of the seven suppressed by aphids were upregulated in G.16 and G.87 uninfested compared to G.935, indicating the insect may suppress these to enable feeding. These included an unknown protein, a detoxifying enzyme (cytochrome p450), and a developmental (MADs-Box) transcription factor. Five of the eight induced by aphids were suppressed in uninfested plants, indicating feeding triggered a strong induction of gene expression. These included 2 LRR disease proteins, an auxin transport P-glycoprotein, stress response ethylene forming enzyme, and a membrane stabilizing protein. Of note, the LRR genes were induced when the CA population fed but not when the WA population fed. Both LRR genes were located on chromosome 5.

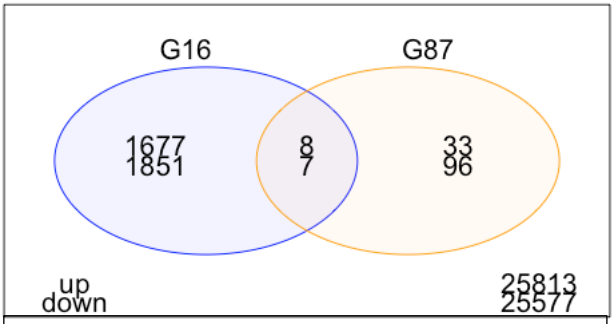

Fig. 4: Aphids feeding on G.16 induced many more genes than when feeding on G.87 where Er2 CA and WA induce different responses in the same rootstocks. 20 genes shared among rootstocks are expressed in opposite directions in CA versus WA relative to controls.

Of note, CA aphids altered rootstocks more than WA aphids but they also shared a set of genes (**Fig. 5**). Plant processes altered similarly among populations included enhanced basic stress response, suppressed photosynthesis, and enhanced cell wall organization. **Six immune related genes found across several chromosomes (1, 2, 4, 5, and 15) were up and may serve as targets to enhance apple resistance to WAA, especially for select aphid genotypes.**

Of the regionally-specific genes, WA aphids triggered two immune genes including one LRR on Chromosome 10 and increased three laccases linked to plant defense hormone signaling (Hu et al., 2018). CA aphids triggered 13 wound and immune related genes including seven LRR on chromosome 5, an R-gene on chromosome 15, and 3 disease resistance proteins on 3 different chromosomes, but only 1 laccase. Because CA aphids also suppressed a JAZ domain gene, five laccases, and seven effector-associated LRR genes, **plant defense hormone signaling may play a stronger role in plants encountering CA aphids than WA aphids**. Additional screening of aphid genotypes and different rootstocks across growing regions will help refine this hypothesis.

Twenty genes shared among rootstocks are expressed in opposite directions in CA versus WA relative to controls. These genes include one disease resistance protein that is up in CA. This differential expression confirms a role for genotype-specific secretions in altering plant response.

Several biological processes not directly functioning in immune or defense responses were altered by aphids, but why are these important? Photosynthetic downregulation is a common plant response to diverse forms of biotic stress ranging from viruses, bacteria, fungi, and arthropods (Bilgin et al. 2010), and WAA suppresses numerous photosynthesis genes. This is important because new evidence on plant perception of stress (PTI; Nomura et al., 2012, ETI; Su et al., 2018) indicates defense genes are induced by the chloroplast (through retrograde signaling) and photosynthetic inhibition is required to recognize effectors. Because WAA alters apple reactive oxygen species (ROS) profiles (Zhou et al., 2013), and breakdown in light harvesting/photosynthesis creates ROS, we predict a link between photosynthesis and ROS gene regulation during aphid attack. In support

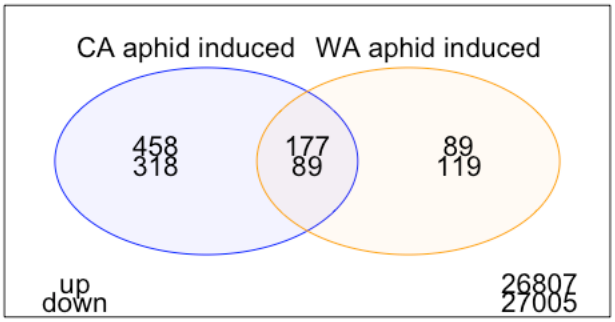

Fig. 5: 177 genes are up and 89 down in response to aphid feeding but the populations differed in how plants responded.

of this we find G.16 has more photosynthesis genes active (and up regulated) compared to G.87 or G.935 when no aphids are present and the majority of these genes are suppressed when aphids attack. This pattern indicates a role for photosynthetic inhibition in mediating WAA resistance. In contrast, G.87 had fewer photosynthesis genes (7 up and 5 down) compared to G.935, and when aphids attacked only 2 in G.87 were down. Additional study on the role of photosynthetic proteins in aphid interactions will help identify how primary growth responses like photosynthesis may provide aphid tolerance through both sustained growth and ROS-mediated protection.

Suberin is a waxy polymer that forms a barrier between the environment and living plant tissue, and functions to prevent desiccation and protect against biotic attack (Graça 2015). We identified the upregulation of 12 genes necessary for suberin synthesis and deposition after aphid infestation. Although suberization of the cell wall has been shown to be a plant response to aphid feeding (Tzin et al. 2015), and may prevent further stylet penetration, it is unclear if suberin deposition is part of the general wound healing response caused by stylet piercing, or is elicited by to benefit the galling habit of WAA. Numerous other cell wall remodeling genes are differentially expressed in a manner consistent with the construction of a new plant phenotype.

INDUSTRY BENEFIT

- Profiling constitutive expression of existing genotypes revealed disease resistance genes differentially active among rootstocks that provide similar aphid performance phenotypes.
- Additional loci outside Er genes exist that provide aphid tolerance (increased aphid mortality)
- Several immune/effector recognition genes found on several chromosomes may contribute to aphid resistance because of elevated expression without aphids.
- Comparisons among treatments revealed effector targets (genes suppressed by aphids), globally effective immune response (genes induced by aphids), and population x genotype-specific gene regulation of apple immune processes.

FUTURE DIRECTIONS

- Expand combined transcriptomic approach to more genotypes (including other species) with greater variation in resistance to identify novel genes active with and without aphids. This may best focus on where resistance is currently failing.
- Apple genotypes vary in constitutive expression of defense and immune-related genes, but other processes (e.g., photosynthesis, RNA processing, protein) have emerged as determinants of successful colonization. Thus, assessment of immune genotypes for similar profiles in non-immune, non-defense processes that indirectly provide immunity will broaden selection for tolerant genotypes.
- Continue genetic mapping to refine gene structure and sequence organization around immune genes of interest. This will aid in additional marker development.
- *New genotypes with variation in resistance await profiling*. In another survival assay, 10 genotypes were scored for performance over 6 days (**Fig. 6**). Six genotypes containing the Er-2 gene but with unknown resistance phenotypes were found to reduce survival below 40% in 4 days, with one genotype preventing colonization entirely, leading to aphid dispersal and eventual death. These new genotypes can now be revisited for further analysis to identify the traits underlying the deterrence and death of WAA.

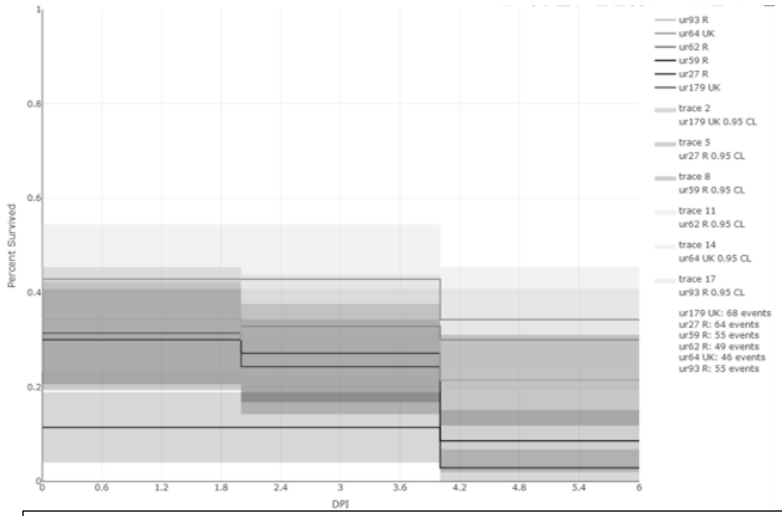

Fig. 6: Unreleased genotypes with known resistance genes were screened for aphid survival over 6 days to assess immediate immune functions. Each line represents a genotype within a confidence interval. All lines indicate low colonization (<50%) and reduced survival through time.

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EXECUTIVE SUMMARY
Project Title: Assessment of apple immune responses to wooly apple aphid saliva
KEY WORDS: insect effector, transcriptome, resistance, genome

ABSTRACT: We identified how *Eriosoma lanigerum* (WAA) colonizes apple using genome and transcriptome profiling. We found effectors enable colonization, differ among populations, and that genotype and population specific responses in apple exist. These data help track WAA resistance at local, regional, and global levels, and reveal processes underlying rootstock performance.

All plants share networks of related genes and proteins that work together to generate immune responses to both insects and pathogens. The main goal of our project was to identify these networks in apple as they relate to aphid feeding, although resolving these immune networks will inform upon any biotic stress response imposed on apple in the future. A complementary goal was to examine how the aphids trigger these networks by characterizing insect genes. For both of these goals, we trained a MS student in current bioinformatics techniques and with expertise in apple-aphid interactions. Our approach combined transcriptomic information on the apple genes induced by aphid colonization with the genes active in aphids as a first hurdle in linking insects to plants. This fundamental research will help us to better understand resistance mechanisms in apple and how insect populations vary across growing regions.

We used a robust sequencing approach to generate a high-quality genome assembly for the WAA, and predict >390 secretory effectors are used to evade immune detection and induce morphological change in apple hosts. Aphid populations from CA and WA differ in their effectors both by having different genes and also by expressing similar genes differently. Altogether, these effectors provide markers for population and genotype specific characterization of aphid performance to identify genes directly involved in colonization and better predict how future rootstocks will perform across insect populations. These effectors also provide a means to track aphid genotypes that overcome resistance and identify how this occurred.

We also profiled genotypes that varied in susceptibility/resistance and the activity of the Er-2 gene. We found that the degree of host response depends on the genotype attacked and the population attacking, but found a core set of immune genes linked to reduced aphid performance. We identify several more genes across chromosomes that are strongly upregulated during aphid attack, thus contribute to resistance, or are suppressed by aphids, thus likely are targeted by aphid effectors. We found more immune genes regulated when Er resistance was not active and also found constitutive and altered expression of non-immune and non-defense processes that can indirectly reduce aphid performance. This provides a source of non-Er-gene tolerance to aphids and a background to screen against when examining how new genotypes may perform. Altogether, these data provide a means to identify and track WAA resistance at local, regional, and global levels, and characterize why rootstocks perform the way they do given where they are grown.

Publications in preparation

- Wemmer J, Zhao C, Borowsky A, Fazio G, Nabity PD et al. Transcriptional remodeling of apple, *Malus domestica* (Borkh.) across a host resistance spectrum upon colonization by a gall-inducing aphid, *Eriosoma lanigerum* (Hausmann).
- Nabity et al. Genome sequence of the woolly apple aphid, *Eriosoma lanigerum*

MS Thesis: Wemmer, JD. 2019. Characterizing the Dual Transcriptomes of Woolly Apple Aphid, *Eriosoma lanigerum* (Hausmann), and its Host, *Malus domestica* (Borkh.), Across a Host Resistance Spectrum. University of California.