

UNMASKING ACQUISITION AND ALLOCATION VARIANCE THROUGH ENVIRONMENT MANIPULATION



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Abstract

To understand how selection affects a population, finding out more about the acquisitional and allocational components of the genetic diversity within it is essential. Studying trade-offs between life-history traits in different environments can reveal differences in the degree of acquisitional and allocational genetic variance within a population. Learning about how selection acts can give us an insight into how adaptation evolves, which is important regarding resistance development against pesticides.

My thesis aims to use *Helicoverpa armigera* moth larvae to quantify the genetic effects of life-history trait trade-offs in different environments to analyze acquisition and allocation patterns within a population. The life-history traits measured was pupal mass, pupal development rate and survival. To manipulate the environment, two different larval diets were used, one rich and one poor, as well as different doses of the microbial biopesticide *Bacillus thuringiensis*. A half-sibling design was conducted to quantify genetic correlations between the life-history traits.

The biopesticide was found to impact both phenotypic covariance and genetic correlations over different environments. Negative genetic correlations were found in the higher doses for pupal mass and development rate in a resource poor environment while in a rich environment the genetic correlations were mostly positive. This indicates that there is allocation variance revealed in high stress environments that might be masked during favorable conditions. Correlations between survival and pupal mass as well as development rate, were mostly centered around zero. Some deviations from zero in both negative and positive directions were found for survival and pupal mass in a rich environment, and survival and development rate in a poor environment. Although, the results for the maximum dose for the survival analyses can be unreliable due to a high number of deaths, there are still interesting patterns in the rest of the treatments worth expanding on.

The results show that environment has an impact when it comes to how much of the genetic variance within a population is acquisitional vs. allocational. This is especially important in the context of biopesticide usage and my thesis demonstrates the need for considering environmental impacts and not only genetic diversity when studying adaptation.

Keywords: *genotype-by-environment, resource acquisition, resource allocation, adaptation, , trade-offs, life-history, genetic variation.*

1. Introduction

One of the causes of agricultural loss is pests and they account for a substantial amount of damages to crops (Ayilara et al., 2023). Most of the methods for combating these problems uses synthetic pesticides but these have many drawbacks and can be harmful to both humans and the environment as well as having problems with resistance development in pests (Ayilara et al., 2023). One solution to these problems that have been more researched in recent years is biopesticides (Kumar et al., 2021). By being a naturally occurring compound derived from plants, animals and microorganisms, biological control can be more environmentally friendly than synthetic pesticides (Kumar et al., 2021).

1.1. *Evolution and selection*

Synthetic pesticides have simple structures that require far less genetic changes to develop resistance against compared to the complexity that biocontrol can provide (Liu et al., 2021; Tinsley et al., 2006). Studies show that by studying genetic diversity within populations you can analyze the selection and adaptation of different traits, including immunity development against pesticides, across environments (R. M. Mangan et al., 2025; van Putten, 2025). Genetic diversity and variation can be sustained in two ways: mutation repair and genotype-by-environment interactions. Mutation breaks down mechanisms for the ability to acquire resources and use them, which creates a directional selection for repair every generation. These processes are present and operates mainly the same in different environments, meaning that the environment will not have an impact on which genotypes are being favored. Recent research suggests that biopesticides can restrict this directional selection towards resistance, especially if combined with increased environmental heterogeneity (R. M. Mangan et al., 2025; van Putten, 2025). On the other hand, genotype-by-environment (hereafter GxE) interactions (section 1.2) will favor different genotypes depending on environment, leading to a fluctuating selection instead. Fluctuating selection will result in an increased genetic diversity and variation to secure the ability to adapt to different environmental changes (Bürger & Gimelfarb, 2002). However, we do not know how much of a populations' genetic variation is under directional (i.e. for acquisition of resources) or fluctuating (i.e. GxE) selection.

1.2. *Genotype-by-environment interactions and trade-offs*

GxE interactions happen when genotypes have different fitness depending on the environment one inhabits (Hunt et al., 2004). These interactions can be studied by looking at life-history trait covariance and the trade-off pattern between them (R. Mangan et al., 2023). Growth and reproduction are two traits that are common when studying these concepts (van Noordwijk & de Jong, 1986). Because they are essential for fitness and some resources have to be invested in both traits, without reproducing there is no need to grow and without growth there is no possibility of reproducing, they fundamentally trade-off. We can think of it in the context of this thesis and look into these traits for insects. Growth, regarding mass, of the insect is a result of the amount eaten and the amount of time available for eating. Reproduction, in terms of development time from hatching to pupation, can impact the exposure time to predators and the available time for eating and acquire resources (Nylin & Gotthard, 1998). This leads to a trade-off where a gain in one trait will lead to a loss in the other, meaning a short development time might be beneficial for limiting predator exposure, but then the growth period will be limited as well which will limit growth.

Some evidence shows that there is a change in trade-off pattern between these traits across various environments which is an indication that an GxE interaction is present (van Putten, 2025). This is central when researching adaptation, because how the relationship between traits changes in a population, depending on environment, can tell us something about how animals attain and distribute resources (van Noordwijk & de Jong, 1986).

1.3. *Acquisition vs. Allocation*

Life-history traits and the covariance between them will look different within a population due to how much of the variance of these traits is due to genetic variance in acquisition or allocation. Acquisition is the ability to gather and attain resources and allocation is the strategy to invest and distribute those resources, and both mechanisms will vary individually within a population (van Noordwijk & de Jong, 1986). David Reznick developed a human analogy for these concepts (Reznick et al., 2000). Acquisition and allocation patterns can be equated to how we spend our salaries on our houses and cars. We agree that resources already spent on a house cannot be spent on a car which is then considered a trade-off in spending. So, one might expect there to be clear trade-offs between houses and cars, meaning that a big house will be accompanied with a small car and vice versa. But this is not the case when looking at people's possessions, big houses are usually accompanied by big cars. This is a contradiction to our trade-off theory but can be explained by inherent difference in income. People with high income will have the ability to spend their resources in several areas, including both a big house and a big car. While people with low income cannot afford either a big house or a big car which results in both being small. This indicates that the differences in house and car size between households are caused by the ability to acquire resources. In an environment where resources are easier to obtain, genetic variance in acquisition ability, or income amount, within a population will determine the difference in house and car size between households. Additionally, in an environment where the resources are hard to obtain, the difference in acquisition ability will create even stronger differences between the households since the ones that have a high ability to acquire can get on pretty well, while the ones who do not have an as large income will have an even harder time to gather their resources.

The analogy then becomes: how would the investment patterns look if we remove the income difference between households? The thought is that when we dissolve the differences in income between households, the investment strategy of your resources will be a more important cause for variation in house and car size between households, than the ability to acquire them. For example, if we add a third cost to the households, in addition to houses and cars, we will see that differences between households is explained by the investment and distribution strategy of the income you do have and not the amount that you have. In this scenario we can imagine the third cost to be insurance for the household. That extra cost will make your resources limited and generate this trade-off pattern mentioned above, where big houses are accompanied by small cars and vice versa. And the insurance may be more or less expensive depending on how big of a risk it is that something will happen to you, with a bigger chance that something will happen the more expensive you want your insurance to be. Allocation variation is exposed when the variation in house and car size between households is explained by the difference in investment strategy.

The relationship between how one is able to acquire and one's investment strategy is what we want to study when looking at genetic variation within acquisition and allocation within a population. When we manipulate the environment and change how easy it is to attain resources or income and how expensive your insurance needs to be, we can expose these acquisition and allocation components. However, how much of a population's genetic diversity is separated into allocation vs. acquisition variation and how the relationship between them changes with a change in environment is difficult to study. For example, the allocation mechanisms that may be present when dissolving differences between households, might be masked by otherwise favorable conditions where resources are easily attainable (van Noordwijk & de Jong, 1986).

1.4. Thesis focus

In this thesis, the cotton bollworm (Lepidoptera: Noctuidae: *Helicoverpa armigera*) was used to analyze trade-off patterns in varying environments. *H. armigera* can develop resistance against chemical pesticides and it has done so for many different kinds (Haile et al., 2021). This moth is a globally spread pest and this resistance development quality poses a big challenge to our agriculture (Haile et al., 2021; Tay et al., 2013). Biopesticide research on this species may be beneficial, both as an alternative to synthetic pesticides, which has several cases of developed resistance in this moth, but also to learn more about the selection mechanisms behind resistance development (Haile et al., 2021).

This study will focus on how *H. armigera* reacts to different environments. Resource limitation in the form of diet nutrient manipulation together with pathogen exposure from a biopesticide, *Bacillus thuringiensis*, will be used to simulate different environments. Two diets were implemented, one rich diet, considered to be a nutrition dense and easily attained resource and one poor diet, constricting nutrient availability. Pathogen exposure will be done through different doses ranging from a control up to the manufacturers' recommended full dose. The traits that will be measured are survival, growth and reproduction. The survival is measured as a binomial observation of alive or dead, growth as pupal mass and reproduction as development time, as the time from hatching to pupation.

For this experiment diet and pathogen exposure is used to expose allocation and acquisition variance within the population across different environments. The poor diet is predicted to reveal more acquisition variance than the rich diet counterpart in a specific dose. The nutrition limitation is thought to create an environment where the ability to acquire resources is a bigger cause for the trade-off variation within the population. The life-history trait covariances as well as the genetic correlations are predicted to be presented as the example in Figure 1a, more positive within a poor diet than in the rich diet, regardless of dose exposure. However, the pathogen dose is expected to reveal allocation variance. An increase in dose should give the same effect as the insurance cost or dissolving of income differences between households and therefore create an environment where strategy and allocation mechanisms are a bigger cause for the trade-off variation, compared to lower doses within the same diet. The anticipated trade-offs and correlations are therefore considered to show a more negative relationship when increasing pathogen exposure, see Figure 1b.

The aspirations of this research are to reveal trade-off patterns within *H. armigera* and get more insight into the acquisition and allocation variance across different environments. Studying the genetic correlations between survival, pupal mass and development time across different environments may give us an understanding of how adaptation and resistance development processes looks.

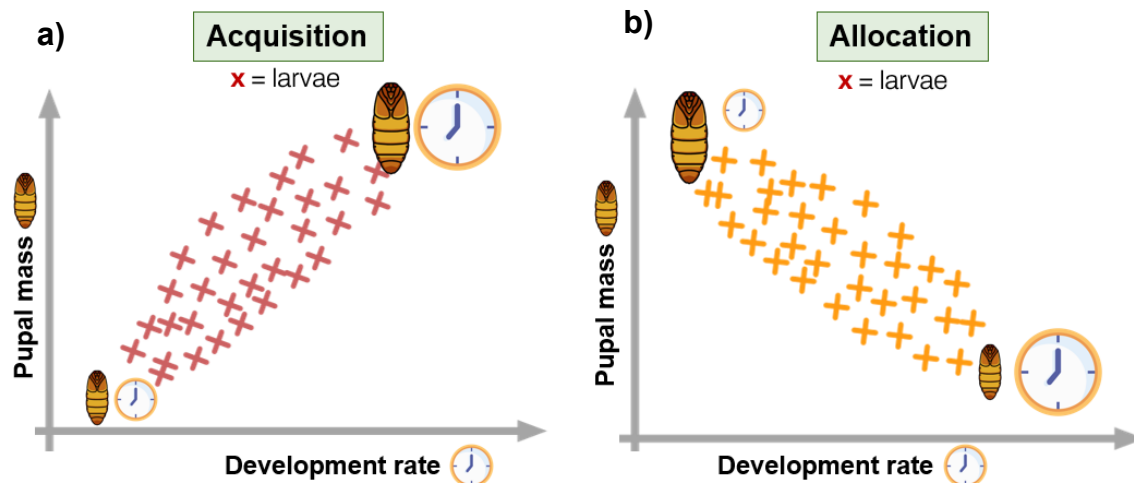


Figure 1: Illustration of acquisition and allocation variance within an *H. armigera* population. The distribution shows the covariance between pupal mass and development rate as an example of life-history trait covariance. A) shows a positive covariance between pupal mass and development rate, indicating that acquisition variance within the population is exposed. B) shows a negative covariance between pupal mass and development rate, indicating that allocation variance within the population is exposed.

2. Aim

The purpose of this thesis is to study trade-off patterns of survival, pupal mass and development rate in different environments by manipulating resource availability and pathogen exposure by a biopesticide BT. The aim is to expose potential genetic variance within acquisition and allocation mechanisms in the global pest cotton bollworm (*H. armigera*). My objectives are:

1. How does resource availability affect acquisition and allocation variation within *H. armigera*, when looking at genetic correlations between life-history traits?
2. How does a change in pathogen exposure affect acquisition and allocation variation within *H. armigera*, when looking at genetic correlations between life-history traits?

I predict that we will see more positive correlations between traits when looking at a poor diet compared to a rich diet, revealing acquisition variance. A more negative correlation is expected when looking at pathogen exposure, revealing allocation variance. A gradual increase in strength of the correlation is also predicted with an increase in pathogen dose. This prediction is for all three life-history trait combinations: survival and development rate, survival and pupal mass, pupal mass and development rate.

3. Material and method

3.1. Experimental system

3.1.1. Moths

Adult moths used for matings were ordered from Andermatt Biocontrol AG (Switzerland) in two shipments one week apart. Pupae were separated into the different sexes and put in plastic boxes with 8-12 pupae each. While awaiting eclosions, all pupae were stored in climate cabinets under reversed photoperiod conditions (14h light: 10h dark, with the dark period between 8.00 and 18.00) and 75% relative humidity. Eclosed adults were separated into isolated vials with a moistened cotton ball at the bottom with added adult diet. All adults were kept under these conditions: 14h light at 25 °C, 10h dark at 16°C, with dark period between 8.00 and 18.00, at 75% relative humidity.

3.1.2. Biopesticide

The pathogen used was *Bacillus thuringiensis* (BT). BT is a bacterium used to make biopesticides and it has been heavily researched world-wide (Ragasruthi et al., 2024). The brand used was NaturePro. The solutions used were prepared the day of inoculation. The highest dose was prepared according to the manufacturer's instructions, while the rest were four-fold serial dilutions from the original solution. The control dose used was water.

3.2. Experimental design

3.2.1. Diet

Adult diet was a liquid solution, as seen in Table 1 and Table 2 (Le Senecha et al., 2017). The diet was mixed in a bottle and kept in the fridge to keep cool and was protected from light. Feedings were done as needed as well as during matings and when separating the mated dams. The adult diet was refreshed whenever the cotton wool seemed dry or contaminated.

In the experiment, two different larval diets were used. One of the diets were considered rich and the other poor. The rich diet was designed to be an optimal, easily attainable diet, as seen in Table 3 (Le Senecha et al., 2017). The poor diet was designed to have less nutrition and not have as much nutrition as the other. To get the difference in nutrition the poor diet had half as much white beans as the rich, which was replaced with indigestible cellulose (Table 3).

Table 1: Ingredients for vitamin solution used in adult diet.

Vitamin solution	
Ingredient	Amount (g)
Niacinamide (Vitamin B3)	1
Calcium Pantothenate (Vitamin B5)	1
Riboflavin (Vitamin B2)	0.5
Thiamine (B1)	0.25
Pyridoxine (Vitamin B6)	0.25
Folic Acid (Vitamin B9)	0.1
Biotin (Vitamin B7)	0.00002
Cobalamin (Vitamin B12)	2
Distilled water	1000

Table 2: Ingredients for adult diet.

Adult diet	
Ingredient	Amount
Distilled water	1,000 g
Sucrose	100 g
Vitamin solution	20 mL
100% ethanol	2 mL

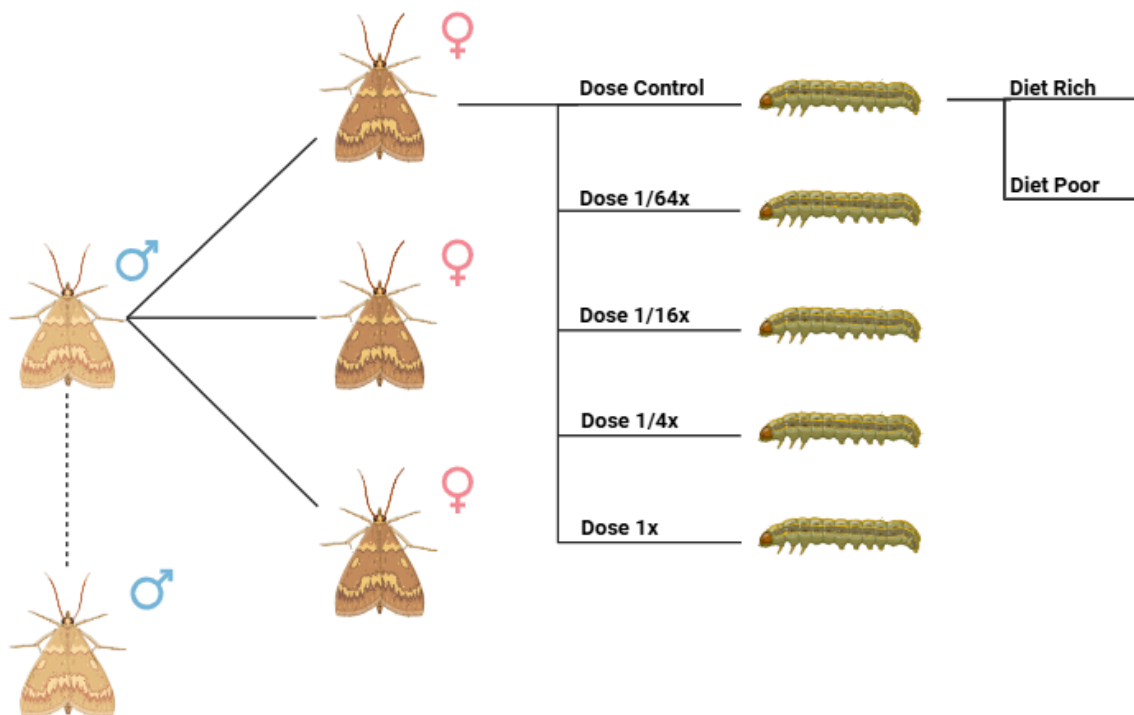
Table 3: Ingredients for the two larval diets.

Larval diets		
Ingredient	Amount (1 batch)	
	Rich	Poor
White bean	51.45 g	27.525 g
Cellulose	0 g	27.525 g
Agar	9.45 g	9.45 g
Ascorbic Acid	2.45 g	2.45 g
Wheat Germ	41.15 g	41.15 g
Soy protein	20.6 g	20.6 g
Casein	15.45 g	15.45 g
Brewer's yeast	25.7 g	25.7 g
Sorbic acid	1.25 g	1.25 g
Vitamin solution	4.1 mL	4.1 mL
Ethanol	2.45 mL	2.45 mL
Distilled water	600 mL	600 mL

3.2.2. Mating set-up

This experiment used a half-sibling design to be able to partition genetic variance for the different traits measured (Falconer, 1996). Each sire was mated with up to three dams (Figure 2). Sires were 2-4 days old and dams were 1-2 days old. A dam and a sire were selected at random, avoiding visibly malformed individuals, and put into a mating box for approximately 1 hour in the dark. A tissue was placed in the mating box to provide a textured surface for individuals to hold onto during matings as well as a cotton ball with adult diet. If mating was successful, they were kept until released from one another.

Sires that mated successfully were kept for a one-day recovery period and prioritized during the next days matings. Dams that mated successfully were put in smaller maternity boxes with a new tissue to lay eggs on for collection as well as new adult diet. Unsuccessful mating pairings were separated after 1 hour and the dam was replaced for a new pairing.



Created in BioRender.com 

Figure 2: Mating set-up and treatment groups. One sire was mated with up to three dams. The full sibling families were then subjected to five different pathogen doses together with either rich or poor larval diet. Inspirations from (van Putten, 2025). Figure created with <https://www.biorender.com/>

3.2.3. Larvae handling

Hatched larvae were fed poor diet before inoculation. Inoculations were done during the second instar and individual larvae was put into vials with the chosen treatment. The treatments were made up of one of the two diets and one of the different doses (Figure 2). Full sibling families were assigned a tray with 100 inoculation tubes each. Treatments were ordered in the trays randomly five tubes at a time, working all treatments once before repicking the first treatment again. The biopesticide solution was applied by spraying it into the vial onto the diet before the larvae were placed into it with a brush. The brush was sterilized with 70% ethanol between treatments. Individual larvae were haphazardly put into the inoculation tubes.

The vials were then checked for mortality and pupation each day. Pupae were cleaned, weighed and sexed within a couple of days after pupation. Dates for mortality and pupation were recorded for the survival and developmental rate traits.

3.3. Statistical analysis

All data has been analyzed using R version 4.6.0 (R Core Team, 2026). The pupal mass and development rate parameters were standardized to be comparable and to improve variation estimation. Pupal mass was separated by sex when looking at phenotypic results. Development rate was inverted by dividing 1 with the number of days between hatching and pupation to make it scale positively with fitness, with faster development rates represented by higher numbers. Survival was interpreted as a binomial response variable.

A Bayesian multivariate model was used to calculate genetic correlations between the life-history traits. The Bayesian model is ideal for multivariate analyses and estimation with a lot of uncertainty, that the levels of treatment combinations and genetic correlations usually generate.

Sub models for each trait were built using a combined variable of dose and diet, called treatment, as a fixed effect and sire and dam as random effects. The sire effects were set to vary with treatment while dam effects were not. The reasoning behind doing it this way is that the maternal effects are considered to uniformly contribute to offspring fitness regardless of treatment (R. M. Mangan et al., 2025).

These sub models were then used in three Bayesian multivariate models, calculating the genetic correlation between traits. The three models were computed with the brms package (Bürkner, 2021). The models were set up to focus on the correlation between two traits each. The traits for each model were as follows: survival and development rate, survival and pupal mass, pupal mass and development rate. From each big model, posterior distributions for genetic correlations between traits could be analyzed for specific treatments. The results were plotted using ggplot2 within the tidyverse package (Wickham et al., 2019; Wilke, 2025). Density ridge plots were used to plot posteriors and illustrate their uncertainty.

3.3.1. Generative AI disclosure

AI was used through Perplexity and focused primarily on code improvement for plotting aesthetics as well as some core concept understanding. No AI has been contributing to any written text or result production.

4. Results

The dataset included 2171 individual larvae and all groupings that were relevant had all a comparable number of individuals (Table 4). Two of the larvae that developed into pupae were not able to be sexed but since this was not a significant number of individuals they were included in the final model. The number of sexed individuals in Table 4 only includes the ones that survived until pupation, since they cannot be sexed during the larval stage. Individuals that died either during the larval phase or the pupation phase is included in the total death count. Genetic correlation analyses that included pupal mass as one of the traits were made with the intact dataset not separated into sex.

Table 4: Data summary. The number of individuals within each identity or treatment group for both adults and larvae observed in the experiment.

Data summary	
Group	Number of individuals
Sires	13
Dams	23
Larvae (total)	2171
Dose: Control	431
Dose: 1/64x	435
Dose: 1/16x	435
Dose: 1/4x	430
Dose: 1x	440
Diet: Rich	1086
Diet: Poor	1085
Female	733
Male	797
Unspecified sex	2
Dead	738

As seen in Figure 3, survival is affected by the dose and the diet. The proportion that survived goes down with increased dose. There is little difference between the two lowest doses, Control and 1/64x, and a bigger difference between the two highest doses, 1/4x and 1x, where the survival goes down close to zero in the 1x dose. The different diets did not have much effect on the survival in the different doses but there is some noticeable difference in the 1/64x and 1/16x dose, with the poor diet showing a higher proportion of survival than the rich diet in both cases.

Development rate was affected similarly to the survival. Figure 4 shows a decrease in development rate with increased dose. For development rate the diet does have an effect within doses. The difference between diets in dose Control, 1/64x, 1/16x and 1x is quite noticeable with the three lowest doses having a slower developmental rate in the poor diet and the 1x dose having the slower development rate in the rich diet. The 1/4x dose also have, like the lower doses, a slower development rate in the poor diet, although the difference between diets is smaller.

The pupal mass for females and males is bigger in the rich diet compared to the poor diet within all doses (Figure 5). The lower doses report the smallest mass for both sexes while the 1x dose for females and the 1/4x dose for males report the biggest.

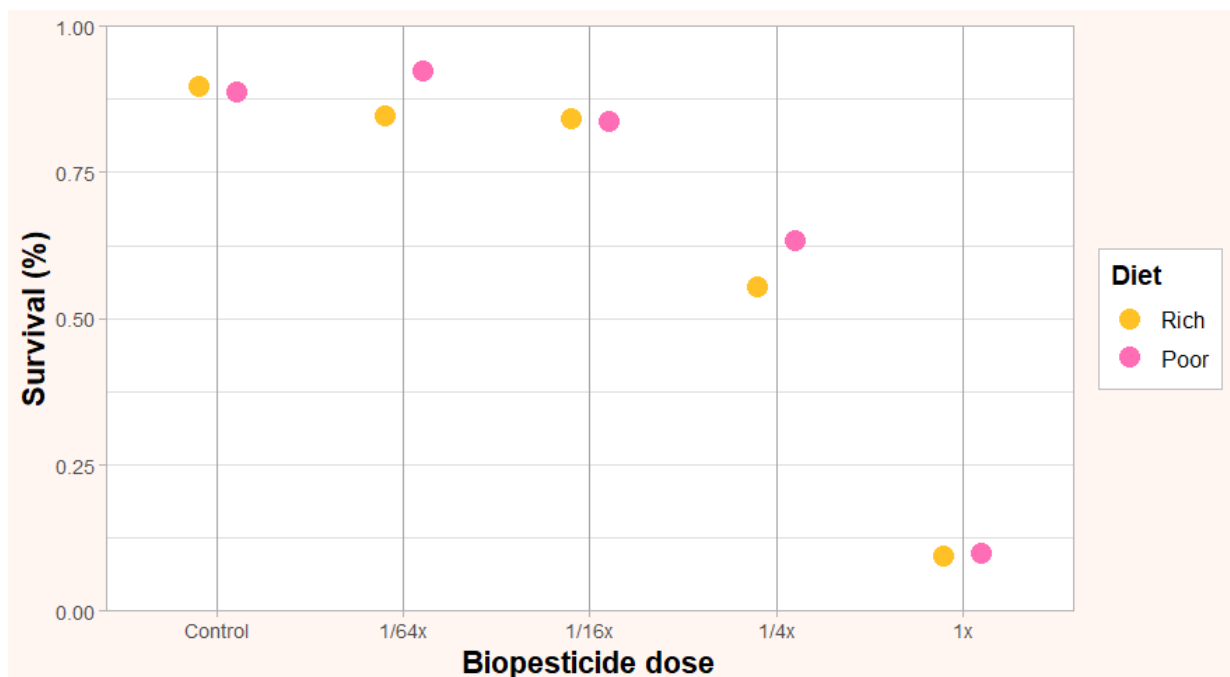


Figure 3: Survival within each treatment. Yellow points represent the percent survived in the rich diet corresponding to each pathogen dose. The pink points represent the percentage survived in the poor diet corresponding to each pathogen dose.

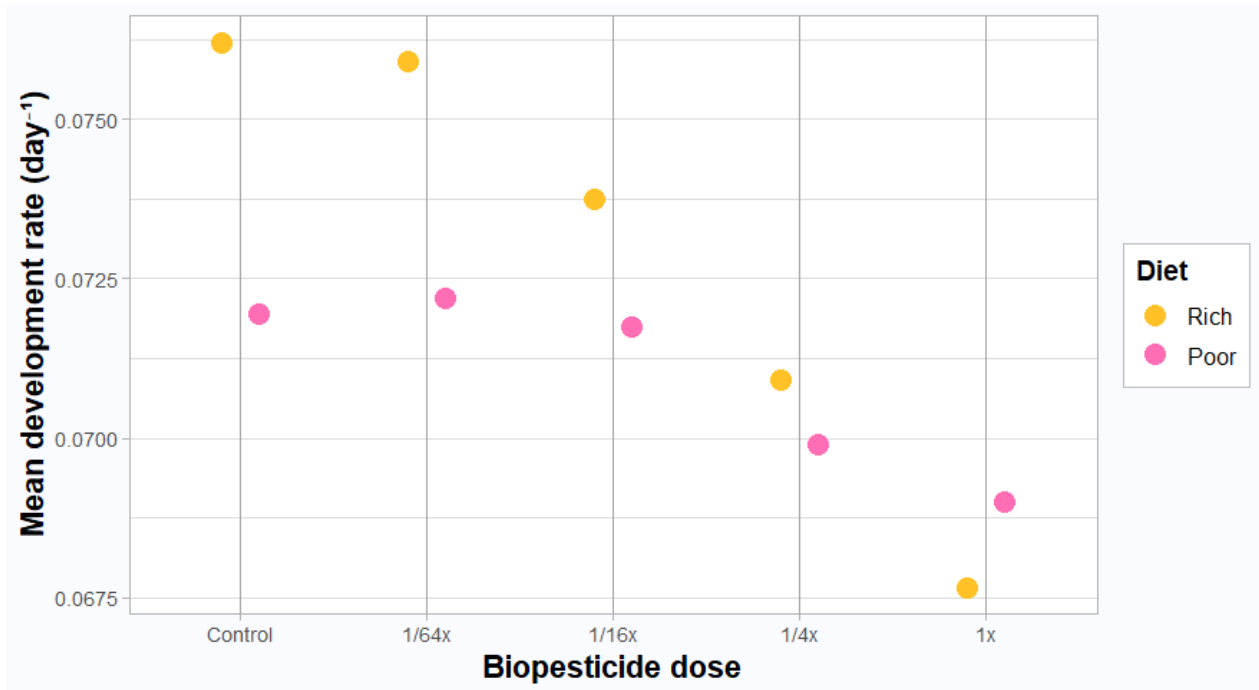


Figure 4: Mean development rate within each treatment. Yellow points represent the mean development rate in the rich diet corresponding to each pathogen dose. Pink points represent the mean development rate in the poor diet corresponding to each pathogen dose.

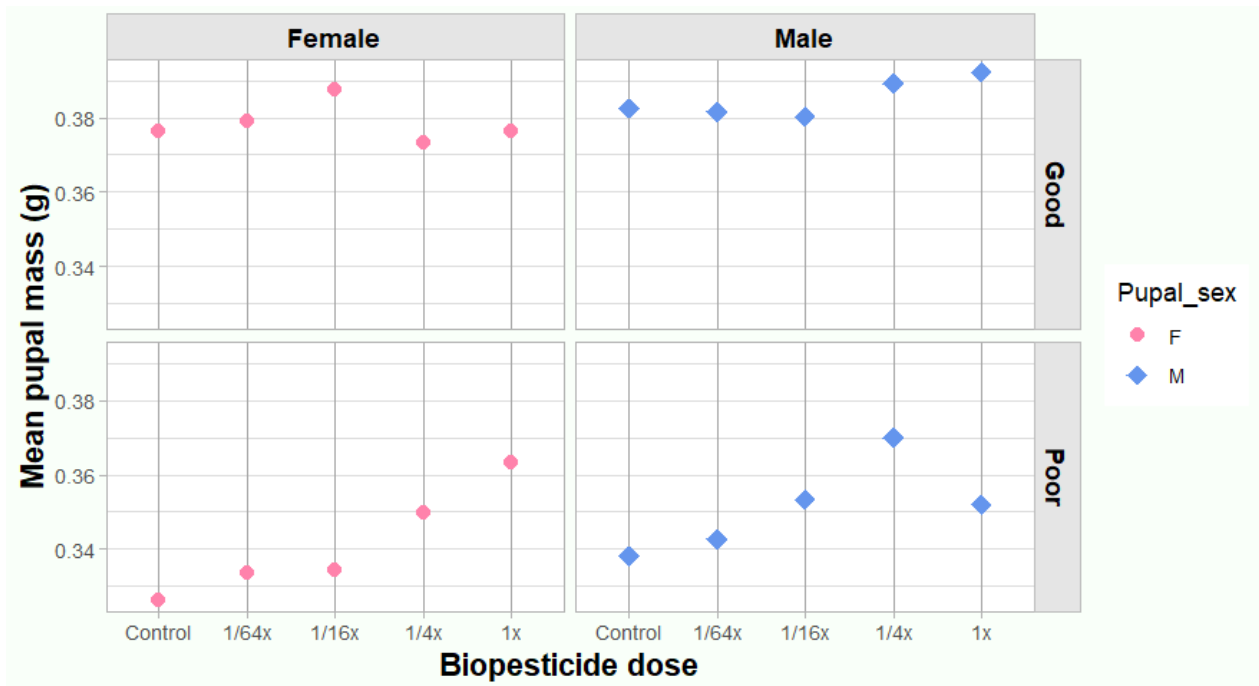


Figure 5: Mean pupal mass within each treatment. Yellow points represent the mean pupal mass (g) in the rich diet corresponding to each pathogen dose. Pink points represent the mean pupal mass (g) in the poor diet corresponding to each pathogen dose.

4.1. Phenotypic covariance between life-history traits

The covariances between the phenotypic traits are represented as family means. One point in the plot represents the mean value for pupal mass and development rate for all offspring related to one sire, generating 13 points. The phenotypic plots portray the population variance within traits based on sire affiliation.

4.1.1. Survival and Development rate

Covariances between survival and development rate suggests mostly weak positive covariance across treatments (Figure 6). There is some stronger covariances in 1/4x, 1x and Control, with the first being positive. Both diets in the 1x dose show negative covariance as well as the poor diet in the Control dose. The negative covariances show a survival goes up with decreased development rate, meaning that if they survive better, they take longer to reproduce.

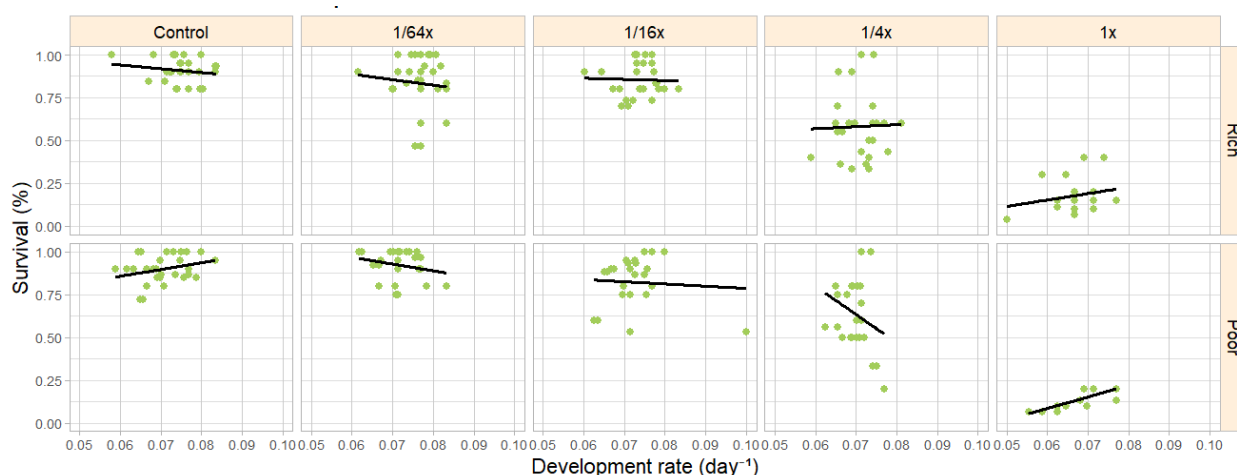


Figure 6: Survival and development covariance. Each point represents the mean survival and mean development rate for all offspring from one sire. A regression line was fitted to more easily interpret the covariance. The data is faceted into treatments with columns representing different biopesticide doses and rows representing different diets.

4.1.2. Survival and Pupal mass

For survival and pupal mass covariances we see that they are weaker than the pupal mass and development rate covariances for both females and males (Figure 7, Figure 8). For females there is stronger negative covariances for dose 1/4x in the rich diet and the 1/16x dose in the poor diet, as well as some negative indication for 1x dose in rich diet. Positive covariances are found in 1/64x dose and 1x dose, both in the poor diet. For males, there is some positive covariance for 1/4x dose in both diets while negative covariances are found for the 1x and 1/16x dose, in the rich and poor diet respectively. The negative covariances show that there is higher survival when having smaller mass within the population.

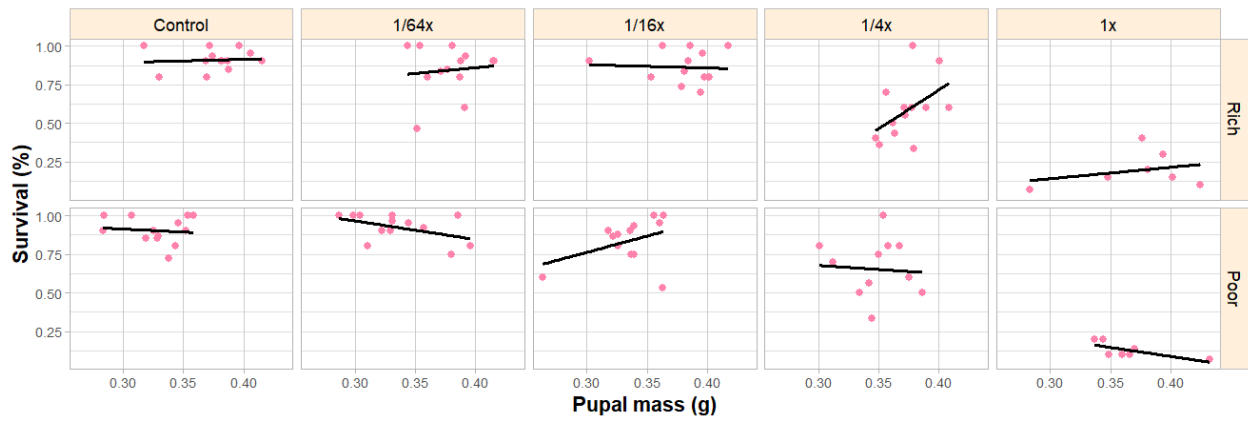


Figure 7: Female survival and pupal mass covariance. Each point represents the mean survival and mean pupal mass for female offspring from one sire. A regression line was fitted to more easily interpret the covariance. The data is faceted into treatments with columns representing different biopesticide doses and rows representing different diets.

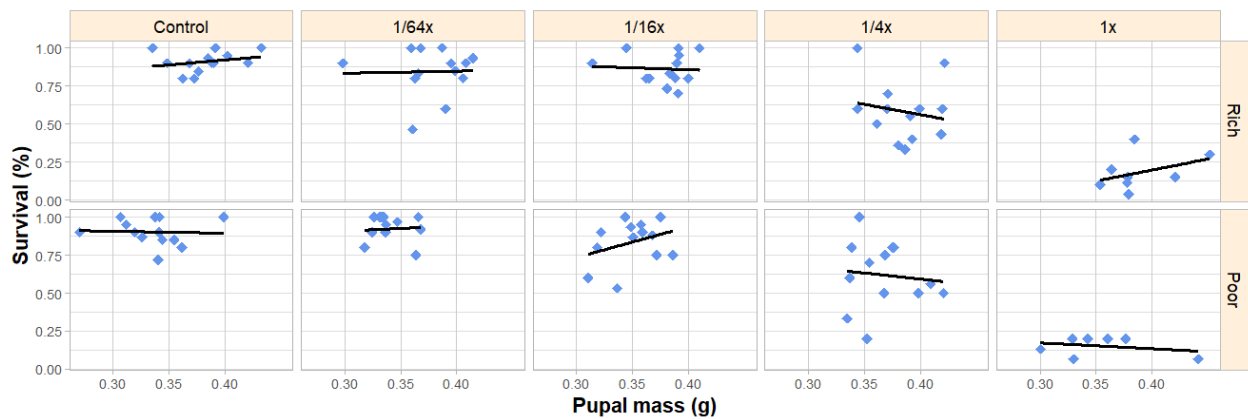


Figure 8: Male survival and pupal mass covariance. Each point represents the mean survival and mean pupal mass for male offspring from one sire. A regression line was fitted to more easily interpret the covariance. The data is faceted into treatments with columns representing different biopesticide doses and rows representing different diets.

4.1.3. Pupal mass and Development rate

There is a positive covariance between pupal mass and development rate for females in both diets for the Control, 1/16x and 1/4x dose, although, there is a weak slope for 1/4x in the poor diet (Figure 9). In the 1/64x dose and poor diet there is a positive covariance as well, meaning that for these treatments there is a higher development rate with bigger mass. For the rest, 1/64x in rich diet and in both diets for 1x dose, there is a negative covariance between the traits, meaning that they do not grow as much when reproducing faster and vice versa, within the population.

For males (Figure 10), there is a positive covariance in both diets for the 1/16x and 1x dose, as well as for dose 1/64x in a poor diet, although, there is a weaker slope for both 1/64x in poor diet and 1x in rich diet. In the Control and 1/16x dose the covariance is negative between the two traits, in contrast to the results for females, as well as the 1/64x dose in rich diet.

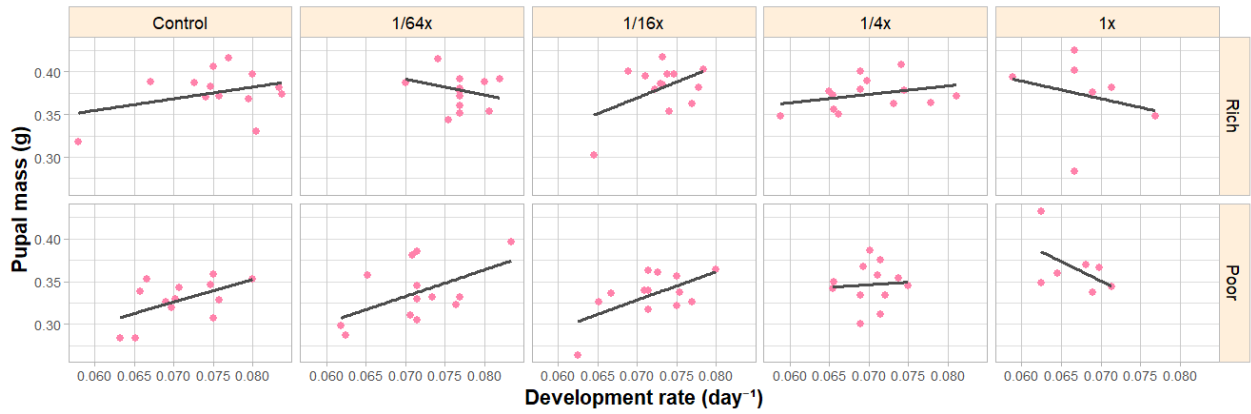


Figure 9: Female pupal mass and development rate covariance. Each point represents the mean pupal mass and mean development rate for female offspring from one sire. A regression line was fitted to more easily interpret the covariance. The data is faceted into treatments with columns representing different biopesticide doses and rows representing different diets.

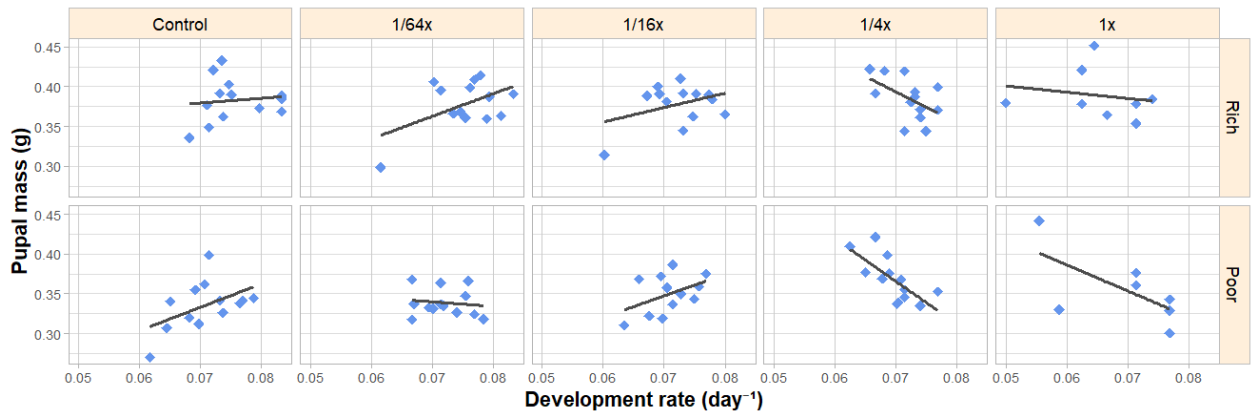


Figure 10: Male pupal mass and development rate covariance. Each point represents the mean pupal mass and mean development rate for male offspring from one sire. A regression line was fitted to more easily interpret the covariance. The data is faceted into treatments with columns representing different biopesticide doses and rows representing different diets.

4.2. Genetic correlations for each dose in different diets

The genetic correlations between traits were analyzed using Bayesian posterior distribution, illustrated as a possibility density function (PDF). The posterior distribution is produced by multiplying preconceived notions about the unknown parameter, in this case genetic correlations, with the likelihood of the observed data, in this case the observations from our experiment (McElreath, 2020). The preconceived notion about our genetic correlations is presented as a PDF as well and is called a prior distribution. The prior PDF is centered around zero, meaning no genetic correlation between traits. The prior distribution is then updated by our provided data and generates a posterior distribution.

The posterior illustrates how frequent predicted values of genetic correlations are, meaning that the highest maximum point, the mode, portrays the most reoccurring value of all predicted values for the genetic correlation based on the data. The mode is an estimate of the most probable value for the genetic correlation between two traits in a specific environment. The distribution variance of the posterior density illustrates the uncertainty around the estimate.

4.2.1. Survival and Development rate

The posterior distributions (Figure 11) show the genetic correlation between survival and development rate for all doses separated into the two diets. Looking at one distribution, we see the possibility distribution of the genetic correlation between survival and development rate within one of the doses and one of the diets. For example, the distribution for the 1/4x dose in a poor diet, we see that the mode is positive and more of the area of the distribution is on the positive side. This indicates that the genetic correlation for survival and development rate is predicted to be positive in this treatment.

Now consider the effect that pathogen dose has on the difference between distributions within one of the diets (Figure 11). In the rich diet we find that there are small to no genetic correlation shifts between the different doses. There is not gradual cline in any direction when considering all of the distributions within the rich diet. The same is found when looking at the poor diet. There seems to be no consistent shifts in any direction when increasing dose (Figure 11). Some evidence for a positive shift is found in the 1/4x dose as stated above, but this is weak.

When looking at only how diet impacts the correlation shifts within each of the doses, little evidence is found with exception to the 1/4x dose where there is a negative shift from the rich to the poor diet, although, this is also very weak (Figure 12). This exception does not indicate any consistent shift in correlation due to a change in diet.

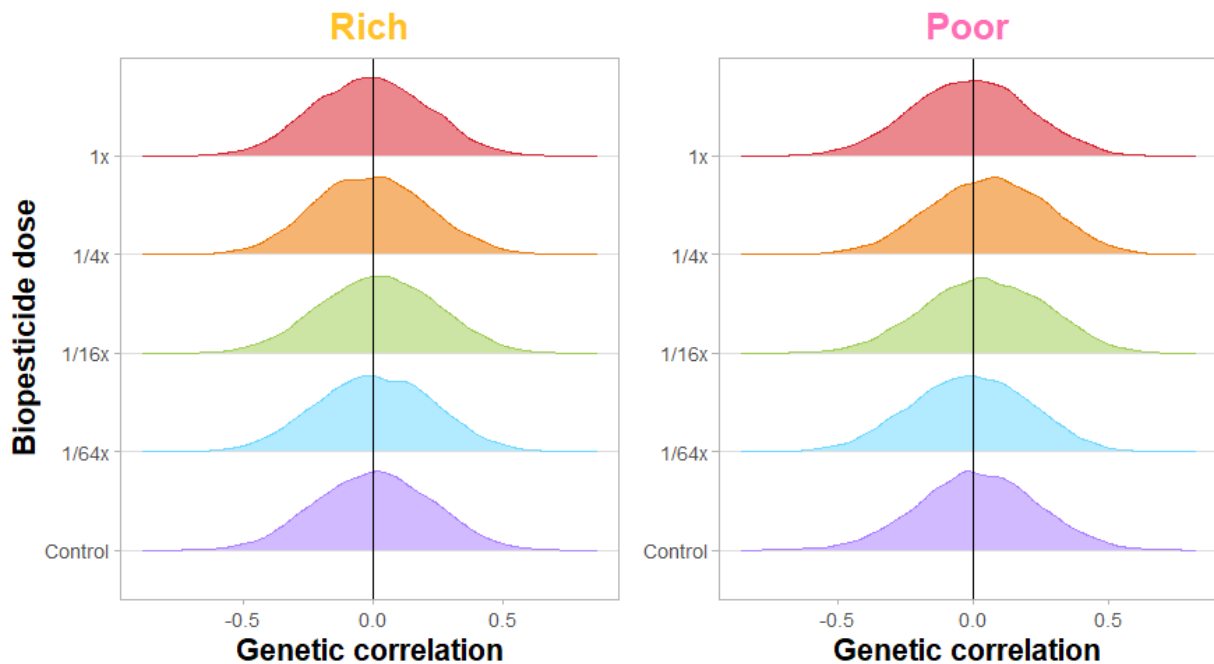


Figure 11: Differences in posterior distributions of genetic correlations, between survival and development rate between pathogen doses. The x-axis shows the genetic correlation between survival and development rate, and the y-axis shows the pathogen doses. The left

panel shows the correlations for each dose within the rich diet. The right panel shows the correlations for each dose within the poor diet.

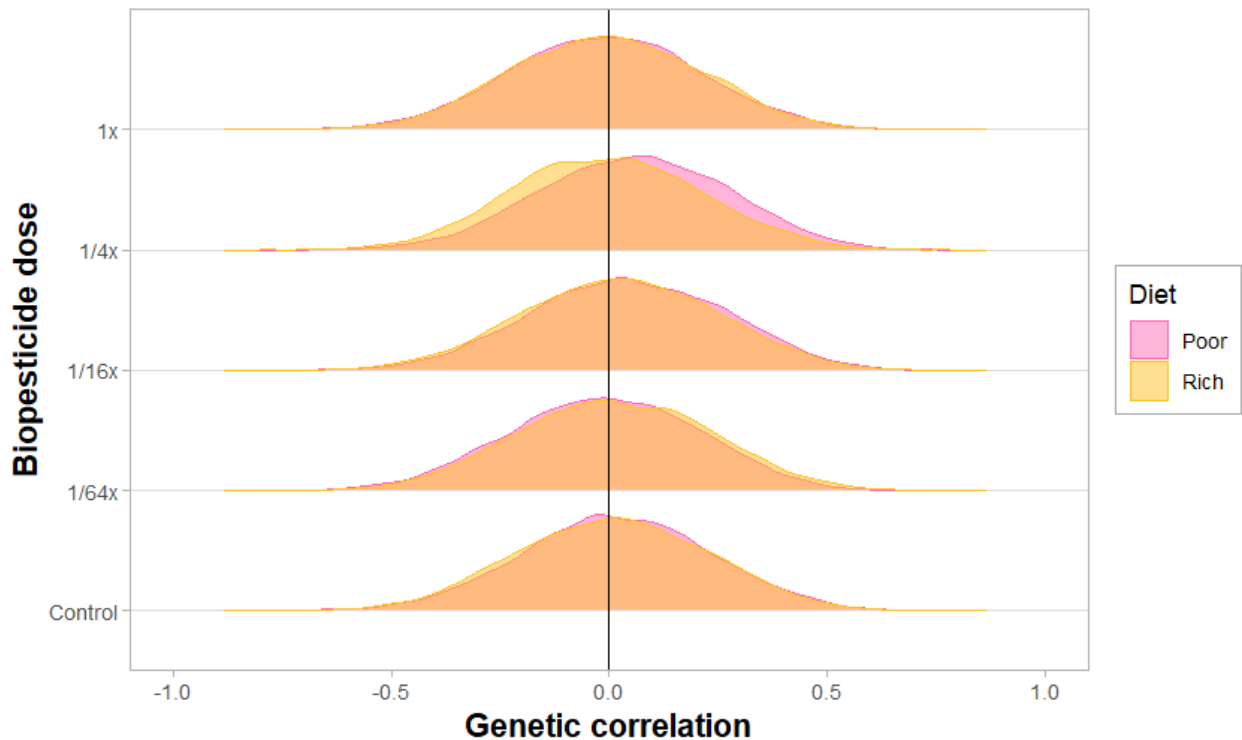


Figure 12: Differences in posterior distributions of genetic correlations, between survival and development rate, between rich and poor diet. The x-axis shows genetic correlation between survival and development rate, and the y-axis shows the pathogen dose. The yellow distributions show the correlation in a rich diet. The pink distributions show the correlation in a poor diet.

4.2.2. Survival and Pupal mass

Looking at the dose effects of survival and pupal mass correlations we also see little to no evidence that it impacts their genetic correlations (Figure 13). In the rich diet there is some indication for a positive shift from control to 1/64x dose. The rest of the doses have distributions centered around zero and there is no gradual cline presented with increased dose in any direction. The same is found for poor diet. The distributions are centered around zero and the dose does not seem to have an impact on the genetic correlation between survival and pupal mass.

When focusing on how only diet affects the correlations within doses, differences between diets are not found, the same result as for the diet effect on survival and development. Rate (Figure 14). There is some indication that within the 1/64x and 1/4x dose, the distributions negatively shift when going from rich to a poor one.

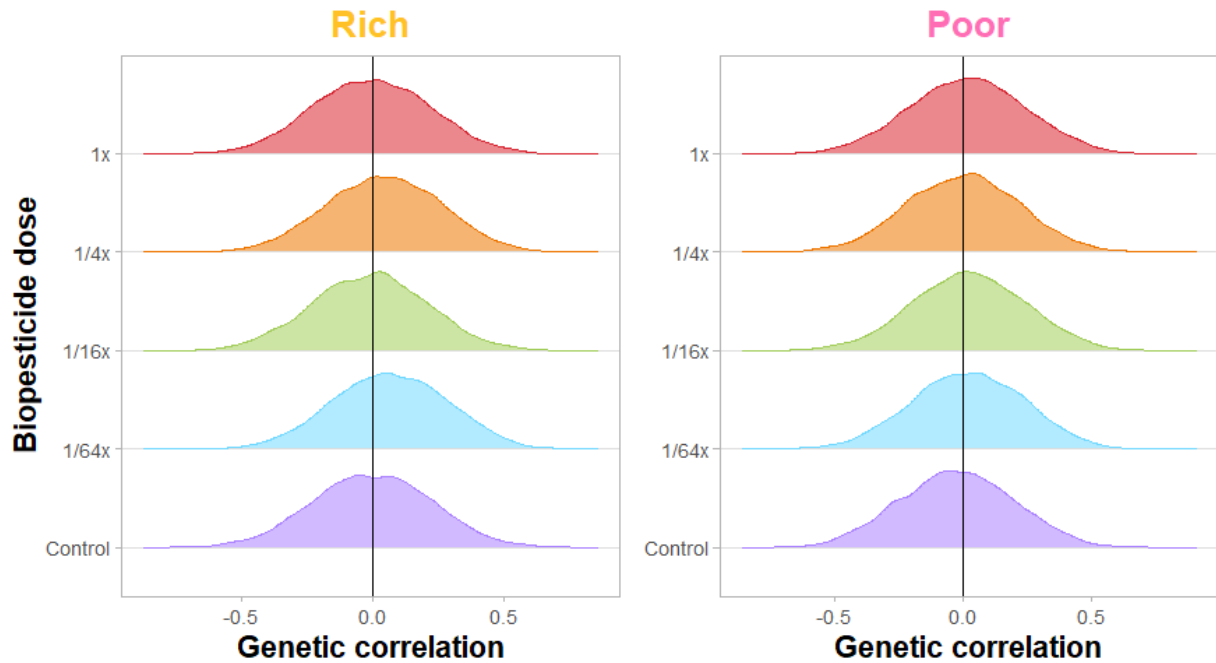


Figure 13: Differences in posterior distributions of genetic correlations, between survival and pupal mass, between pathogen doses. The x-axis shows the genetic correlation between survival and pupal mass, and the y-axis shows the pathogen doses. The left panel shows the correlations for each dose within the rich diet. The right panel shows the correlations for each dose within the poor diet.

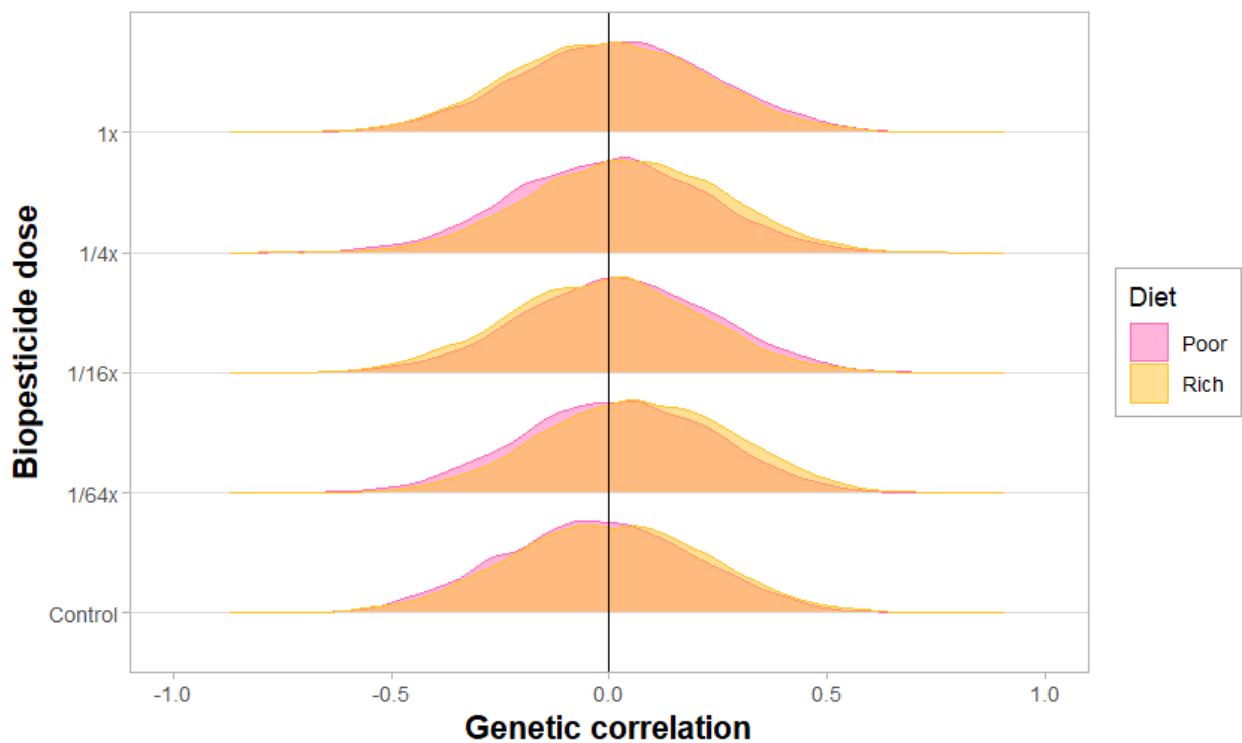


Figure 14: Differences in posterior distributions of genetic correlations, between survival and pupal mass, between rich and poor diet. The x-axis shows genetic correlation between

survival and pupal mass, and the y-axis shows the pathogen dose. The yellow distributions show the correlation in a rich diet. The pink distributions show the correlation in a poor diet.

4.2.3. Pupal mass and Development rate

The dose effects for pupal mass and development rate do, in contrast to the other trait correlations, have an impact in certain environments. In the rich diet there is some modal shift towards a more positive distribution in the 1/16x and 1x dose compared to the Control, while 1/64x and 1/4x dose seem to center around zero, close to the control (Figure 15). However, the differences compared to the control are not very strong. Contrary to the rich diet, in the poor diet we see that with an increasing dose there is a negative modal shift for most of the doses (Figure 15). Compared to the control, dose 1/64x, 1/4x and 1x, have modes and distribution shifts towards a more negative correlation. There is also a greater difference between doses that are far apart in concentration, meaning that there is a stronger shift from control to 1x dose than there is from control to 1/64x dose. A deviation from this is within the 1/16x dose where the correlation is similar to or slightly more positive than the control. Variance is similar across all posterior distributions, meaning they have similar uncertainty around the predicted modal estimate for the genetic correlation between pupal mass and development rate.

Focusing on the effect of diet on the genetic correlation within each dose we can see that there is little difference between the distributions (Figure 16). The effect of diet is strongest in the Control and 1x dose. The distributions representing the poor diet (pink) is more negative than the one representing a rich diet (yellow) in the 1x dose, while the opposite is true for the Control dose where the poor diet is more positive than the rich diet. Dose 1/16x has the same pattern as the Control, but the effect is smaller. The 1/4x dose has a similar effect strength as the 1/16x dose except it follows the 1x dose pattern rather than the Control pattern for the genetic correlations. The effect of diet in the 1/64x dose is faint to none.

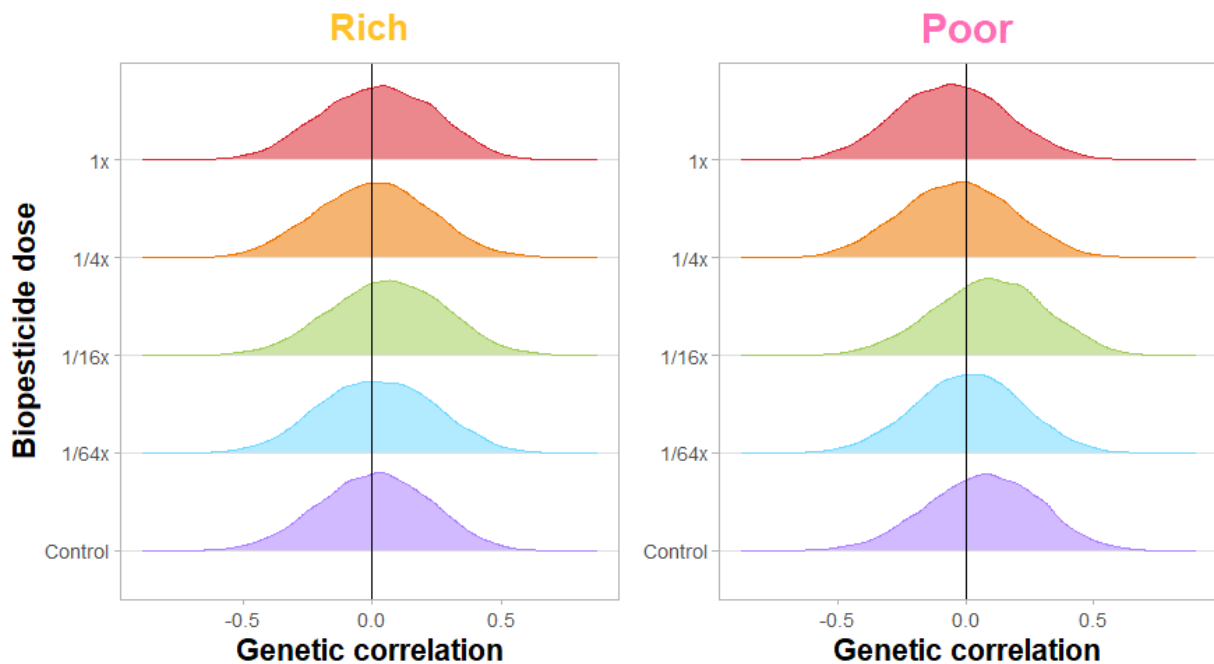


Figure 15: Differences in posterior distributions of genetic correlations, between pupal mass and development rate, between pathogen doses. The x-axis shows the genetic correlation between pupal mass and development rate, and the y-axis shows the pathogen

doses. The left panel shows the correlations for each dose within the rich diet. The right panel shows the correlations for each dose within the poor diet.

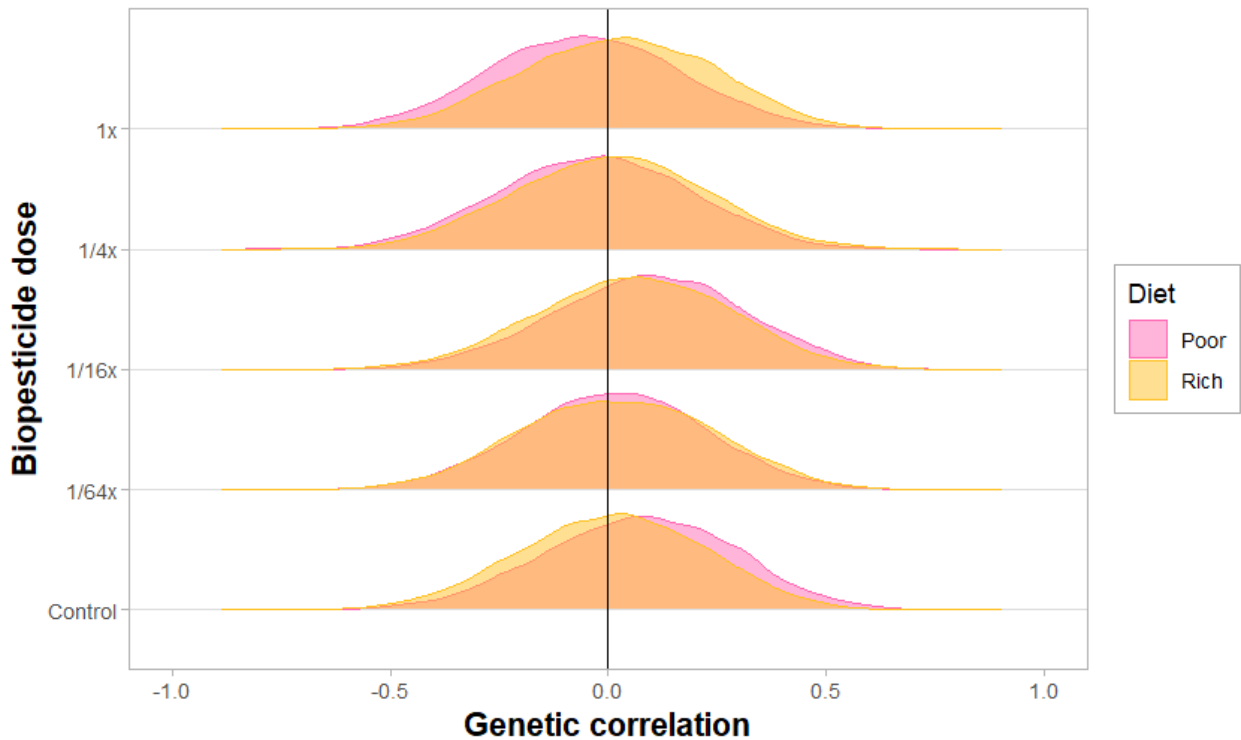


Figure 16: Differences in posterior distributions of genetic correlations, between pupal mass and development rate, between rich and poor diet. The x-axis shows genetic correlation between pupal mass and development rate, and the y-axis shows the pathogen dose. The yellow distributions show the correlation in a rich diet. The pink distributions show the correlation in a poor diet.

5. Discussion

The aim of this thesis was to reveal acquisition and allocation variance within *H. armigera*, through analyzing genetic correlations between three life-history traits: survival, pupal mass and development rate. I specifically asked: (1) How does resource availability affect acquisition and allocation variation within *H. armigera*, when looking at genetic correlations between life-history traits? (2) How does a change in pathogen exposure affect acquisition and allocation variation within *H. armigera*, when looking at genetic correlations between life-history traits?

From my results I could not find any change in genetic correlation when going from a rich diet to a poor diet. There were some indications for a shift in specific environments, for example in the 1/4x dose with a positive shift from rich diet to poor diet between survival and development rate, but these were quite weak (Figure 12). The predicted outcome when moving from a nutrient rich environment to a nutrient poor environment was that acquisition variance would be revealed due to acquiring ability being responsible for the trait variance when resources get harder to obtain. I conclude that there is little evidence that diet itself impacts the genetic correlation between any combination of life-history traits when looking within each dose (Figure 12, 14, 16) and thus does not expose any acquisition variance in this experiment. However, as stated, there were some positive shifts from rich to a poor diet within doses which might be worth studying further. That said, a nutrient poor environment did reveal a dose effect on genetic correlations, see in paragraph below.

Where we could find the most evidence for pathogen exposure effect on genetic correlation was between pupal mass and development rate (Figure 15). In the rich diet, all the distributions presented for each dose centered around zero, meaning when looking at the collective distributions there seems to be no gradual cline with increasing dose. There is some indication for a positive shift within the 1/16x dose which might point towards acquisition variance exposure, but the extent of this shift is not substantial when comparing with the control. Though, it could be interesting to look more into how the acquisition and allocation components are expressed during moderate pathogen exposure in a resource rich environment. However, in the poor diet, pathogen exposure seemed to impact the genetic correlations between pupal mass and development rate (Figure 15). The distributions for each dose shifted towards a more negative genetic correlation value showing a negative cline with increasing pathogen exposure, meaning that the bigger the difference in dose concentration, the stronger negative shift in genetic correlation. This negative cline between doses indicates that an increased dose exposes allocation variance within the population in a resource poor environment. This is consistent with the prediction made about pathogen presence. When dissolving differences in income between households or in this case resources between moths, allocation variance was thought to explain more of the population variance and with increasing dose this effect would be more extreme. The effect of pathogen does seem to expose allocation variance in this manner.

When looking at genetic correlation between survival and either of the other life-history traits, it showed little to no evidence for dose or diet having an impact (Figure 11, 12, 13, 14). Like for pupal mass and development correlations, survival correlations did not show much of a shift between doses in the rich diet and most distributions were centered around zero. But contrary to pupal mass and development rate, survival correlations for each pathogen dose in the poor diet did not reveal any gradual cline in any direction. Thus, there was no evidence for acquisition or allocation variance exposure between doses for survival correlations like there were for pupal mass and development rate correlations. There is some positive shift in the 1/4x dose from the control in the poor diet between survival and development rate indicating exposure of acquisition variance (Figure 11). But this is an exception to the complete comparison between all doses which does not contribute to any definite conclusions regarding how an increase in pathogen exposure will reveal acquisition variance. One thing to point out about this deviation is that when working with binomial survival data, that the 1x dose and to some extent the 1/4x dose have a very low survival rate which decreases the amount of data to put into the model. This may affect the credibility of the posterior distributions in the 1x dose especially. Due to the lack of data for these treatments compared to the other doses, the risk is that the prior distribution, explained in section 4.2, has not been updated correctly and therefore is not showing a posterior distribution but a reconstruction of the prior. If we had been able to gather more data to increase the number of observations in the higher doses the results may have shown that the genetic correlation, between survival and development rate within the 1x dose in the poor diet, followed the same direction as the 1/4x dose, positive compared to the control. This would show that there is a gradual positive cline between doses, and the argument for acquisition variance being exposed with increasing dose in a nutrient poor environment between survival and development rate, would be stronger. It could also have resulted in other relationships closer to the one between pupal mass and development rate. One would imagine that the cost of survival is high, yet it is very important for fitness, and therefore causes a negative covariance between, as argued in section 1.3. For example, pupal mass and survival might show a negative cline between doses due to the need for investing in both. This would be interesting to study further with a higher amount of data to support genetic analyses for the higher doses.

It is also worth considering that the number of sires impact the models' ability to predict genetic correlations. If we were able to mate more sires the correlations may have shown a stronger and more certain effect for both diet and dose. Since the correlations are close to zero it

is hard to conclude any definite evidence for shifts in genetic correlations between any of the life-history traits across different environments. Although, the effects that do come through in the results are still interesting to analyze and especially considering that some of the findings were in accordance with predicted outcomes. This indicates that the theory behind these concepts need to be tested further to determine more about how acquisition and allocation variance behave in a population.

To summarize, I found that allocation variance was revealed to an extent through an increase in pathogen exposure but only in a nutrient poor environment and when looking at growth and reproduction traits. However, there was little evidence that the resource restriction itself, in the form of a poor diet, caused any exposure in either acquisition or allocation variance within the same pathogen dose. When looking at survival correlations there were also little evidence supporting that the environment manipulation impacted the acquisition and allocation components of the populations' genetic diversity.

6. Conclusion

That the results showed an exposure of allocation variance indicates that there are genotype-by-environment interactions present in *H. armigera* when using resource restriction and pathogen exposure to manipulate the environment. Since *H. armigera* is a global pest, finding out how acquisition and allocation components vary across environments may help us understand more about the resistance development for this species (Tay et al., 2013). A varying environment will influence the allocation of resources and therefore change how organisms invest under certain stress and GxE interactions are very important when considering how biopesticides can influence pests (Lazzaro & Little, 2009). This may also help us engineer better and more efficient biocontrol products and procedures which can help reduce agricultural loss and increase food security (Haile et al., 2021).

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