UNIVERSITY OF GOTHENBURG

DEPARTMENT OF BIOLOGICAL AND ENVIRONMENTAL SCIENCES

PATHOGEN INDUCED ANOREXIA IN *HELICOVERPA ARMIGERA* MOTHS

A study on the genetic correlation between a reduction in feeding when infected and survival



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Table of Contents

Abstract 2
Introduction 3
Pesticides 3
Pathogen-induced anorexia 3
Adaptation or symptom (genetic correlation) 3
Aim 4
Material and method 4
Diet preparation 4
Adult diet 4
Larval diet 4
Moth parent preparation 5
Moth mating 5
Experiment design 6
Data analysis 7
Results 7
Simple trait analysis 7
The phenotypic response of pathogen-induced anorexia 9
The posterior distribution of feeding and survival 10
The genetic corelation of pathogen-induced anorexia and survival 11
Discussion 12
Survival 12
Feeding 13
Phenotypic response 13
Genetic correlation 13
Predation 13
Conclusion 14
References 14

Abstract

Pathogen-induced anorexia is a wildly accepted concept, where during an infection an organism reduces how much they are feeding. The reason for this is not yet fully understood and has over the years been attributed to it increasing survival during an infection and would therefore be considered an adaptive trait. Currently the basis for pathogen-induced anorexia being adaptive is not explored to a degree sufficient for that claim, as it currently stands and is only explored on the phenotypic level. This makes it very difficult to tell if it is an adaptive trait or only a symptom of infection, in this study I aim to examine if pathogen-induced anorexia can be considered and adaptive trait. To do this I quantify the genetic correlation between a reduction in feeding when infected and survival, in other words I examine to what degree genes that encode for reduced feeding when infected, also encode for increased probability of survival when infected. I did this by breading adult *Helicoverpa armigera* to create families of larvae consisting of siblings and half siblings, creating multiple individuals that share part of each other's genes. I then infected haphazardly selected individuals in each family with different doses of pathogen (high, low, and control) and two qualities of diets (standard and poor). Then in a Bayesian model I extracted the genetic correlation as a posterior distribution for each treatment. The distributions indicated no evidence for pathogen-induced anorexia being an adaptive trait, as the estimated values for the genetic correlation contained both large quantities of likely negative and positive values.

Keywords: Pathogen-induced anorexia, genetic correlation, adaptive trait, Helicoverpa armigera

Introduction

Pesticides

Weeds, bacteria, fungi and animals together are responsible for us loosing ca 40% of our annual harvests (Hakeem et al., 2016, p. 259). Among these, insects cause great damages and are attributed to upwards of 20% agricultural loss (Mangan et al., 2023). One such problematic species of insects pest is the *Helicoverpa armigera* also known as the Cotton bollworm, is a species of moths that has historically developed resistances to several classes of synthetic insecticide independently across several populations, and remains a problematic pest (Mangan et al., 2025). Currently it can be found globally and is established permanently in parts of south America, Africa, Australia, Asia and Europe (EPPO Global data base, 2026; Mangan et al., 2025). To combat pests such as *H. armigera* we sometimes use pesticides but as previously mentioned, it can often lead to the development of resistance with in the target pest species, rendering them mostly ineffective after a period of time (Marrone, 2019). Biopesticides are composed of biological agents such as bacteria, fungi, viruses and insects (Samada and Tambunan U.s.f., 2020).

Pathogen-induced anorexia

When infected by a pathogen it is not uncommon for animals to express a reduction in feeding, this as a phenomenon is referred to as pathogen-induced anorexia (also known as infection-induced anorexia or illness induced anorexia) (Adamo et al., 2010; Bashir-Tanoli and Tinsley, 2014; Jindal et al., 2024). The exact reason to why this is occurring is not fully understood, but a possible reasoning is that it increases the likely hood of survival (Adamo et al., 2010). The mechanisms pathogen-induced anorexia increasing survival are yet to be fully understood and multiple different theories have been tested (Adamo et al., 2007). In Adamo et al. (2007) they list four different, previously proposed mechanisms for pathogen-induced anorexia increasing survival from other studies including (1) that by reducing feeding the nutrient availability to the pathogen is lowered, starving the pathogen from macronutrients, (2) preventing the organism from consuming more contaminated food, (3) reducing the risk of pathogens spreading from the gut to the bloodstream and (4) increasing immune response via resource allocation. There is also the possibility that (5) by reducing the will to feed animals are less inclined to forage for food and there by lower the risk of encountering a predator (Hart, 1988). Furthermore, (6) there is evidence for the moth species *Manduca sexta* expressing pathogen-induced anorexia to consume less fat as lipid digestion was believed to inhibit immune function (Adamo et al., 2010, 2007).

Even if the proposed mechanisms by which pathogen-induced anorexia could increases survival are fundamentally different, there seems to be an underlying assumption that pathogen-induced anorexia is an adaptive trait, from phenotypic evidence and to my knowledge no test has been done to determine adaptivity. I feel that it is important to note that the validity of pathogen-induced anorexia being adaptive is not without question, and has been brought up (Adamo et al., 2007). For a trait to be adaptive it needs to be genetically dependent, it is formed over countless generations of selection in an environment that result in increased fitness (Dobzhansky, 1956). What studies that I found lack to include is the genetic element, as they never tested for pathogen-induced anorexia being an adaptive trait and instead only measured on the phenotypic level. The reason why this is important is that we cannot determine if a trait is adaptive by only looking at a phenotypic level, as it is verry difficult to then determine if, in this case, a moth is feeding less because it is combating the infection or if it is feeding less because it is about to die.

Adaptation or symptom (genetic correlation)

To determine is a trait is adaptive we need to include the element of genetics, as we have established in the previous paragraph adaptive traits are genetically bound. Testing for adaptivity

within a trait requires that we examen multiple groups of genetically similar individuals, meaning that the individuals within a group to some degree share DNA (Falconer, 1996) Then by assuming that individuals that share DNA will to some degree respond similarly to environmental conditions due to them sharing DNA, we are able to then deduce that if we measure many genetically similar individuals responses to different environments we are able to test only how the individuals themselves interact with the environment but how their genes interact with the environment (Falconer, 1996). By then doing this with multiple groups of genetically similar individuals we would be able to compare the genetic responses between groups for traits in different environments, and then by using this relationship determine the genetic correlation between traits (Falconer, 1996). The genetic correlation is defined as; to what degree genes that encode for one trait also encode for another trait (Falconer, 1996). Then to find out if pathogen-induced anorexia is an adaptive trait we need to find out to what degree do genes that encode for a reduction in feeding when infected (pathogen-induced anorexia) also encode for survival.

Aim

The aim of this study is to determine (1) if pathogen-induced anorexia can be found in *Helicoverpa armigera*, (2) if pathogen-induced anorexia is a symptom of being sick or an adaptive trait, by finding the genetic correlation between reduction in feeding and survival when *H. armigera is* exposed to the pathogen *Bacillus thuringiensis* (Bt).

Material and method

Diet preparation

Moth diet is separated between ‘Adult diet’ and ‘Larval diet’, both diets contain the same vitamin solution made from the ingredients listed in Table 1. The vitamin solution was made once in a big batch, and the vitamin solution was stored in glass bottle covered in aluminium foil to stop light from breaking down certain components and it was placed in a fridge.

Adult diet

The Adult diet is a liquid diet consisting of the ingredients listed in Table 2, mixed in a 1L bottle and is stored like the vitamin solution covered in aluminium foil and at a temperature of 5℃.

Larval diet

Preparation of the larval diet was done in batches as needed over the time of the experiment. Ingredients for one bottle are shown in Table 3 & 4. All ingredients except for the ethanol, distilled water and vitamin solution are weighed out to 3 decimal places on the first day of diet preparation. When preparing the dry ingredients one batch of both the ‘good’ and ‘poor’ diet is prepared in separate containers and covered with aluminium foil and stored in room temperature until it is time to add liquid ingredients. The difference between good and poor diet is that half of the white beans are replaced with cellulose in the poor diet, making it less nutritious as the cellulose is non digestible.

After liquid ingredients are added, the mixture is processed in a blender until reaching a semi homogenous liquid state. The mixture is added to an autoclave safe bottle and autoclaved at 121°C liquid setting for 90 minutes. A tray is sprayed with 70% ethanol, let's rest for 30 seconds and then wipe off excess. As soon as the diet is done in the autoclave it is poured into the tray lined with baking paper at an incline to get an even layer (ca 0.5 cm thick), under a laminar flow cabinet. Tray is then left under a UV light for 30 minutes to harden. The hardened media is then cut into pieces (ca 2 cm x 5 cm) and put into boxes and marked with the date of it being made and diet type and stored in fridge or cold room.

Table 1: Ingredients for the vitamin solution.

Ingredient	Amount
Niacinimide (Vitamin B3)	1 g
Calcium Pantotenate (Vitamin B5)	1 g
Riboflavin (Vitamin B2)	0.5 g
Thyamine (B1)	0.25 g
Piridoxine (Vitamin B6)	0.25 g
Folic Acid (Vitamin B9)	0.1 g
Biotin (Vitamin B7)	0.00002 g
Cobalamin (Vitamin B12)	2 g
Distilled water	1,000 g

Moth parent preparation

Helicoverpa armigera moth's pupa were ordered from a factory in Switzerland (Andermatt Biocontrol AG). The got sorted into groups separating male and female and separated into two separate climate cabinets. The male cabinet set to 27°C and the female cabinet set to 25°C, to speed up the emergence of male individuals and synch up the sexual maturity. Mature adults were placed into plastic disorphola tubes, with a piece of cotton soaked into adult diet and a cotton plug in the opening and then placed back into the 25°C cabinet.

Moth mating

Mating occurred in the climate-controlled rooms at Gothenburg University Natrium building, keeping a stable temperature of 25°C and a dark environment except for a lamp covered in red film to create a low light environment of red light. The red light was so that we could see but it would not disturb the moths during, after or before mating, and all other light sources were not used, except for screens at low light and pointed away from the moths to mitigate its impact.

Table 2: Ingredients for the adult diet.

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

Adult moths of separate sexes got paired haphazardly and transferred to clear plastic containers large enough to not restrict flight but small enough to keep the adults close to one and other, with a locking lid with breathing holes in. A piece of dark paper tissue was affixed to the side of the box so that the moths had an easy to grip surface and a piece of cotton soaked in the adult diet.

Table 3: Ingredients for the standard quality diet.

Standard larval diet	
Ingredient	**Amount for 1 bottle**
White bean	51.45 g
Agar	9.45 g
Ascorbic Acid	2.45 g
Wheat Germ	41.15 g
Soy protein	20.6 g
Casein	15.45 g
Brewer's yeast	25.7 g
Vitamin solution	4.1 mL
Sorbic acid	1.25 g
Ethanol	2.45 mL
Distilled water	600 mL

Table 4: Ingredients for the poor-quality diet.

Poor larval diet	
Ingredient	**Amount for 1 bottle**
White bean	27.525 g
Cellulose	27.525 g
Agar	9.45 g
Ascorbic Acid	2.45 g
Wheat Germ	41.15 g
Soy protein	20.6 g
Casein	15.45 g
Brewer's yeast	25.7 g
Vitamin solution	4.1 mL
Sorbic acid	1.25 g
Ethanol	2.45 mL
Distilled water	600 mL

Time of pairing, sire and dame ID was noted and until mating was completed behaviour of moths was noted to later determine if the sire or dame was "able" to mate. *Helicoverpa armigera* mates by docking the abdomens together of the sire and dam, a mating was noted as successful if it lasted more than or equal 20 min.

For a successful mating dam, it was transferred to a smaller plastic container with a new piece of dark tissue and fresh adult diet, so that it could start to lay eggs. The male counterpart it got put back into its disorphola tube and labelled as a mating sire; it then was made able to mate with multiple dams in the coming sessions with a night in between each mating. Unsuccessful mating moths got the opportunity to mate with another adult, but after continued failure they were assumed to not be of good enough to mate and where frozen. Dead and severely unwell moths where also frozen to make sure that it does not spread outside of the lab environment. The days following a successful mating, the eggs laid by the dam were collected and incubated in the climate cabinet at 25°C until they hatched. The emerging larvae was kept in the incubation box and feed the poor diet until they grew into the second instar.

Experiment design

The experimental design involved studying the feeding behaviour of moth larvae across different treatments of pathogen exposure (control, 1/16 and 1 dose) and diet quality (good and poor) then comparing sire families, to see if (1) pathogen exposure and subsequently infection caused larva to reduce feeding, (2) reduced feeding increased the odds of surviving the infection, (3) the traits of reduced feeding and survival were therefore genetically linked. The bacteria used was *Bacillus thuringiensis* and applied with a small spray bottle in the tube containing a piece of weighed out food and one larva. After 4 days the food was weighed again, the larva was weighed and then

given a new piece of food of the same diet and put back into incubation until it developed into a pupa. Pupa where sexed and put into incubation to see if they emerge as an adult moth or died.

Data analysis

The goal was to collect 100 larvae that reached second instar from each dam, and by the end there were 1306 individuals total adding together all sire families, 894 which survived 4 days past inoculation and could therefore be measured. Then to determine to what degree the genes that encode for reduced feeding when infected also encode for increased probability of survival when infected, we need to extract the genetic correlation. To do that I fit a Bayesian model for the effect of sire has on the relationship between traits (pathogen-induced anorexia and survival) and environment (pathogen and diet) while asking it to estimate the genetic covariance. It then preformed many rounds of sampling, exploring the data where after it stayed at the most likely values. Then by summarizing this collection of values can be expressed as a posterior distribution (i.e. the most likely new estimate of the relationship between the previously listed traits). This can then be plotted and used to visualize the genetic correlation of these traits, even when the genetic correlation cannot be directly measured.

Results

Simple trait analysis

The level of feeding for *H. armigera* depending on the exposed treatment is illustrated in Figure 1 and illustrates the individual level of feeding. The overall feeding in the standard diet treatment slightly decreases as the dose of pathogen increases, with a notable difference between the control and low dose when compared to the high dose. The difference between the control and low dose treatment in for the standard diet seems to be verry small, the control only has a few individuals that feed more than most low dose individuals. In the poor diet treatments, the level of feeding is more uniformed across doses, the control and low dose are very similar in, same as in the standard diet, while this time the high dose also show similar distribution and individual scores. Notably the difference in number of individuals in the control and low compared to the high dose regardless of diet, indicate that there are much fewer individuals in the high dose treatment.

Survival is itself illustrated in Figure 2 where the percentage of survival is depicted for every treatment and the similarly to in Figure 1, the difference between control and low dose is very small regardless of diet, with the low being slightly lower in both diets. The high dose treatment is much lower than all other doses regardless of the diet, with the standard diet having a survival less than 65% and the poor diet having slightly above 65% survival. This data is to be taken with a pinch of salt as we have established in Figure 1, the number of individuals that are described in the high dose treatments are much lower than the other treatments. This is a biproduct of this data set only including the moths that feeding could be measured from, this data there for excludes all the individuals that died before they could be measured for feeding. Therefor the percentage survival should be lower still in the high dose treatments.

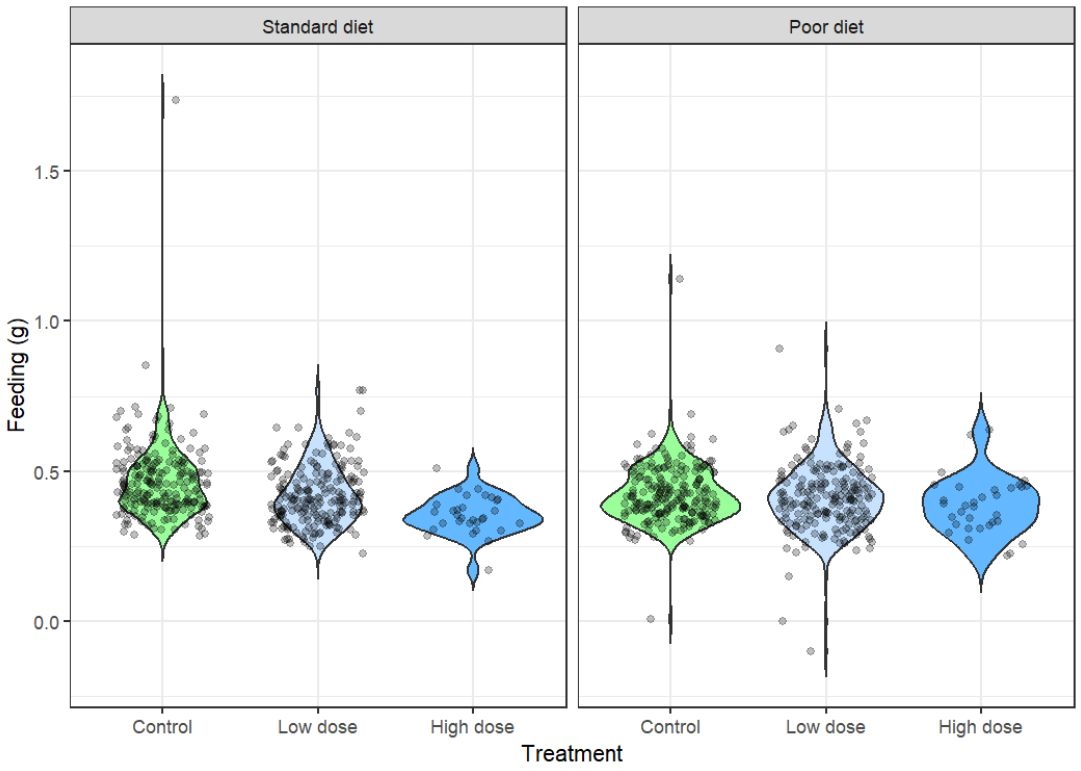


Figure 1: The level of feeding on an individual level grouped by the treatment (dose and diet) illustrated by a violin distribution that shows what the most common level of feeding was with in a treatment and the points depict individual feeding.

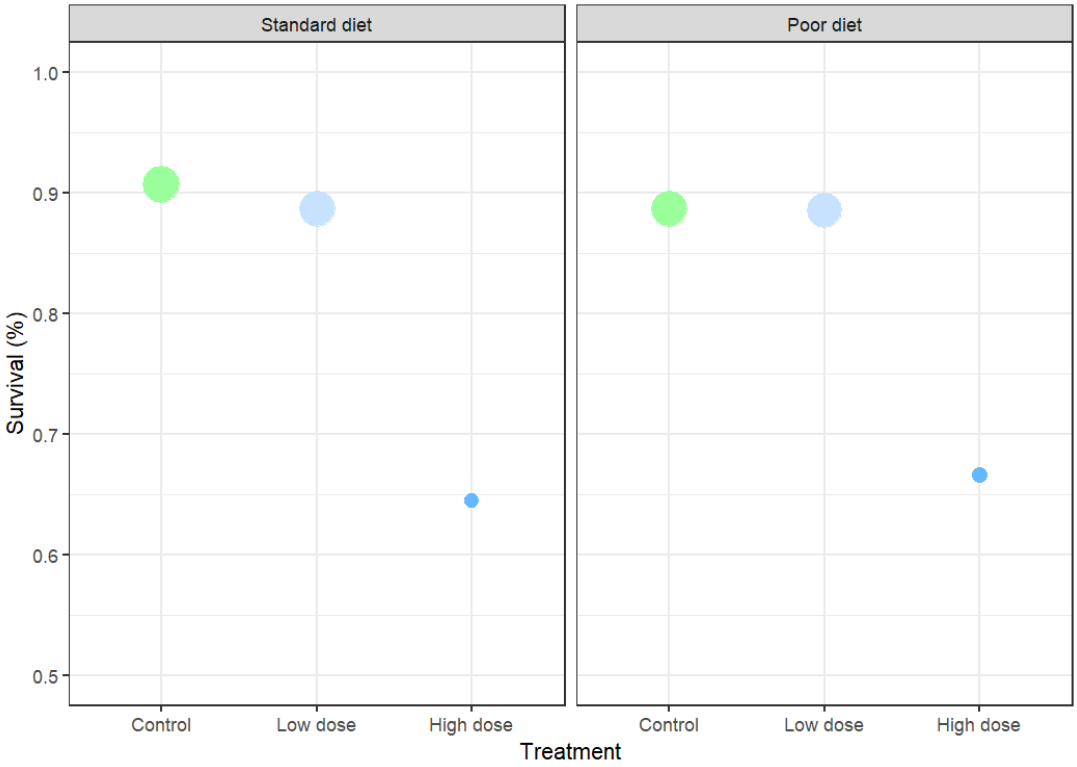


Figure 2: The percentage survival for every treatment indicated as a single point where the size of the point illustrates that some groups are represented by more individuals than others, the bigger points illustrate more individuals but not by any exact measure. Survival in standard treatments was ca 92 % (control), ca 88% (low dose) and ca 64% (high) respectively. Survival in the poor diet treatments was ca 88% (control and low dose) and ca 67% (high) respectively.

The phenotypic response of pathogen-induced anorexia

In my data I analysed 13 different families of *Helicoverpa armigera* larvae consisting of siblings and half siblings for a total of 894 individuals, examining the effect that reduced feeding have on survival during an infection. This relationship is visualized in Figure 3 where the percentage of survival is plotted against the level of pathogen-induced anorexia (the feeding in control subtracted by the feeding in an infected treatment, low dose for all), across treatments for each family. The more positive value corresponds to a stronger effect of anorexia in a treatment per family, what this means is that if a family has an anorexia value above zero, they feed less when infected by a pathogen then when compared to not infected. The percentage of survival (depicted on a decimal scale) is the proportion of individuals from each treatment that survived divided by the total number of individuals for each family. As mentioned, previously this does not include individuals that could not be measured and that is true going forward.

Then in Figure 3 if a family have a high anorexia value, they could also have a high percentage of survival when compared to a family that has a low anorexia value, if we believe that pathogen-induced anorexia increases survival. In Figure 3 we can see that for high dose treatments there no indication of survival increasing survival. For the low dose standard diet treatment there might be a slight positive correlation between the traits. The low dose poor diet treatment hade a somewhat uniformed survival regardless of anorexia. This is importantly only illustrating the phenotypic response of survival and anorexia for each family within a treatment and has little to do with adaptivity.

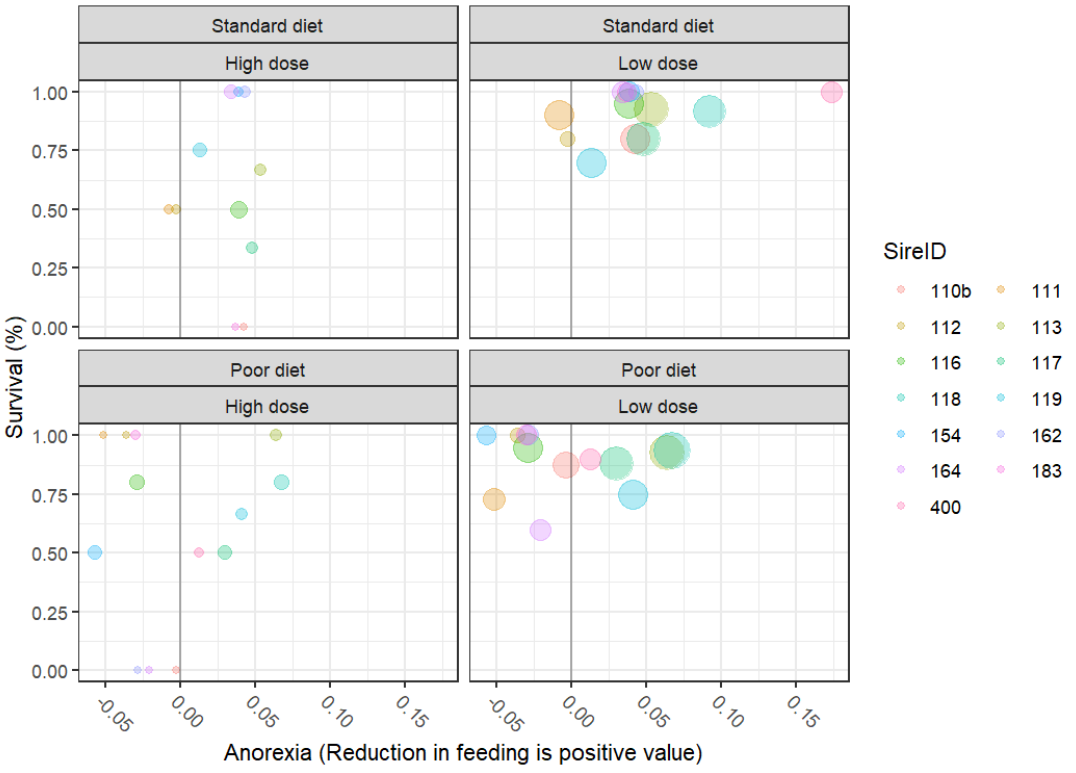


Figure 1: The phenotype of survival and when decreasing feeding across families (sires) of H. armigera. Each square represents a treatment of diet (standard or poor) and dose (high or low). The points depict the mean reduction in feeding and the percentage of survival for a given family and the size of points visualises the quantity of individual larvae that each point represents. Anorexia is the difference in food consumed between the treatment low dose and control; survival is the number of individuals for a treatment and family that survived divided by the total number of individuals in the same treatment and family.

The posterior distribution of feeding and survival

Previously in Figure 1 & 2 we talked about the level of feeding and percentage survival on an individual level. In Figure 4 & 5 we examine the posterior distribution of feeding (Figure 4) and probability of survival (Figure 5). In Figure 4 the posterior distribution is a probability distribution containing estimates of values feeding for a treatment. We can see that the distributions for the control and low dose treatments are still similar but the standard control being estimated as higher than other treatments. The high dose treatments are estimated to feed less than all other treatments. For Figure 5 we see how the probability of survival is verry similar regardless of diet in the control and low dose, as much of their distribution seem to overlap and have a very low uncertainty. In contrast, the high dose treatments are estimated to have a much lower probability of survival regardless of diet, but the data holds a much higher uncertainty.

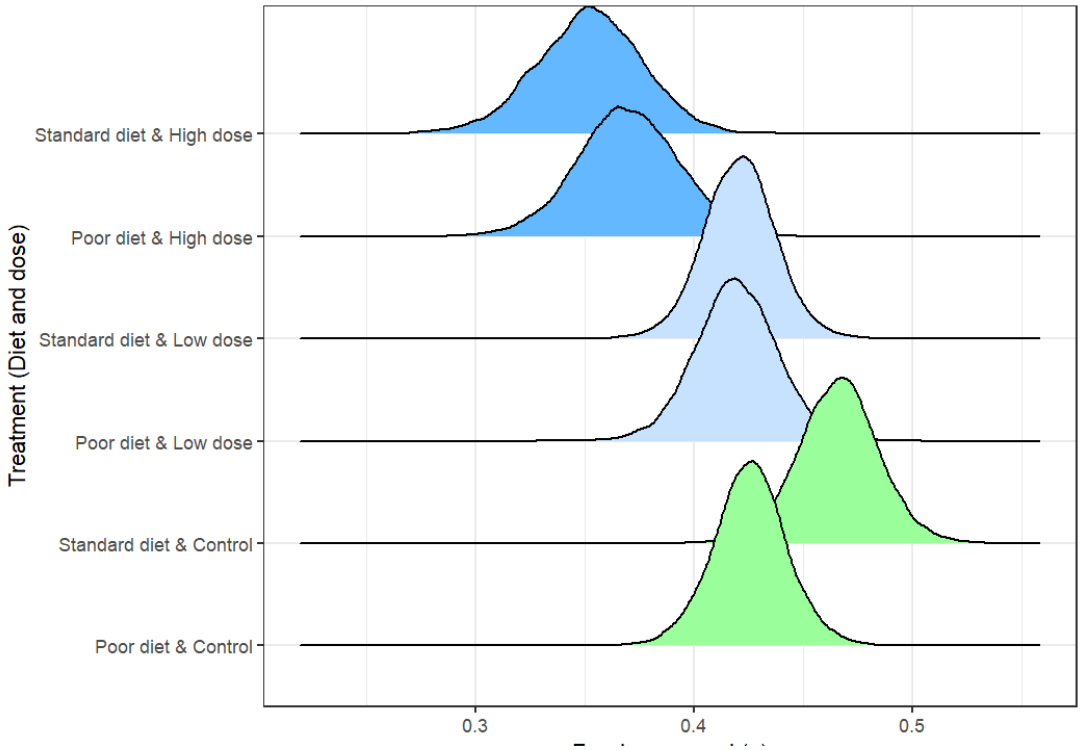


Figure 4: The posterior distribution of feeding across treatments. It is probability distribution depicting the most likely estimated values for feeding depending on treatment.

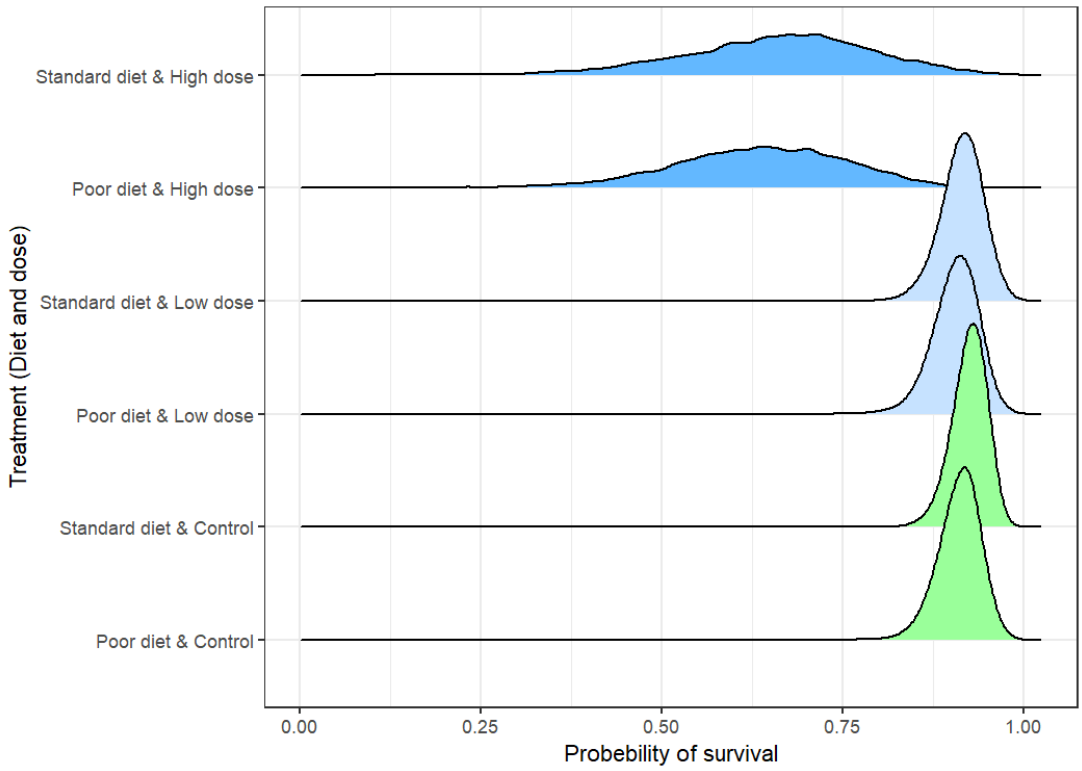


Figure 5: The posterior distribution for likelihood of survival across treatments. It is a probability distribution depicting the most likely estimated values for the probability for survival depending on the treatment.

The genetic corelation of pathogen-induced anorexia and survival

In Figure 6 we see the posterior distribution of the genetic correlation between pathogen-induced anorexia and survival, depicted as a probability distribution containing the most probable values for the genetic correlation. For the standard diet low dose treatment, we see the highest likelihood of a positive genetic correlation between the traits, as the peak of the distribution is situated slightly above zero. Still, many of the estimated values that are also likely are show negative correlation and the split between positive and negative correlation is somewhat even. For the standard diet high dose, we find the highest likelihood of pathogen-induced anorexia being negatively correlated with survival, but similarly to the standard low dose the treatment, many of the estimated values are also above zero. The uncertainty across treatment gives no clear indication of positive or negative correlation between pathogen-induced anorexia and survival.

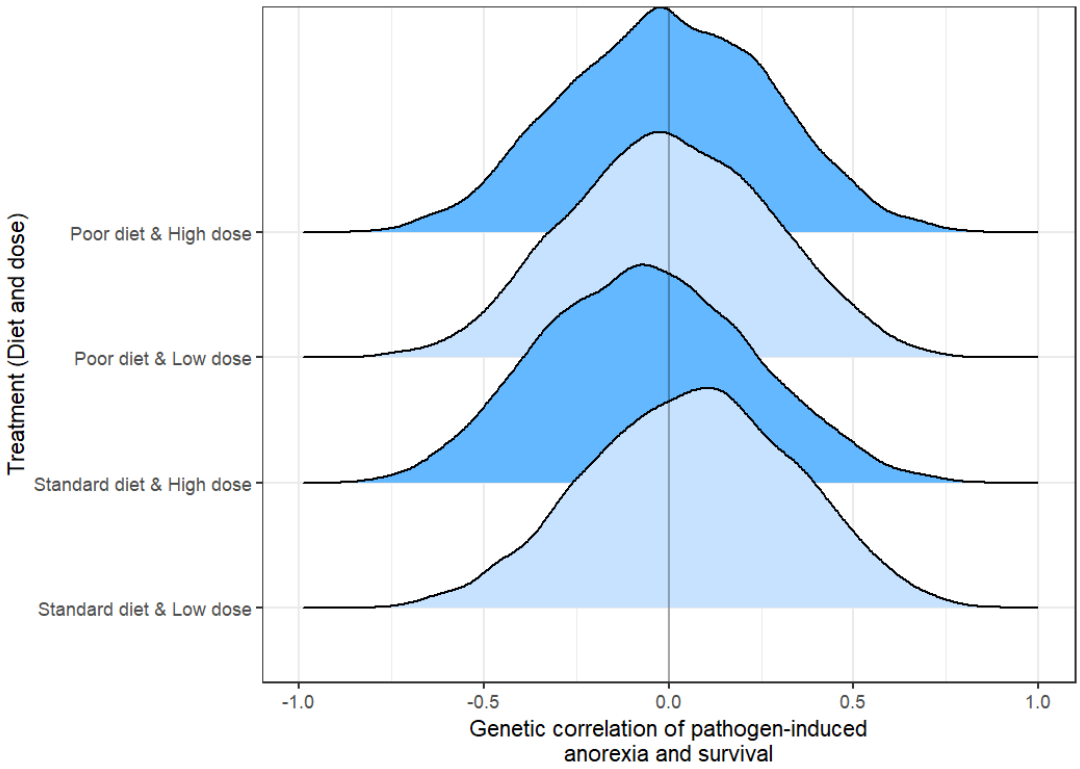


Figure 2: The posterior distributions for the genetic correlation between pathogen-induced anorexia and survival. It is multiple probability distribution made up of the most likely estimated values for the genetic correlation depending on the treatment.

Discussion

I studied 13 families of *H. armigera* larvae for a total of 894 individuals, examining the response in feeding and survival when infected by a pathogen, in this case Bt. The larvae where infected by different doses of the pathogen (in relation to the supplier's recommendation), and one control without pathogen, while also being given one of two diets of different quality. Then I observed how much the larvae where feeding and if they died before emerging as an adult. I used frequentist analysis approach; to examine the phenotypic response to treatments and I use a Bayesian model to extract the posterior distribution of feeding and probability of survival in addition to extracting the genetic correlation between pathogen-induced anorexia and feeding.

Survival

I found that the survival was mostly affected by the dose of pathogen as we can see in both Figure 2 & 5, the survival percentage and probability estimate was much lower in the high dose treatments regardless of diet. This is of course only including the 894 individuals that made it past the measurement of feeding and taking that into consideration the true survival in the high dose is much lower than shown in ether figure. The problem is that due to the low survival in the high doses, the Bayesian model has a harder time estimating probable values for those treatments due to the low number of individuals. As this is a direct result of the pathogen dose it is difficult to avoid and to get more reliable data the group sizes would have to be much larger and include more sire families. This is not said that Figure 5 that is derived from the Bayesian model is showing nothing, rather it might be a reason for the large uncertainty in the probability of survival.

Feeding

I found that feeding is affected by the dose of pathogen and partially by the quality of diet. We can see that in Figure 1 & 4 that the level of feeding becomes lower as the dose of pathogen increases, this is only slightly apparent for the low doses but rather clear for the high doses. In Figure 1 I feel that the differences are not as apparent as in Figure 4, this could be a result of the posterior distribution depicts the estimated most probable values. In this case that could indicate the individual moths that consumed much more than the population are outliers as their data is considered very unlikely by the model. Regardless, the effect of pathogen on feeding is apparent with increasing doses of pathogen, leading to decreased feeding, indicating the existence of pathogen induced anorexia as a trait in *H. armigera*.

It was also interesting to observe the effect diet had on feeding as in most treatments there does not seem to be much of a difference, but in Figure 4 control and standard diet the posterior depicts much higher values then for the poor diet. I found this strange as would it not be more logical for the poor diet to experience a higher level of feeding, as they would have to feed more to meet the same nutritional values. A possibility is that the larvae in the standard diet where able to feed more for the simple reason that the more nutrient rich diet might allow for more rapid growth, and subsequently bigger mouths allowing for more efficient feeding.

Phenotypic response

In Figure 3 we see the phenotypic response of anorexia and survival for each family for every treatment. What we see is that for most treatments their does not seem to be a correlation between pathogen-induced anorexia and survival except for maybe a correlation in the standard diet low dose treatment as survival slightly decreases as anorexia becomes lower.

Genetic correlation

Looking at Figure 6 there is little to no evidence for a genetic correlation between pathogen-induced anorexia and increased probability of survival when infected regardless for treatment. The strongest evidence is found in the low dose standard diet treatment, that displays a slightly positive mean genetic correlation. This is not how you examine a posterior distribution, as when we model using Bayesian statistics we include the uncertainty in the data. The posterior distribution is a probability distribution composed of the most likely estimated values, for the genetic correlation in this case. Therefore, we need to take the posterior distribution into consideration and not just the mean. Then when examining the distribution, we see that, yes, much of the distribution for the standard diet low dose treatment is positive, but at the same time it is only a slight majority and many negative values are also estimated to be likely. This is a trend in the other treatments as well, where no distribution shows any clear evidence for ether a positive or negative genetic correlation. This leads me to conclude that pathogen induced anorexia is most likely not an adaptive trait but instead just a symptom of infection in *H. armigera*.

Predation

There is one final topic I would like to discuss as it is relevant to the following conclusion bellow and the end of the section above. In this experiment I examined if pathogen-induced anorexia is an adaptive trait or just a symptom of infection, and that is what I have done. Still there is a very important distinction that I need to make regarding one of the previously mentioned theoretical mechanisms by which pathogen-induced anorexia might increase survival. During this experiment I did not test for how the presence of predators impacted the survival of *H. armigera* and I can therefore not conclude that pathogen-induced anorexia dose not increase survival in an environment where predators exist.

Conclusion

The aim of this study was to examine if pathogen-induced anorexia is found in *H. armigera* and the genetic correlation between pathogen-induced anorexia and increased survival. The point being that if pathogen-induced anorexia and increased survival have a genetic correlation that would indicate it being an adaptive trait, rather than just a symptom of infection. Considering the evidence laid fourth in this paper I conclude the following:

(1) That when infected by a pathogen (ex. Bt), *H. armigera* will experience a reduction in feeding, indicating that pathogen-induced anorexia per definition is expressed in the species.
(2) The genetic correlation between genes that encode for a reduction in feeding when infected and genes that increase the probability of survival when infected is very low and the uncertainty across treatments containing many positive and negative values. This indicates that there is no evidence for pathogen-induced anorexia being an adaptive trait but is more likely just a symptom of infection.
(3) Even if pathogen-induced anorexia might not be considered an adaptive trait from this experiment it is still possible it might increase the likelihood of survival if predators are a factor. This was never tested for during the experiment, this a biproduct of the lab environment.

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