

Theoretical and experimental studies on behavioural syndromes in aphids

by

Franz Wesley Simon

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Approval

Name: Franz Wesley Simon
Degree: Master of Science
Title: *Theoretical and experimental studies on behavioural syndromes in aphids*
Examining Committee: Chair: Allison Kermode
Professor

Bernard Roitberg
Senior Supervisor
Professor

Ron Ydenberg
Supervisor
Professor

David B Lank
External Examiner
University Research Associate and
Adjunct Professor
Centre for Wildlife Ecology

Date Defended/Approved: July 24th, 2014

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Abstract

Individuals' behaviors can be correlated across time and contexts, in a phenomenon now known as behavioral syndromes. Using an experimental approach, I demonstrate that genetically identical pea aphids are highly repeatable in multiple behavioral traits, however these behaviors are uncorrelated. Then using a state variable model I show that asymmetries in size can maintain a hierarchy between least and most bold individuals in foraging intensity across development. However, individuals that complete compensatory growth show an inversion in the rank-order of foraging activity between early and late development (ie individuals that are the boldest early in life are less bold than their "timid" counterparts late in life). In conclusion, I demonstrate both theoretically and empirically that non-genetic differences are capable of explaining repeatability in the expression of a single behavior; however I found no evidence that non-genetic mechanisms can correlations between multiple behaviors.

Keywords: Behavioral syndromes; pea aphids; animal personality; state-dependence; dynamic programming

Dedication

*I would like to dedicate this thesis to Sarah,
Frank, and Sandy Simon. For always listening
when I get excited.*

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List of Acronyms

ESS Evolutionary Stable Strategy

Glossary

Behavioral Syndrome Behaviors that are correlated across time and contexts.

Chapter 1.

Introduction

An animal's behavioral expressions are often correlated across time and contexts. Suites of correlated behavioral traits have been called coping strategies, animal personalities, and behavioral syndromes (Sih, Bell, & Johnson, 2004). Behavioral syndromes describe both the variation within a single trait, as well as the correlations among multiple traits. However, for there to be correlations among traits, a focal individual must be consistent when expressing each behavior. Individuals might even show personality within plastic traits so long as such behavioral expressions retain their rank order across contexts. Both of these phenomena suggest that individuals have limited plasticity and that a latent variable is stabilizing their behavior both within a single trait and across multiple traits.

Ecologists and evolutionary biologists have previously studied variation in and among phenotypic traits, and their previous research can help inform behavioral ecologists as to what is causing behavioral syndromes. For example, they have shown that genetically different populations of sticklebacks can have variation in pelvic spines due to different predation rates (Shapiro et al., 2004). Variation in pelvic spines is maintained due to a trade-off between energy investment and protection resulting in low and high predation- risk populations diverging in pelvic spine investment. Or, behavioral ecologists have used game theory to explain variation within a single population through negative-frequency dependence, in which rare strategies perform better than common strategies, allowing for the persistence of different strategies in the same population (Maynard Smith, 1982a). Additionally variation in behavior has been explained as individuals making the best of a bad job (Smith, 1982) where such individuals perform a strategy that maximizes their fitness although that fitness will be lower than that of other

individuals in the population. These three mechanisms could potentially maintain variation in behavioral expression, as well.

The typically high plasticity and reversibility of behavioral traits differentiates behavioral syndromes from phenotypic traits more traditionally studied in evolutionary biology. For example, investment into morphological anti-predator responses such as predator cues induces daphnia to grow tail spines to protect from predation (Walls & Ketola, 1989); however once acted upon, this investment is fixed. In contrast, animals may respond plastically to predation by rapidly adjusting their foraging intensity allowing an individual to adapt to varying levels of predation risk throughout their lives (Nussey, Wilson, & Brommer, 2007). Additionally, theory describing behavioral syndromes needs to explain the variation across individuals, but also maintain low variation within individuals (i.e. consistency or repeatability). For example, a mixed evolutionary stable strategy (ESS) does not necessarily result in a behavioral syndrome since although multiple actions could be present, all individuals could be playing the same strategy (Maynard Smith, 1982b). However, asymmetries, such as ownership (Smith, 1982), can lead to a mixed ESS that comprises of consistent individuals. Empirical studies have shown that personality traits are correlated with life history traits (Careau, Thomas, Humphries, & Re, 2008; Biro & Stamps, 2008). Wolf et al. (2010) demonstrated that repeatable behaviors can result from games among individuals with different life-history trade-offs by modeling individuals with short versus long life-spans resulting in longer lived individuals being bolder than shyer ones across their entire life. Still, personality traits have been discovered in populations where there was no support for negative frequency dependence as the driving mechanism (Kurvers, van Santen de Hoog, van Wieren, Ydenberg, & Prins, 2011). However, Luttbeg and Sih showed that pure conditional strategies could lead to long-term consistency when there is reinforcement (positive feedbacks) in size (i.e. large individuals are always larger than small individuals throughout life). Positive feedback loops stabilize individual differences and will stabilize labile traits resulting in the latent variable not converging and in turn the state-dependent behavior (Luttbeg & Sih, 2010; McElreath, Luttbeg, Fogarty, Brodin, & Sih, 2007). Therefore, theoretically, state-dependent differences can lead to stable behaviours for long periods of time, unfortunately few empirical studies have explored whether state-dependent differences are causing personality

Theories explaining the causes of behavioral syndromes have predominately explored the repeatability of a single trait, while ignoring the cause of correlations among multiple behaviors. The boldness-explorative syndrome, with individuals who are “risky” in one context being bold in others, is the most well studied correlation in traits (Moretz, Martins, & Robison, 2007). Examples of traits associated with the boldness-explorative syndrome are exploration of novel objects and reaction to danger cues (Moretz et al., 2007). More interestingly, syndromes are not always similar across different populations of the same species (Scales, Hyman, & Hughes, 2011). For example, the presence of predators predicted a boldness syndrome in sticklebacks while predator-naïve populations lacked a boldness syndrome (Dingemanse et al., 2007). Therefore, syndromes appear to be context-specific, suggesting that behavioral syndromes evolve in the context of a population’s environment. However, we know little about whether genetics and pleiotropy, maternal effects, or state-dependent differences are generating the personalities we see in nature.

This thesis combines both experimental and theoretical approaches to demonstrate that non-genetic factors can create and maintain behavioral syndromes. Using pea aphids I study whether behavioral syndromes can arise within a clone, and compare three different clones from two different evolutionary contexts to see if behaviors related to foraging are repeatable, and in turn if correlations arise among individuals’ behavioral expression. This is the second study to use clonal animals for studying behavioral syndromes and the first to study correlations of behaviors within genetically identical individuals. I also further expand Luttbeg and Sih’s (2010) model to explore the role of size, acting as a latent variable, to stabilize foraging rate if individuals can accrue damage and can undergo compensatory growth. Using a dynamic programming model I show how an individual’s size can stabilize its foraging intensity across development depending on growth strategy. This model introduces more biological realism than Luttbeg and Sih’s model and determines whether repeatability across development arises simply due to the restrictive assumptions of the model. This thesis works to disentangle the difference between repeatability and correlations among behavioral expressions and then using a state-dependent model I show that repeatability in behavior should be expected in systems where positive feedback loops occurs.

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Chapter 2.

Genetically identical pea aphids show high behavioral repeatability, while lacking a boldness syndrome

2.1. Abstract

Among individuals, multiple behavioral traits are often correlated across time and context, a phenomenon known as animal personality or behavioral syndrome. Previous empirical studies have shown that behavioral syndromes can differ between low and high predation risk environments. I investigated whether genetically identical pea aphids have behavioral syndromes and whether the same behavioural syndromes arise across an experimentally constructed productivity gradient and across different evolutionary backgrounds. I found that pea aphids have high behavioral variation among individuals but low variation within individuals (repeatability) in behaviors associated with boldness and exploration across a productivity gradient and regardless of evolutionary background. However, I found no evidence of correlated traits in pea aphids for personalities. The high repeatability of behavioral traits within clones demonstrates that non-genetic sources can cause repeatable behavioral expression, showing that either maternal effects or differences in experience during development can stabilize individual behavioral traits.

2.2. Introduction

Behavioral traits are often correlated across time and contexts. Suites of correlated behaviors have been called coping strategies, behavioral syndromes, and animal personalities (Sih, Bell, & Johnson, 2004). Behavioral syndromes are defined by two general concepts. First, an individual's behavior is highly repeatable, i.e. individuals act more like themselves than like other individuals in the population. Formally, repeatability is defined as greater variation between than within individuals. For example, individuals who are more active than the average individual when their activity is resampled are still more active than the average individual (Sih et al., 2003; Dingemanse & Wolf, 2010; Stamps & Groothuis, 2010a; Stamps & Groothuis, 2010b). Second, behavioral traits relating to different contexts are correlated. For example, female spiders who are highly voracious on heterospecific prey will also cannibalize their mates, pre-copulation, at higher rates (Johnson & Sih, 2005). Both of these phenomena suggest that individuals have limited plasticity or that a latent variable is stabilizing their behavior both within a single trait and across multiple behaviors.

Working with aphids, Scheutt et al. (2011) demonstrated that genetically identical individuals had highly repeatable escape-attempt behavior. But why would such behavioral syndromes arise in genetically identical individuals? Individuals with limited plasticity could avoid the high cost of learning by having lower plasticity in rapidly fluctuating environments, even if they are not performing optimally in all situations (Stephens, 1991). For example, if the predation level changes rapidly within an environment, sampling the environment to learn the predation risk is expensive. Therefore, it might be better to forage at one particular rate regardless of predation risk and not spend time sampling that predation risk. While this explains consistency in behavior it does not generate inter-individual variation in clonal organisms as reported by Scheutt et al. (2011). However, in combination with maternal effects, behavioral syndromes could arise wherein mothers bet-hedge by generating a diversity of strategies in their offspring. Alternatively, Luttbegg & Sih (2010) predict that small differences in ontogeny in less labile traits (i.e. size) could result in stabilized behavior across contexts due to reinforcement of small state-dependent differences (Luttbegg & Sih, 2010). Repeatability would thus be a function of productivity, with intermediate

levels of productivity yielding the highest repeatability since it allows variation to be maintained over the course of development. As such, in poor rearing environments (low resource quality or availability), all individuals would be in poor condition (starving) and therefore all individuals would be foraging at high intensity to avoid starvation. Despite the development of the theory studying behavioral syndromes, little empirical research has been conducted to test the predictions of these theories.

Theories explaining the causes of behavioral syndromes have predominately explored the repeatability of a single trait, while most empirical studies have focused on looking at the correlations among multiple behavioral traits. These empirical studies have shown that in multiple taxa, a boldness-explorative syndrome exists, with individuals who act “risky” in one context being bold in others (Moretz et al., 2007). Examples of traits associated with the boldness-explorative syndrome include exploration of novel objects and reaction to danger cues (Moretz et al., 2007). More interestingly, syndromes are not always similar in different populations of the same species (Scales et al., 2011). For example, a boldness syndrome in sticklebacks was associated with the presence of predators while this same boldness syndrome was absent in predator-naïve populations (Dingemanse et al., 2007). Therefore, syndromes appear to be context-specific, suggesting that behavioral syndromes evolve in the context of a population’s environment.

Most models of behavioral syndromes are designed to explain the repeatability of a specific boldness trait and assume that all such traits are highly linked. These models assume that multiple traits are stabilized by the same latent variable, that all boldness traits are controlled by a single locus, or that all bold traits will coalesce spontaneously (Dingemanse & Wolf, 2010). While it has been demonstrated that the presence of behavioral syndromes can change between evolutionary contexts, it is unclear whether different behavioral syndromes can evolve in different evolutionary contexts. For example, aphids commonly drop from a plant to avoid predation; therefore, dropping would be a risk-averse behavior while staying on the plant would be considered a bold tactic. However, for pea aphids in arid climates dropping is riskier than staying on the plant, due to high mortality on the ground, (Roitberg and Myers 1979), so in this context,

dropping would be bold while staying on the plant would be risk-averse. Thus populations in different environments might evolve different syndrome structures.

To study how gene-environment interactions could lead to repeatability and behavioral syndromes, I studied pea aphids *Acyrtosiphon pisum* (Hemiptera: Aphididae) and assayed their behaviors associated with boldness. In a 3X3 factorial experiment, I measured how quickly aphids began feeding on new host plants, their reaction to danger, and their exploration of the environment. The effect of evolutionary background and rearing environment on behavioral syndromes was tested by studying three clonal lines (from arid and humid environments) that were reared on plants of different nutritional quality. To partition the variation between and within individuals, repeated trials of each assay were completed to determine whether individuals were repeatable. I further extended this work to determine (i) if behavioral syndromes arose within a clonal line from non-genetic sources and (ii) whether different behavioral syndromes arose due to rearing environment, and evolutionary background of the clonal line.

2.3. Methods

Ease of study in the laboratory combined with the large number of previously studied behaviors makes pea aphids a good model system in which to study behavioral syndromes. Pea aphids are parthenogenic with short life spans thus, clonal lines can be maintained to study genetic versus non-genetic (i.e. maternal and ontogenical, respectively) contributions to behavior. Pea aphids previously have been shown to have highly correlated behaviors for host adaption due to genetic correlations (Via & Hawthorne, 2012), yet other than dropping behavior (Schuett et al., 2011), pea aphids have not been studied in the behavioral syndrome context (Via, 1999; Hawthorne & Via, 2001; Via & Hawthorne, 2012). Scheutt et al. (2011) found pea aphids have highly repeatable expression of their dropping behavior; however they did not study a suite of behaviors to see if such behaviours were correlated within individuals. Pea aphids have a wide range of behaviors that have been studied previously that can be analyzed together in a behavioral syndromes context: thanotaxis, ground search, latency until

feeding on a novel host, and escape-attempt behavior (Roitberg & Myers, 1979). Pea aphids can be used to test predictions of models and for testing the stability of repeatability and the phenotypic structure of behavioral syndromes across environments (Kralj-Fiser & Schuett, 2014).

2.3.1. Aphid source and colony rearing

Pea aphids were collected by sweep-netting alfalfa in Agassiz (49.250579, -121.848446) and Lillooet (50.685977, -121.921359), British Columbia, Canada during June 2012. Aphids from these two locations were used because previous studies had shown that aphids from these two populations differ in their population level dropping (Roitberg & Myers, 1979), and distance traveled after dropping on the ground (Roitberg, Myers, & Frazer, 1979). In the Lillooet area, due to higher summer temperatures, the ground is significantly more dangerous where aphids face significant danger from desiccation if they remain on the ground (Roitberg & Myers, 1979). Higher mortality on the ground has led to aphids being less likely to drop from plants when exposed to danger cues. In contrast, aphids face a low risk of desiccation on the ground in Agassiz, therefore aphids drop from the plants at a higher rate (Dill, Fraser, & Roitberg, 1990a). Three unique, genetically-identical aphid colonies were started from three different aphids two of the colonies from aphids from Agassiz, BC (AG1 and AG2) and one colony from aphids from Lillooet, BC (H99). All three colonies were maintained on broad bean plants at 21°C and 16:8 daylight cycles.

Bean plants, *Vicia faba* variety Windsor, were grown in a greenhouse under 16:8 day length cycle. Beans were planted along a productivity gradient in vermiculite (low quality), non-fertilized soil (medium quality), and fertilized soil (high quality). All plants were watered three times per week and the fertilized soil treatment received weekly fertilizing with 4:4:4 NPK. Aphids reared on higher quality plants were expected to be in better energetic condition and so should be more risk-averse due to asset-protection (Clark, 1994.) Aphids reared in poorer quality environments are expected to be less likely drop from a plant and will be more likely to travel long distances to the next plant following a simulated predation event. Previously, rearing aphids on fertilized plants has led to larger aphids than those rearing on non-fertilized plants (Gruber & Dixon 1988).

2.3.2. Focal aphid rearing

Individual aphids selected for use in the experiments were prepared by isolating an adult aphid from a focal colony and transferring it onto either a low, medium, or high quality 4-leaf bean plant. The adult aphid was maintained on the plant for 24 hours so that it could produce offspring before it was removed. The offspring were then raised on the plant for 9 days (the time it takes an aphid to reach fourth instar) before use in behavioral assays. The resulting 3x3 factorial design of rearing environment (a plant's nutritional quality) and evolutionary background (clone) allowed for testing whether behavioral syndromes are present among clonal individuals and whether rearing environment changes behavioral syndromes.

2.3.3. Behavioral assays

Behavioral traits associated with boldness were assayed, including: novel host exploration, latency until escape attempt, type of escape attempt behaviour, length of thanotaxis, distance traveled, and running speed. Assays were all conducted on medium-quality bean plants to remove the confounding factor of differing resource quality during the behavioral assays. To measure repeatability, twenty individuals per combination of clone and rearing environment behavioral expressions were assayed twice. Aphids were tested in a fixed order according to the schedule listed in Table 2.1. A fixed schedule with a non-random order was employed given that a design across three separate behavioral assays with each individual repeated twice would result in $6!/(2!2!2) = 90$ possible permutations and as such, low statistical power. This would have made it difficult to explore possible carryovers between assays (Bell, 2012). Therefore, I used a fixed order since this experiment was designed to measure repeatability and not the effect of test order (Bell, 2012). The description of each of the assays follows below.

Novel host exploration

All experiments were conducted on individual aphids that were transferred to fresh medium-quality bean plants with a paintbrush. The amount of time till aphids

started feeding was measured, which was defined as the moment the aphids began probing the plant with their stylet. In fact, aphids do not begin imbibing phloem fluid until they have completed navigating their stylets around cells and finally secured their stylet in place (McLean & Kinsey, 1964); however, this would be nearly impossible to discern, thus we employed the readily observable metric. Observations were ended if an individual had not begun probing within 5 minutes of arrival on a plant.

Time until escape attempt and escape type

One hour after aphids had settled on their plant and were actively feeding, they were exposed for thirty seconds to a 0.02% solution, by volume, of 2µl of aphid alarm pheromone (E-beta-farnesene) dissolved in hexane (Clegg & Barlow, 1982). Time from the aphids' initial exposure to alarm pheromone until escape attempt behaviour was measured. An aphid's reaction, from the least energetically costly to the most energetically costly (Dill, Fraser, & Roitberg, 1990b), was categorized into no response, antennae waving, removal of stylet (stops feeding), running from the feeding site, and dropping from the plant.

Thanotaxis, distance, and speed

The aphids were allowed to settle for two hours after the escape attempt behaviour assays. Individuals were then exposed to the same dose of alarm pheromone. If an aphid had not dropped from the plant after thirty seconds, it was prodded gently with a paint brush until it dropped. Aphids dropped onto a 30cm by 30cm sheet of paper that was marked with a 1cm by 1cm grid. Thanotaxis, the amount of time until aphids started moving after falling from the plant, was measured. The total distance an aphid covered after it began moving was measured by recording an aphids' location every five seconds until they stopped moving for three minutes, returned to the plant, or left the arena. Speed was calculated by measuring the total distance traveled divided by the length of time the individual remained active.

2.4. Statistical analysis

The statistical analysis is divided into three sections: 1) calculating the repeatability of traits, 2) testing if the mean population response differed among clonal lines or rearing environments, and 3) examining the correlations between trait expressions.

Repeatability

Intraclass correlation was used as a metric for repeatability of traits and was calculated by fitting generalized random effects models with individual as a random intercept for all traits (see Nakagawa & Schielzeth, 2010). To calculate repeatabilities for escape attempt behaviour for droppers versus non-droppers, I used a logistic regression with a logit-link. The confidence intervals for repeatability were constructed by bootstrapping individuals to maintain the error structure of the data (Nakagawa & Schielzeth, 2010) and 95% percentile confidence intervals ($\alpha_{2.5}$, $\alpha_{97.5}$) were built.

2.4.1. Population level effects

The mean population level effects were calculated for each behavior to determine if the plant-quality treatments and the differences in clones caused the predicted population level effects. Generalized linear mixed effects models were fit to each trait with individual as a random effect and clonal line and plant quality as fixed effects. Handling times, speed, thanotaxis, and escape-attempt latency were fit with a linear mixed model.

2.4.2. Behavioral axis

The correlation structure of behavioral traits was examined using an exploratory factor analysis. All continuous traits were scaled and centred and then a principle component analysis (PCA) was completed on all traits. A strong correlation was considered to be >0.7 while weak correlation was defined as <0.4 (Jackson, 1991).

Principle component analysis was completed both on the pooled populations and then on each treatment individually to see if the behavioral syndrome changed between clones and plant quality.

All statistics were performed in the 3.0.11 R environment. The mixed effects models were fit using the LME4 packages. The cumulative ordinal regression was computed using the VGAM package in R.

2.5. Results

2.5.1. Repeatability

All behavioral traits had high repeatability except for novel host exploration (Table 2.4). Repeatabilities were considered to be non-zero or different if the confidence interval did not overlap. Additionally, repeatability did not change significantly between aphids reared on different quality plants or between clonal lines.

2.5.2. Population level effects

Population level effects are reported in Table 2.2 and 2.3. Novel host exploration, latency to escape, and thanotaxis showed no differences due to any treatment. Aphids from Lillooet walked significantly further and ran faster when on the ground than from Agassiz (Table 2.2). Escape-attempt type changed significantly with plant quality and clonal line. Aphids on lower quality plants were more likely to drop, run, or stop feeding than when tested on higher quality plants (Table 2.3). Aphids from Lillooet were less likely to engage in dropping and running than the two clonal lines from Agassiz (Table 2.3).

2.5.3. Behavioral axis

The PCA loadings are reported in Table 2.5-2.8. Every trait loaded onto its own axis with no traits having higher than 0.4 correlations providing no support for any correlated behaviors. A confirmatory factor analysis was not performed due to the low loadings. The observed behaviors associated with boldness were not correlated in aphids suggesting that within a clone aphids do not have a boldness syndrome.

2.6. Discussion

Do non-genetic mechanisms generate repeatability or suites of correlated traits in pea aphids? Pea aphids are known to have genetic correlations (Via & Hawthorne, 2012) between behavioral traits and different clones have different mean level responses. Irrespective of clonal line and rearing conditions, we found high repeatabilities at the individual level for all behaviours, except novel host exploration. Scheutt et al. (2011) found that aphids had high repeatability in their dropping behaviour across five days of an adult aphid's life. The repeatabilities in all traits reported here were even higher than previously described for pea aphids. High repeatability across rearing environment quality despite changes in mean population level effects changed demonstrates that high repeatability will be maintained even when individuals are raised in different environments. All clones had similar repeatabilities despite having differences in their mean level behavioral traits based upon average responses between clones. Aphids from Lillooet engaged in behaviors that removed them from the ground. They traveled farther, dropped less, and ran faster. Aphids grown on high quality plants (fertilized) were also more risk-averse (Table 2.2). The high repeatability in multiple traits across rearing environments and evolutionary background suggests that pea aphids have highly repeatable behaviours within a clone.

What is causing genetically identical individuals to express highly repeatable behaviours, ontogeny or maternal effects? Asymmetries in ontogeny could result in personalities as explored theoretically by Luttbeg & Sih (2010) and experimentally by Tremmel & Muller (2012). In animals, such as fish, an initial difference in size during

early development has been shown to lead to individuals reaching extremely different sizes due to positive feedback loops. A positive feedback loop exists for size due to its link with predation risk, which leads to larger individuals foraging more boldly and growing to even larger sizes because they have lower predation risk. A similar mechanism could be working in insects where larger/stronger individuals gather more resources and molt at larger sizes than their competitors, preventing smaller individuals from ever catching up. While I did not directly test whether variation in size determined personality, Luttbeg and Sih's (2010) model predicts that repeatability is a function of productivity. Luttbeg & Sih (2010) predicted decreased repeatability due to lower variation in size between individuals at low and high values of environmental productivity. However, since no changes along the productivity gradient were seen in repeatability, this suggests that differences in size or other dynamic latent variables are not the mechanisms generating the stable personality in pea aphids.

Maternal effects likely cause personality in aphids. Maternal effects are known to occur in aphids and personality has previously been shown to be generated by maternal effects in birds (Duckworth, 2006). Via maternal effects, clonal lines have been demonstrated to change their phenotypes over time resulting in changes to the mean population level responses to danger in pea aphids (Andrade & Roitberg, 1995). Maternal effects in pea aphids can occur through chromatin re-modelation (methylation), sRNA, and endosymbionts (Srinivasan & Brisson, 2012). This gives aphid mothers the ability to bet-hedge and generate variation among their offspring without performing sexual reproduction (Crean & Marshall, 2009, J. Marshall & Uller, 2007). Future studies in pea aphids will need to determine if repeatable behaviours can be generated by manipulating mothers and/or grandmothers to cause repeatable individual differences in behavioral traits.

The high levels of repeatability across environments and clones did not translate to correlations between traits, suggesting that the mechanism(s) that stabilize repeatability do not cause traits to coalesce. Are there latent variables that can stabilize a single trait but not a suite of traits? For example, maternal effects could be causing high repeatability traits in individual traits, but are interestingly not having pleiotropic effects leading to suites of correlated traits. This suggests that pea aphids will generate

repeatability but maintain flexibility with individuals having a different behavioral response in each behavioral trait.

In the future, studies should begin studying the genetic architecture versus the phenotypic structure of behavioral syndromes. Phenotypic correlations in behavioral traits could be caused by genetic correlations of behavioral traits. Pea aphids previously have been shown to have highly correlated behaviours for host adaption at the genetic level (Via, 1999) (Hawthorne & Via, 2001, Via & Hawthorne, 2012). Studies with pea aphid host adaption have the added benefit of links between behaviors and loci on chromosomes being well-characterized allowing for connection between repeatable traits and their underlying genetics (Hawthorne & Via, 2001). This concept could be extended to the boldness and exploration-related traits used in this study. Aphids also have well-described variation in traits associated with social behaviors such as host suicide (McAllister & Roitberg, 1987; Losey & Denno, 1998). Additionally, pea aphids' wide geographic and host range make them an ideal candidate to study the different selective forces generating genetic correlations between behaviors

In conclusion, the mechanisms that cause repeatability may not be the same ones that cause correlations between traits. Repeatability was generated through non-genetic factors, however, within a clone pea aphids' behaviors did not correlate into a boldness syndrome. This research highlights the need to develop theory for why repeatability in multiple traits is common without seeing correlations between traits. Although this study did not find evidence of behavioral syndromes generated by maternal effects or ontogeny, pea aphids have already been shown to have genetic correlations in traits associated with host feeding. Therefore, future studies in pea aphids investigating behavioral syndromes should investigate behavioral syndromes between genetically diverse individuals.

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2.8. Tables & Figures

Table 2.1. The schedule of behavioral assays.

Behaviors were assayed in a fixed order and all assays were repeated twice to create a measure between and within individual repeatability.

Behavioral assay	Time of day
Host Latency 1	9:00
Escape Attempt Latency 1	10:00
Escape Attempt Type 1	10:00
Host Latency 2	11:00
Escape Attempt Latency 1	12:00
Escape Attempt Type 2	12:00
Thanotaxis 1	14:00
Displacement/Speed 1	14:00
Thanotaxis 2	16:00
Displacement/Speed 2	16:00

Table 2.2. Differences in mean population-level behavioral responses of pea aphids based on clone and rearing environment.

All values are regression coefficients from linear mixed effects models except for escape attempt behavior which is a logistic regression with a logit link. Individual was included as a random effect for all behavioral traits (n = 180). Pea aphid mean population-level behavioral responses and their difference from aphids of the AG1 clone reared on vermiculite. Reported with the mean effect and the 95% bootstrapped confidence intervals ($\alpha_{2.5}$, $\alpha_{97.5}$). A regression coefficient was taken as significant if the confidence interval did not overlap zero and are denoted with an “a”.

Regression Coefficients	Behavior				
	Novel Host (seconds)	Latency to escape attempt (seconds)	Thanotaxis (seconds)	Displacement (centimeters)	Velocity (centimeters/second)
AG1 Poor (Vermiculite)	81.96(49.92, 115.00)	44.45 (25.32, 63.31)	80.57(-7.43, 168.50)	13.36(14.19, 11.80)	2.4(0.77, 5.17)
AG2	-48.36(-98.43, 8.08)	-18.28 (-36.48, 0.54)	-14.15(-41.06, 13.18)	6.96(-2.33, 16.21)	0.21(-1.64, 2.10)
H99	-31.82(-63.02, -1.50)a	-11.74 (-30.24, 10.72)	-15.01(-46.93, 14.22)	17.78(6.23, 29.3)a	2.97(0.77, 5.16)a
Medium (Soil with no Fertilizer)	9.02 (-24.53, 43.56)	9.97 (- 0.10, 0.72)	-11.27(-11.74, -9.82)	2.12(-20.39, 25.06)	-0.21(-1.41, 1.90)
High (Soil with Fertilizer)	-13.10(-46.60, 19.90)	29.29 (7.85, 50.72)a	-14.14(-18.24, 45.89)	7.80(-3.78, 19.58)	-0.09(-2.20, 2.22)

Table 2.3. Ordinal mixed effect regression of pea aphid escape attempt behavior.

The coefficient estimates for pea aphid escape attempt behavior from an ordinal regression analysis. The full model's log-likelihood is 290.10 resulting in a $p = 0.054$, $n = 180$. Aphids from higher quality plants (fertilized) are less likely to engage in a highly energetic escape attempt.

Treatment coefficient	Escape attempt behavior	Standard Errors and P-Values
AG1 Poor (Vermiculite)	NA	NA
Agassiz 2 (AG2)	-0.28 SE 0.35	Z = -0.8, P = 0.42
Lillooet (H99)	0.5 SE 0.5	Z = 1.3, P = 0.18
Medium (Soil with no Fertilizer)	-0.84 SE 0.4	Z = -2.0, P = 0.04
High (Soil with Fertilizer)	-1.03 SE 0.4	Z = -2.6, P = 0.01

Table 2.4. Behavioral repeatability of individual aphids.

The behavioral repeatability (intraclass correlation) for individual aphids and their bootstrapped 95% confidence intervals for all measured pea aphid behaviors. All behaviors had high repeatability except for novel host exploration which was extremely low and statistically non-significant. The average repeatability across all clones and rearing environments is also reported in the pooled column. Within a behavior repeatability did not vary across clones or rearing environments since the confidence intervals always overlapped.

Behavior	Treatment									
	Pooled n = 180	Agassiz 1 (Ag1)			Agassiz 2(Ag2)			Lillooet (H99)		
		Low n = 20	Medium n = 20	High n = 20	Low n = 20	Medium n = 20	High n = 20	Low n = 20	Medium n = 20	High n = 20
Novel Host	0.20 (0.00, 0.27)	0.13 (0.00, 46)	0.53 (0.00, 0.71)	0.01 (0.00, 0.02)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)	0.49 (0.00, 0.80)	0.00 (0.00, 0.01)	0.16 (0.00, 0.31)
Dropping	0.79 (0.62, 0.87)	0.72 (0.55, 0.87)	0.68 (0.43, 0.92)	0.72 (0.450, 0.87)	0.93 (0.60, 0.96)	0.86 (0.61, 0.87)	0.74 (0.43, 0.91)	0.73 (0.43, 0.91)	0.78 (0.42, 0.89)	0.94 (0.73, 0.98)
Latency to Escape	0.83 (0.20, 0.92)	0.57 (0.32, 0.87)	0.95 (0.60, 0.98)	0.90 (0.60, 0.96)	0.80 (0.60, 0.91)	0.83 (0.60, 0.93)	0.97 (0.60, 0.99)	0.78 (0.60, 0.86)	0.90 (0.60, 0.94)	0.89 (0.60, 0.96)
Thanotaxis	0.70 (0.40, 0.87)	0.80 (0.42, 0.99)	0.83 (0.60, 0.91)	0.75 (0.60, 0.89)	0.68 (0.60, 0.86)	0.72 (0.60, 0.90)	0.77 (0.60, 0.90)	0.80 (0.60, 0.93)	0.69 (0.60, 0.91)	0.82 (0.60, 0.95)
Displacement	0.75 (0.35, 0.87)	0.57 (0.32, 0.90)	0.95 (0.78, 0.99)	0.91 (0.76, 0.98)	0.86 (0.81, 0.96)	0.81 (0.78, 0.94)	0.96 (0.88, 0.99)	0.87 (0.76, 0.96)	0.90 (0.78, 0.98)	0.89 (0.74, 0.97)
Velocity	0.54 (0.32, 0.71)	0.60 (0.60, 0.87)	0.60 (0.60, 0.87)	0.60 (0.60, 0.87)	0.60 (0.60, 0.87)	0.60 (0.60, 0.87)	0.60 (0.60, 0.87)	0.60 (0.60, 0.87)	0.63 (0.60, 0.87)	0.67 (0.60, 0.87)

Table 2.5. Correlations among behaviors, pooled from all three clones, explored through a principle components analysis (PCA).

Principle component scores for each individual population and for the pooled populations. All behavioral traits loaded onto their own axis, since no two correlations were higher than 0.40 per axis suggesting that aphids do not have a boldness syndrome in the observed traits. In the table trivially small correlations are marked by a dash (-).

Pooled	Trait	PCA 1	PCA 2	PCA 3	PCA 4	PCA 5	PCA 6
n = 180	Escape attempt latency	-	-0.117	0.988	-	-	-
	Novel host	-0.911	-0.312	-	-	-	-
	Escape attempt type	-	-	-	-	-	-1.00
	Running speed	-	-	-	-	-0.996	-
	Displacement	-	-	-	-0.993	-	-
	Thanotaxis	-0.304	0.904	0.136	-	-	-
	Cumulative variation explained	0.167	0.333	0.500	0.667	0.833	1.000

Table 2.6. Correlations among behaviors within the clone from a humid environment (AG1) explored through a principle components analysis (PCA).

Principle component scores for each individual population and for the pooled populations. All behavioral traits loaded onto their own axis, since no two correlations were higher than 0.40 per axis suggesting that within an aphid clone there is not a boldness syndrome in the observed traits. In the table trivially small correlations are marked by a dash (-).

Agassiz 1	Behavior	PCA 1	PCA 2	PCA 3	PCA 4	PCA 5	PCA 6
n = 60	Escape attempt latency	0.210	-0.102	0.970	-	-	-
	Novel host	-0.905	-0.296	0.153	-	-	-
	Escape attempt type	-	-	-	-	0.101	-0.995
	Running speed	-	-	-	-	-0.990	0.101
	Displacement	-	-	-	-0.991	-	-
	Thanotaxis	-0.269	0.912	0.172	-	-	-
	Cumulative variation explained	0.167	0.333	0.500	0.667	0.833	1.000

Table 2.7. Correlations among behaviors within the clone from a humid environment (AG2) explored through a principle components analysis (PCA).

Principle component scores for each individual population and for the pooled populations. All behavioral traits loaded onto their own axis, since no two correlations were higher than 0.40 per axis suggesting that within an aphid clone there is not a boldness syndrome in the observed traits. In the table trivially small correlations are marked by a dash (-).

Agassiz 2	Trait	PCA 1	PCA 2	PCA 3	PCA 4	PCA 5	PCA 6
n = 60	Escape attempt latency	-	-0.142	0.985	-	-	-
	Novel host	0.959	-0.277	-0.516	-	-	-
	Escape attempt type	-	-	-	-	0.141	0.990
	Running speed	-	-	-	-	-0.988	0.141
	Displacement	-	-	-0.102	-0.990	-	-
	Thanotaxis	0.275	0.950	0.137	-	-	-
	Cumulative variation explained	0.167	0.333	0.500	0.667	0.833	1.000

Table 2.8. Correlations among behaviors within the clone from Lillooet (H99) explored through a principle components analysis (PCA).

Principle component scores for each individual population and for the pooled populations. All behavioral traits loaded onto their own axis, since no two correlations were higher than 0.40 per axis suggesting that within an aphid clone there is not a boldness syndrome in the observed traits. In the table trivially small correlations are marked by a dash (-).

Lillooet	Trait	PCA 1	PCA 2	PCA 3	PCA 4	PCA 5	PCA 6
n = 60	Escape attempt latency	-0.121	0.976	-0.173	-	-	-
	Novel host	-0.852	-0.194	-0.484	-	-	-
	Escape attempt type	-	-	-	-	-	0.999
	Running speed	-	-	-	-	-0.976	-
	Displacement	-	-	-	-0.994	-	-
	Thanotaxis	-0.308	-	0.857	-	-	-
	Cumulative variation explained	0.167	0.333	0.500	0.667	0.833	1.000

Chapter 3.

Repeatability across development: compensatory growth destabilizes long-term adaptive personalities

3.1. Abstract

Behavioral syndromes have become an extremely popular area of study in behavioral ecology; however, we still understand little of the mechanisms that generate them. There is theoretical support that state dependent differences, even in labile traits, could generate behavioral syndromes. Focusing on size, as an example of a latent variable that could cause personality, I model an ontogenic period of an animal's life where individuals can grow, and accumulate damage via metabolic processes. Using a state variable modeling approach, I built a model in which individuals can optimize their foraging intensity, and the amount of energy they allocated to repairing accumulated metabolic damage. Optimal growth trajectories constructed from this model show that small initial differences in size can generate behavioral syndromes. However, when animals are capable of performing compensatory growth size no longer stabilizes behaviour across the entire development period because small individuals will grow very rapidly early in their development. This results in the rank-order of activity being reversed between small individual early and late in development. This study highlights that in depth study of potential latent variables is important for understanding behavioral syndromes and long-term repeatability of behaviour. However, the long-term stability of behavioral syndromes depends on whether individuals are capable of undergoing compensatory growth.

3.2. Introduction

The study of phenotypic variation is a mainstay of evolutionary biology, however most research has focused on variation in traits that are non-reversible or even non-plastic within an individual. For example, genetically different populations of sticklebacks can express fixed variation in pelvic spines due to different predation rates (Shapiro et al., 2004) where investment in pelvic spines is fixed. However, investment into morphological anti-predator responses can also be flexible. For example, predator cues induce daphnia to grow tail spines to protect from predation (Walls & Ketola, 1989); however once acted upon, investment is fixed. In contrast, animals respond plastically to perceived predation risk via behavioral changes and can rapidly adjust the expression of such behaviours to adapt to different contexts throughout their lives (Nussey et al., 2007). Despite the labile nature of many behaviors, long-term inter-individual variation exists for some behaviors across contexts and time, a phenomenon known as behavioral syndrome. For example, individuals who are relatively more active at the beginning of their life are relatively more active later in life (Sih et al., 2004). The proximate (Duckworth, 2006) and ultimate mechanisms (Wolf & Weissing, 2010) for the maintenance of consistency in behavioral traits has become a major area of study of behavioral ecology. If animals are able to change their behaviour between contexts, why do different individuals in the same environment behave differently?

Studying variation in behavior is not completely new since the study of alternate strategies has previously been ongoing for more than 2 decades; however behavioral syndromes are more specific in that the variation is high across individuals, but low within individuals (i.e. individuals are consistent or repeatable). Negative-frequency dependence, in which rare strategies perform better than common strategies, can explain persistence of different strategies in the same population (Maynard Smith, 1982a). Yet, a mixed ESS does not imply polymorphism since all individuals could be playing actions at the same rate (Maynard Smith, 1982b). However, asymmetries, such as ownership (Smith, 1982), can lead to a mixed ESS comprise of consistent individuals. Supporting this idea empirical studies have shown that personality traits are correlated with life history traits (Careau, Thomas, Humphries, & Re, 2008 Biro & Stamps, 2008). Wolf et al. (2010) previously demonstrated that repeatable behaviors can result from

games among individuals with different life-history trade-offs by modeling individuals with short versus long life-spans resulting in longer lived individuals being bolder than shyer ones across their entire life. I note that in that study, life-history strategy was fixed at the beginning of life and could not be changed. However, personality traits have been discovered in populations where there was no support for negative frequency dependence as the driving mechanism (Kурvers et al., 2011) and many asymmetries are not fixed (eg ownership, energy reserves, or size). Therefore, while game theory and the theory of mixed ESSs are integral to the study of behavioral syndromes, negative-frequency dependence alone does not always account for them.

Asymmetries in a flexible trait could result in differences in behaviors among individuals that have the same strategy (Dingemanse & Wolf, 2010). For example, daphnia that have invested into extended tail spikes can be expected to act consistently different than their non-armored counterparts; armored individuals tend to have reduced activity in comparison to their less defended counterparts (Bourdeau & Johansson, 2012). The behavioral differences between armored types should be stable throughout a daphnia's lifespan because once the commitment is made, the armor is fixed. In contrast, an individual's energy reserves and resultant behaviors can change readily throughout one's life. Energy-state-based foraging plasticity is well described by asset protection ala Clark (1994); however asset protection only leads to short-term repeatability since individuals with high energy levels will avoid foraging until their energy reserves are low and starving animals become bolder due to the risk of starvation (Clark, 1994). The avoidance of foraging by high-energy individuals will result in all individuals eventually converging on low energy and eventually leading to all individuals expressing risky tactics due to impending starvation. While a labile trait can cause a short-term expression of behaviour during a substantial part of the life cycle, can a labile trait generate a stable behavior (Reale et al. 2000; Dingemanse et al. 2002; Bell & Stamps 2004; Caspi et al. 2005; Johnson & Sih 2005; Roberts et al. 2006)?

Positive feedback loops in latent variables can stabilize individual differences and resulting in behavior not converging over long periods of time (Luttbeg & Sih, 2010; McElreath, Luttbeg, Fogarty, Brodin, & Sih, 2007). Previously, Luttbeg and Sih (2010) showed that pure conditional strategies could lead to long-term consistency when there

are positive feedbacks in size. Traditionally, this situation is called “making the best of a bad job” (Smith, 1982) where individuals will perform a strategy that maximizes their fitness, yet the relative fitness differs between strategies. For example, when males are attempting to access females for mating, a large aggressive guarder male will generally accrue high fitness. However, a small individual may accrue higher fitness when acting as a sneaker than as a guarder since a small individual would not be able to protect the female and the small individual is less detectable, making sneaking more successful. Therefore, theoretically state-dependent differences can lead to variation between individuals who differ in their state, even across long periods of time.

If a positive feedback loop in size can stabilize personalities, will personality hold across the spectrum of growth strategies during development? The goal of this paper is to explicate whether compensatory growth and non-compensatory growth strategies are both capable of generating long-term stability of behavior. Some animals grow at a constant rate throughout their lives maintaining a constant growth rate even after periods of food deprivation. In contrast, other animals when deprived of food will grow at an accelerated rate than had they not been deprived of food. , Yet, individuals who undergo compensatory growth after being food-deprived will have lower swim speeds or pay other costs for their accelerated growth rates (Farrell, Bennett, & Devlin, 1997). To understand the impact of growth strategy on repeatability, I will use a similar model derivation as Munch and Mangel (2002) in which animals grow and accrue damage due to metabolic activity Munch and Mangel’s model was capable of producing compensatory and non-compensatory growth.

Size dependent mortality is normally assumed to be associated with aquatic vertebrate (usually a fish) however this need not be so. The aphids described in the previous chapters may also show compensatory growth though it may be constrained by the moulting process (see Dyar’s Law; Hoi et al. 2014 for mosquito larvae). Further, size-dependent escape is known in aphids in a similar manner to fish although the mechanism is different (e.g. gape size vs ovipositor length). Thus, the theory introduced below is general and could be tweaked for different systems.

With this paper, I aim to address whether size will generate long-term repeatability in behaviour given that individuals can grow at an accelerated rate during compensatory growth and thus potentially decreasing variation in size near the end of development. I predict compensatory growth could reduce long-term stability of behavior by allowing smaller individuals to rapidly grow in size and match the size of their larger counterparts. This paper addresses explores the impact of compensatory growth on repeatability, by first deriving a model that is capable of generating a range of growth trajectories, secondly using *in silico* experiments to identify parameter sets that generate different patterns of growth, and finally to determine different growth trajectories impact on the long term stability of the behavior. This study will determine whether behavioral syndromes arise even in the presence of compensatory growth.

3.3. Methods

Dynamic state variable models (DSV) optimize an individual's expected lifetime fitness by calculating the optimal decisions for all possible dynamic states. Dynamic state variable models are solved by calculating backwards through time from the end of a strategy's life having defined the terminal fitness values, in a process called 'backward iteration' (Houson, Clark, McNamara, & Mangel, 1988; Mangel & Clark, 1986). Backward iteration generates an optimal decision matrix, which reports the optimal decisions for any state at any time. In this way, DSV models provide an optimal conditional strategy for each decision an individual will make based on their state(s). These decision matrices can then be used, in *in silico* experiments, to reconstruct an individual's growth trajectory and activity schedule.

3.3.1. The Model

I modeled an ontogenic period during which individuals might grow, accumulate cellular damage, choose the amount of energy to allocate to repairing damage, and to choose their foraging intensity. My model further develops the ideas of Luttbeg and Sih (2010), since I explored the stabilizing effect of size on individuals' foraging intensity, while reflecting a wide range of growth patterns seen in nature (Ali, Nicieza, & Wootton,

2003), by using a comparable framework to that of Munch and Mangel (2002). Allowing me test the impact of compensatory growth on repeatability while still This maintaining the simplicity and tractability of the model.

3.3.2. Energy dynamics

Growth was modeled as a function of size (X_t), activity (a), and energy allocated to repair (u):

$$X_{t+1} = X_t + C(a)X_t^{3/4} - R(a)X_t - u \quad (1)$$

therefore energy intake rate increases with size and foraging activity. Anabolism is a saturating function of activity intensity so that,

$$C(a) = \frac{za}{k+a} \quad (2)$$

where z is the maximum rate of energy gain and k is the activity level at which energy intake is at half its maximum. Metabolic rate is function of size and activity. Catabolism is proportional to mass (Essington, Kitchell, & Walters, 2001), while anabolism scales to the $3/4$ power of size and scales as a linear function of activity $R(a)$,

$$R(a) = 1 + a \quad (3)$$

Metabolic rates were allowed to scale up to six times of an individual's resting metabolism (Lindstrom & Kvist, 1995). The amount of energy devoted towards repair enzymes and anti-oxidants at time t is u_t . Individuals will optimize how much energy to spend on repair in each time step. Qualitatively, the energy dynamics are the similar to Munch and Mangel (2002) but I also allow for optimal allocation of energy to repair damage.

3.3.3. Damage dynamics

I assumed that damage accrues as a consequence of metabolism, therefore, as activity increases, more damage is accumulated. Unrepaired damage could lead to further deterioration of tissue. Damage could be repaired (Promislow, 1994). This results in the damage dynamics as follows

$$D_{t+1} = D_t(1 + D_r) + D_m R(a)X_t - G(u) \quad (4)$$

where D_r is the rate at which damage is reinforced. The conversion of metabolism to damage is D_m . The amount of damage repaired $G(u)$ is a saturating function of the amount of energy allocated to repair.

$$G(u) = \frac{D_{max}u}{D_{half} + u} \quad (5)$$

D_{max} is the maximum amount of damage repaired per time step while D_{half} is the half-saturation constant and determines how sensitive the amount of damage repaired is to the amount of energy allocated to repair damage.

3.3.4. Mortality

Mortality is dependent on an individual's activity, size, and accumulated damage (Roitberg and Mangel 2010). Instantaneous mortality increases as individuals become more active. Individuals who have accrued more damage will experience higher mortality rates (e.g. fish that undergo compensatory growth have lower sprint swimming rates increasing their mortality rates (Ali et al., 2003). Finally, as individuals become larger, their predation risk decreases linearly.

$$M(X_t, D_t, a) = m \left(1 - \frac{X_t}{X_{max}}\right) \left(1 + \frac{a}{a_{max}}\right) \left(1 + \frac{D_t}{D_{max}}\right) \quad (6)$$

3.3.5. Terminal Fitness

The terminal reproductive value $V(X, D)$ is a function of size and the accumulation of damage. I assume that residual reproductive value increases with size and decreases with damage:

$$V(X_T, D_T) = X_T^{\theta_1} (1 + D_T)^{-\theta_2} \quad (7)$$

where θ_1 is the sensitivity of fitness to size and θ_2 is the sensitivity of fitness to damage.

3.3.6. Dynamic Equation

A strategy's fitness is a function of the probability of surviving through the focal interval and its final size and damage at the end of the period when it matures and can reproduce. Instantaneous survival is the complement of $M(X, D, a)$. The survival rate $1 - M(X_t, D_t, a, t)$ is an immediate discounting of the fitness at each time step while $F(X_t, D_t, t)$ is the potential future fitness. Then, using backwards iteration, individuals will choose their foraging intensity (a), and the amount of energy allocated to repair (μ) to maximize their fitness at each time step and state combination. From the dynamics, fitness function, and instantaneous mortality the dynamic equation is.

$$F(X_t, D_t, t + 1, T) = \max_a \{ \max_u \{ (1 - M(X_t, D_t, a)) F(X_t, D_t, t, T) \} \} \quad (8)$$

The dynamic equation is solved by maximizing $F(X, D, t, T)$ by choosing the optimal activity and repair values for all of $[0, T]$.

3.3.7. Forward Simulation

3.3.8. Construction of growth trajectories

I constructed optimal growth trajectories from the optimal decision matrices and the size (equation 1) and damage dynamics (equation 4). All of the trajectories were started at a small initial size and zero damage. Then by referencing the optimal decision matrices, an individuals' foraging intensity and the amount of energy allocated to repair were determined. I then inserted the foraging intensity and repair values into the energy and damage dynamics equations (1) and (4), to generate optimal growth and damage trajectories. If generated values fell between the calculated size and damage values linear interpolation was used (Mangel and Clark 1988; Clark and Mangel 2000). Then by recursively using this procedure the growth trajectories and activity schedules could be constructed until the end of time ($t = T$).

3.3.9. Identification of growth trajectory type

After generating the decision matrices, *in silico* experiments were used to identify parameters that would generate either normal or compensatory growth trajectories. For each parameter set, three growth trajectories were compared to diagnose whether compensatory growth occurred and what the mechanism behind the compensatory growth was. The first growth trajectory constructed allowed individuals to grow from a small predetermined size (2.5 units) and low damage (0.1 units) and no manipulations were applied to this treatment. This trajectory was then compared with a growth curve where individuals were given only a maintenance ration. Food deprivation was simulated by holding size constant until $t = 15$ and during this time individuals would accumulate damage, since their metabolism was still active, but they would not grow. At $t = 15$ deprivation period ended and normal growth was allowed to resume. The food deprivation treatment is similar to the way compensatory growth would be studied empirically. The third growth trajectory was constructed to see if individuals were growing faster due to time limitation after deprivation or the high accumulation of damage at a small size. The time-limitation trajectory was constructed by starting an

individual at the same size as the individual who had experienced the deprivation period but had no accumulated damage.

Growth trajectories were categorized by their growth behavior after being deprived of food into compensatory or non-compensatory growth. To identify parameters that generated compensatory growth, I compared the growth of individuals who had been deprived of food and individuals who had been allowed to grow normally. I confirmed compensatory growth when individuals exhibited faster-than-normal growth immediately following the end of the deprivation period (short-term compensation) (Ali et al., 2003). I determined whether growth trajectory was induced by time-limitation or damage by comparing whether individuals grew faster based on damage accumulated. Using this treatment the food-deprived individuals could be compared with the time-limitation treatment and if only the food deprived individuals showed accelerated growth compensatory growth was caused by damage. If both food-deprived and the time-limitation trajectory should compensatory growth then approaching the time horizon is triggering compensatory growth. Time-limited individuals were defined as those that grew at the same rate as individuals who increased growth after food deprivation but grew at the same rate as the time-control treatment.

3.3.10. Stability of Behaviour

The stabilizing effect of size on foraging intensity was closely examined by selecting three case studies; one with no compensatory growth and two with compensatory growth to see if rank-order was maintained across development. The two types of compensatory growth differed as to whether such growth was due to damage-accumulation or time-limitation. Rank-order of foraging intensity was then examined by starting individuals from 0 to 10 size units on a 0.1 interval. I then constructed growth trajectories and activity schedules for all the individuals. Over the course of the individuals' development, rank order could be observed and determined if the rank-order of activity intensity remained over the course of development. The dynamic programming model was solved in C with standard libraries. All graphics were generated using base graphics in the R 3.0.3 programming environment

3.4. Results

3.4.1. Description of Growth Trajectories

Non-compensatory growth and compensatory growth were both prevalent in the explored parameter space however; non-compensatory growth dominated most of this space. Some individuals, after being food deprived, did not increase their foraging intensity but returned to their previous growth such as in Figure 3.1a. While in Fig 3.1b individuals will undergo damage-induced compensatory growth since the food-deprived individuals grew faster than the time-control and the normal growing individual. In Fig 3.1c, time-induced compensatory growth is seen because individuals who were small in size regardless of their damage levels (the time-limitation and food-deprived individuals grew at the same rate) increased their growth over the normal growth. I did not vary deprivation length nor did I thoroughly investigate the parameters that led to compensatory growth since this process was developed for finding qualitatively different conditional strategies in which compensatory growth occurred (for a deep analysis of the parameters leading to compensatory growth see Munch and Mangel (2002)).

3.4.2. Decision Matrix

In figure 3.2, I report the decision matrices for the three growth types and how they change through time. The optimal foraging intensity changes little as time progresses when individuals do not undergo compensatory growth Fig 3.2 a-c. In contrast, foraging intensity should increase in small individuals as time progresses however the increased growth happens regardless of accumulated damage Fig 3.2 d-f. The decision matrices of parameter sets that had damage-induced compensatory growth differed systematically from the non-compensatory and compensatory foraging decisions since damage had an interaction with size. Small individuals that have low damage should forage at a low rate but as damage levels increase individuals should increase their foraging intensity Fig 2.3 g-h. The interaction between size and damage explains why only the food-deprived individuals increased their growth rate and not the individuals of the same size who lacked damage Fig 3.1 c. When removing the effect of damage,

we return to a model similar to Luttbeg & Sih (2010). Next, I report the decision matrix for compensatory growth.

3.4.3. Repeatability of Behaviour

Rank-order of foraging intensity was explored in individuals who varied in their initial size to see if size maintained the boldest individuals rank across all development. When compensatory growth did not occur, the largest individuals always corresponded with the most active individuals across all development (Fig. 3.3b). In Figure 3.2a-c, it is always optimal for larger individuals to have a high foraging intensity. Having larger individuals forage at higher rates leads to larger individuals always being bolder than their smaller counterparts (Fig 3.3a) resulting in the rank-order of foraging behavior is also maintained. Damage induced compensatory growth did not occur in this experiment since all individuals started life at low damage values therefore the growth trajectory was similar to Fig 3.3a and the activity schedule was comparable to Fig 3.3b. However, individuals that underwent compensatory growth due to time limitation maintained rank order in foraging intensity was not maintained throughout development (Fig 3.3d). When looking at the growth trajectories (Fig 3.3c) of these individuals, the initially largest individuals retained their rank but the small individuals appeared to catch up. When looking at the activity schedule, smaller individuals were seen to have high activity levels early in development and then dropped following this burst (Fig 3.3d). While initially large individuals had low foraging activity, their foraging intensity increased monotonically over development until they surpassed their smaller counterparts. The activity schedules of small and large individuals cross and this inverts their rank order between early and late development. Therefore, we see size can stabilize behavior in development but compensatory growth vs non-compensatory growth changes whether size will maintain the rank-order of foraging intensity across all of development.

3.5. Discussion

Explaining why individuals are consistent across development is one of the main challenges of behavioral ecology since during development, an individual's morphology,

physiology, and neurological traits are rapidly changing. However, behavioral syndromes have been found to be stable across life stage transitions, such as metamorphosis (Tremmel & Muller, 2012), and stable across long periods of time in indeterminate-growing animals (Moretz et al., 2007). Luttbeg and Sih (2010) showed that behavioral syndromes could occur during an ontogenic period when positive feedback loops dominate over negative feedback loops stabilizing variation in state-dependent differences and hence behavior. However, their model only considered size, which limits the diversity of growth trajectories (Marc Mangel & Munch, 2005) and they did not explore the differences in selection pressure during early and late development. Therefore how compensatory growth affects the stability of behavior across development is still not well understood.

Similar to Luttbeg and Sih (2010), I found initial differences in size can stabilize behaviors of individuals, whom do not undergo compensatory growth. Larger individuals are consistently more active than their smaller counterparts and the rank order of activity is maintained throughout ontogeny (Fig 3.3d). If individuals are given enough time, they will all eventually converge to the same size, removing variation between individuals due to constraints on maximum size. However, in nature, individuals rarely reach their maximum size due to time constraints (Day & Rowe, 2002) or other conflicting selective pressures. The time at which individuals matured (the end of time) was always fixed for these strategies and as such, individuals did not decide when to mature. However, the decision of when to mature is extremely important for many if not most organisms (Day & Rowe, 2002) and could potentially reinforce differences even further as to whether individuals subscribe to growing fast or slowly (Dmitriew, 2011). I was able to confirm the prediction that individuals that do not complete compensatory growth have stable behaviors across their development.

Damage accumulation and time-limitation both generated compensatory growth in the food deprivation trials. Time-limited compensatory growth occurs when individuals grew faster near the end of time as seen in Figure 3.2 d-f. Individuals who performed this type of compensatory growth grew faster than their non-deprived counterparts, but grew at the same rate as individuals who were the same size but had accrued no damage. In time-limited compensatory growth an approaching time-horizon triggered

compensatory growth in small individuals resulting in the smaller individuals becoming increasingly bolder as the time horizon approaches (Fig 3.3d-f). While increased activity to avoid starvation at extremely low sizes is present in the decision matrices of parameter sets that have non-compensatory growth (Fig 3.2a) the high levels of foraging intensity the size threshold of beginning high foraging intensity foraging is reached at continually larger sizes as time progresses under time limited compensatory growth(Fig 3.3d-f). Damage induced compensatory growth occurs when individuals increase their activity based after accumulating high levels of damage (Fig 3.2g). Individuals at high levels of damage and small size will grow faster than their healthier counterparts of the same size (Fig 3.1c and Fig 3.2 g-i). This strategy allows individuals to compensate for the damage they have accrued; additionally individuals could begin to repair their high levels of damage once they had reached a large size. However, damage-induced compensatory growth only occurs in small and heavily damaged individuals Fig 3.2 g-i.

Unfortunately, the current experimental protocols would not be able to tease these two mechanisms apart. But, two experimental approaches could test which mechanism is causing compensatory growth. First, the compensatory growth due to time-limitation could be simulated by tricking animals into perceiving they are approaching the end of their life making them time-limited (Roitberg, Sircom, & Roitberg, 1993). If individuals increased their growth rate due to perceiving they are near the time horizon, small individuals should greatly increase their growth rates. While damage induced compensatory growth could be generated by creating oxidative stress in animals and see if high oxidative stress triggers compensatory growth (De Block & Stoks, 2008). Compensatory growth was generated both by high damage accumulation and individuals having a limited time left to forage.

How does compensatory growth and time-limitation versus damage-generated compensatory growth affect stability of foraging activity? In the case where compensatory growth is caused by high levels of damage accumulation, I found that size stabilizes foraging intensity across the entire period of development. The maintenance of rank-order of foraging intensity across development is maintained since no compensatory growth occurs. Activity intensity did not increase since all the individuals started at the same amount of damage accumulation, negating difference in size among

individuals (Fig 3.3b). But, if individuals were allowed to start along a distribution of damage (as we might expect in nature) we might begin to see compensatory growth. In contrast time-limited compensatory growth resulted in the inversion of foraging intensity regardless of damage. Small individuals initially grew slower but increased their activity rapidly to grow faster than their larger counterparts. However, in time-limited compensatory growth, we see that individuals accelerate their growth by increasing activity due to being small in size (Fig 3 b). After this short-burst of increased activity, smaller individuals' activity decreased and their larger counterparts once again foraged more boldly. Interestingly, after the period of accelerated growth activity, the rank ordering of activity was maintained for the rest of development. This suggests that time-limited compensatory growth leads to short-term repeatability in behavior but when comparing between early and late development there would be little repeatability seen due to the inversion in activity intensity from early to late development. This study really highlights that instead of investigating repeatability *per se* it is important to analyze repeatability throughout life and to elucidate repeatability as individual reaction norms (Stamps & Groothuis, 2010).

My work shows that short-term repeatability occurs within both early and late development but not so for long-term behavior under compensatory growth. Therefore, it is important to begin empirically studying foraging across all of development and determine whether behavioral consistency is maintained across development. When two sampling events are used, one early and the other late in development, no repeatability would be detected. However, if the investigator sampled multiple times in early and late development the rank-order of foraging intensity would be predictable but simply inverts between early and late development. The study of growth trajectories and the corresponding foraging schedule generates the testable hypothesis that species that don't undergo compensatory growth should have stable behavior due to size, if positive feedback loops dominate. However, in species that undergo compensatory growth we should expect to observe inversions in foraging rate between early and late development in cases where individuals undergo compensatory growth. Additionally, regardless of growth strategy at large sizes, size-rank ordering of smallest to largest individuals with largest being the most bold will be maintained throughout development. Empirical studies need to address behavior across development in order for behavioral ecologists

to begin constructing the foraging schedule of individuals and to determine whether behavioral syndromes are actually occurring.

Using dynamic programming models to study variation in behavior is extremely informative and can be used to study additional growth types, across life-stage boundaries and, even investment into other latent variables. Since this model just focused on development we might also consider the transition from a juvenile into a reproductive adult and ask whether we should expect behavioral consistency over maturation. Dynamic programming can be used to study how repeatability of behavior occurs in animals that metamorphose or have separate instars (Kralj-Fiser & Schuett, 2014). For example, one might study whether behavioral repeatability as a larval stage should carry over into being an adult. While this paper focused on expanding a few types of growth trajectories that are known for fish, there are 11 suggested growth trajectories across a broad range of taxa (Ali et al., 2003); we should begin to study the pattern of behaviour across these other growth strategies. Additionally, different latent variables could be studied. A fruitful area of study would be studying inducible immune systems investment which can prevent disease allowing individuals to be bolder but in contrast to size investment can be reduced after. Potentially the plasticity in induced defences could generate only short-term repeatability. This model provides a modeling framework for being able to study how state-dependent differences do or do not generate behavioral syndromes in animals.

In conclusion, I show that size can stabilize personalities when individuals are capable of short-term accelerated growth while reducing stability of behavior overall. This does not mean compensatory growth reduces the predictability of behavior and it highlights that we can move past the moniker of personality and instead study individual reaction norms (Stamps & Groothuis, 2010). Personality is a subset of a greater phenomenon known as a behavioral reaction norms or individual reaction norm. Just as variation between genotypes and their interaction with the environment can be studied the same approach can be used to study behavior at the individual level (Nussey et al., 2007). Using a behavioral reaction norm approach we can study whether individuals differ in their behavioral across development. This approach can be extended to study whether individuals have different plasticity or mean level effects across time or even

different environments. Potentially, we could begin to see individuals who are sensitive to environmental changes while others are insensitive. This study shows how differences in life histories are important for determining the mechanism that generates personality.

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3.7. Tables & Figures

Table 3.1. Parameter and variables names and values used in the model.

The minimum and maximum values and the description of all parameters and dynamic variables. The specific parameter values that led to non-compensatory growth, damage-induced compensatory growth, and time-limitation induced compensatory growth used in depth studies are also reported.

Variable or Parameter symbol	Description	Min Value	Max Value	Non-compensatory growth	Damage Induced Compensatory Growth	Time limitation induced compensatory growth
X	Body size	0	50			
D	Accumulated Damage	0	10			
a	Foraging intensity	0	6			
z	Maximum consumption rate	-	-	0.61	0.61	0.61
k	Half consumption rate	-	-	0.10	0.10	0.10
D_r	Damage Reinforcement	-	-	0.001	0.001	0.001
D_m	Metabolism to damage conversion	-	-	0.01	0.01	0.01
λ_D	Damage-dependent mortality	-	-	0.001	0.001	0.001
D_{max}	Maximum repair rate	-	-	7	7	7
D_{half}	Half-saturation of repair	-	-	1	1	1
Θ_1	Fitness size	-	-	2.931	2.931	2.931
Θ_2	Fitness damage	-	-	2.931	2.931	2.931

Figure 3.1. Sample growth trajectories of strategies with compensatory and non-compensatory growth.

Sample growth trajectories of the three types of growth patterns a) Non-compensatory growth: Individuals grow at the same or lower rate than both the time and damage control. b) Damage-induced compensatory growth: Compensatory growth due to damage accumulation c) Time induced compensatory growth Compensatory growth due to time limitation

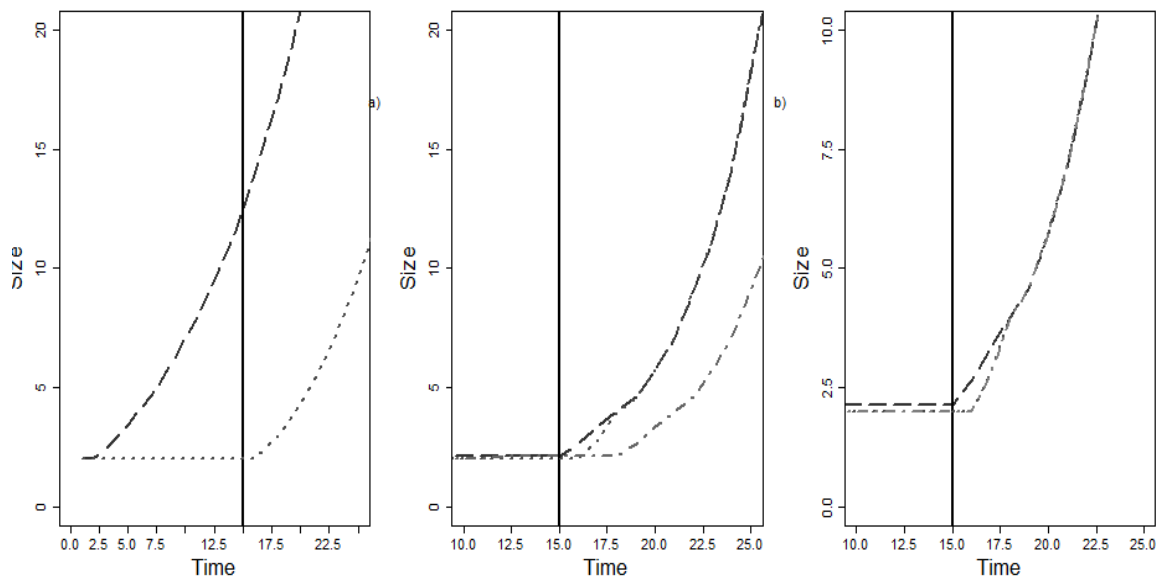


Figure 3.2. Optimal foraging intensity matrices from the solved dynamic state variable model.

The left column shows the optimal decisions at $t = 0$, the centre column shows at time = 15, and the right column is the foraging values at $t = 25$. Light grey represents low foraging intensity and higher foraging intensity is shown in darker greys. a-c) Non-compensatory growth d-f) Damage induced Compensatory Growth g-j) Time limitation compensatory growth

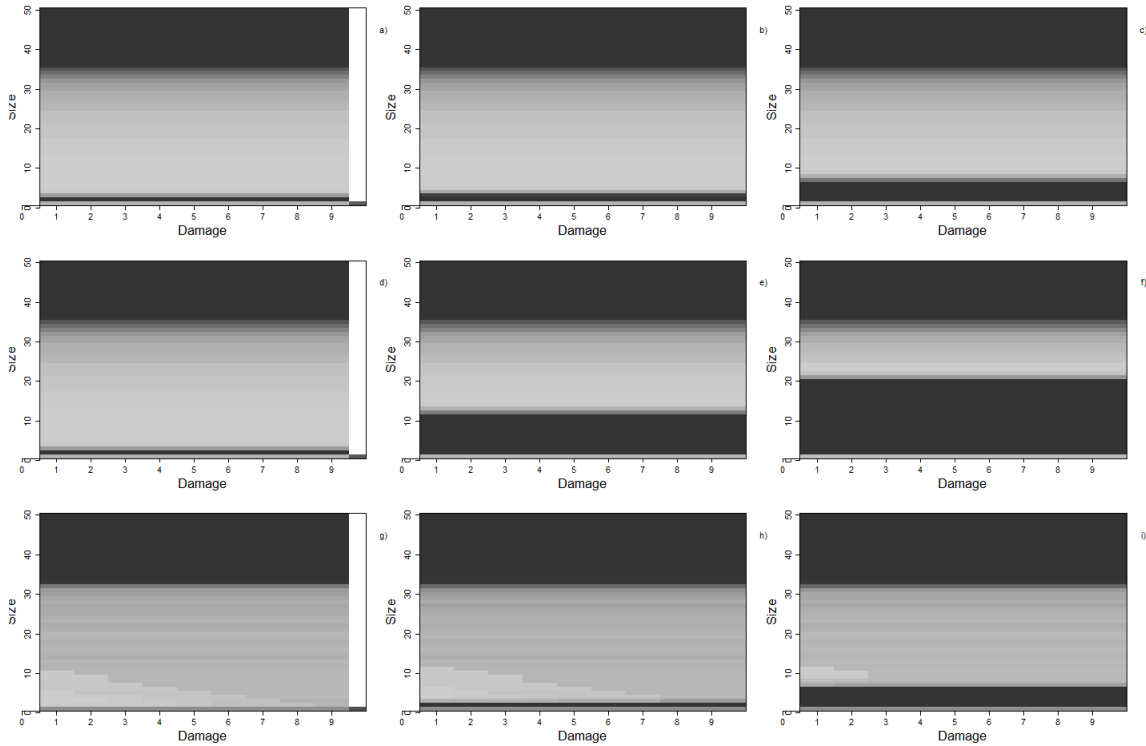
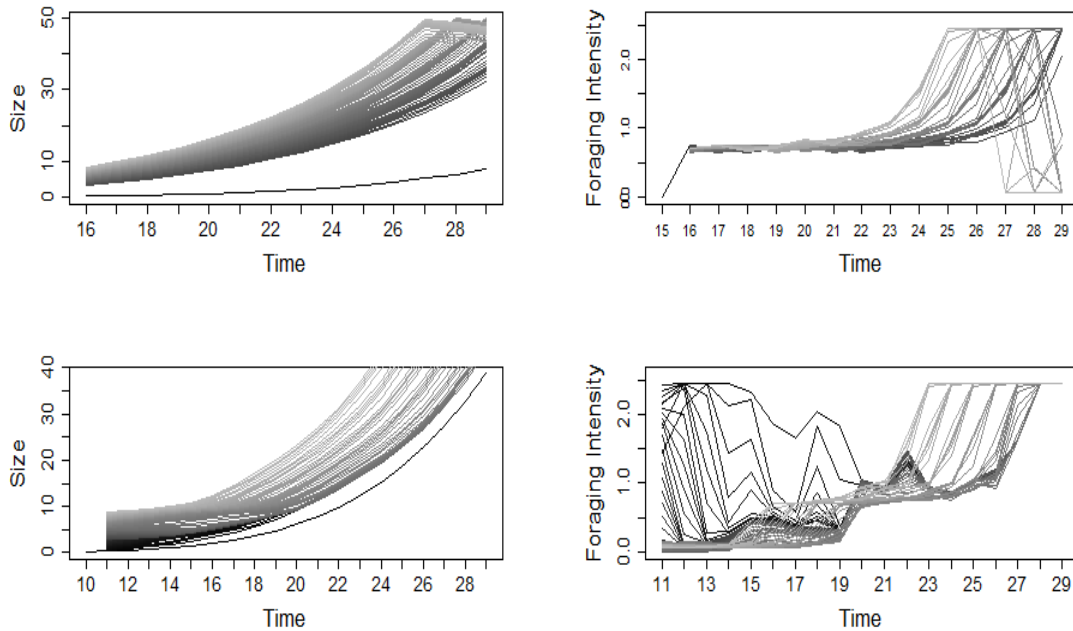


Figure 3.3. Effect of initial size on growth trajectories and optimal activity schedules.

Individuals starting at a small size are dark grey while individuals started at an initially larger size are lighter grey. a) Non-compensatory growth b) Non-compensatory growth c) Time-induced limitation compensatory growth d) Time-induced limitation compensatory growth



Chapter 4.

Conclusion

I show that we need to disentangle the study of repeatability and the correlation among behaviors because the focus on correlated traits has hidden the complexity possible in variation in a single trait. First, we see that repeatability appears readily among genetically identical pea aphids in the absence of a coalescent trait complex. High repeatability of behavioral traits is common (Smith & Blumstein, 2007). When investigating how size can play a role in stabilizing behavior I found not only were a lot of individuals consistent across development but when individuals underwent compensatory growth the smallest individuals early in life were the boldest before switching to becoming the least bold later in life. A pattern in the behavioral syndromes paradigm would be simply called not a behavioral syndrome or inconsistent. However others have begun pushing for understanding the other patterns of variation among traits (Nakagawa & Schielzeth, 2010) but using the behavioral reaction norms framework.

In chapter two, I show that repeatability but not multiple correlated traits can arise within an aphid clone proving that repeatability can be caused by non-genetic factors. Using three separate aphid clones I show that aphid clones may differ on a population level between hot/dry and cool/humid areas with aphids from hot areas minimizing contact with the ground. However, within clones there is still a lot of variation yet, the individual aphids are very consistent. However, there were no correlated traits among behavioral traits. Luttbeg & Sih (2010) predict that repeatability should be a function of environmental quality but repeatability and correlations did not change with environmental quality, providing evidence that maternal effects were generating personalities. Personalities have been found to be generated by maternal effects in other systems and aphids dropping behavior has previously been shown to change due to maternal effects (Andrade & Roitberg, 1995). I suggest that pea aphids are a model system for studying the interaction between personalities and maternal effects due to

known mechanisms, and their clonal nature. Additionally, with such a system, we can begin to analyze behavioural syndromes existing within clones (non-genetic) or across clones (genetic).

In chapter three, I studied foraging intensity during development when individuals are rapidly changing in size. Luttbeg & Sih (2010) predict that asymmetries in size should stabilize individuals foraging rate across development due to positive feedback loops in size. Large individuals will experience lower predation rates allowing them to forage at higher rates than their smaller counterparts. However, commonly in nature smaller individuals will grow at disproportionately rapid rates after being deprived of food in a phenomenon known as compensatory growth. However, when I investigated whether individuals that underwent non-compensatory and compensatory growth, I found that both maintained rank-order of their behavior across development, except for a subset of the individuals who underwent compensatory growth. Those individuals who did not maintain rank order underwent compensatory growth but due to time-limitations. Therefore, small individuals initially grew faster than their larger counterparts but slowed down over time. Eventually, the initially larger individuals would become the boldest. Therefore while individuals are not repeatable over the long-term over early or late development, a rank order of behavior was maintained. This research lends support to studying long-term vs short-term repeatability. We can expand the idea of personalities to the study of behavioral reaction norms.

Instead of simply analyzing short-term vs long-term repeatability I suggest we expand repeatability to a behavioral reaction norm framework. This framework deemphasizes plasticity and since individuals' behavior can be measured multiple times throughout their life, the variability between individuals average response and plasticity can be studied. Currently very few studies have measured the same individuals behavior across environmental contexts to see if some individuals are more plastic than others (Nussey et al., 2007). Potentially, the rank order of behavior could be constant across environments or we could see plasticity in traits and variation in plasticity. Few studies have studied variation in plasticity in individuals. Unfortunately, experiments about multiple behaviors are so unwieldy that in depth questions about single traits are

difficult to address properly. However, this variation in plasticity of which personality is just a subset is a fruitful area of study.

Theoretical models for population dynamics have traditionally treated animals as having inflexible behaviour, although allowing flexible behaviour changes the dynamics of a population. These still treat individuals within a population as being flexible in their behaviour, but assume all individuals are identical. The behavioral ecology literature, however suggests that variation between individuals in flexible behaviour is the rule, not the exception (Andrew Sih et al., 2004). The mechanisms for generating behavioral syndromes can either be evolutionary or ecological (Wolf & Weissing, 2010). These mechanisms operate at different time scales, and will generate different patterns of variation. Since variation in traits can fundamentally change population dynamics, it is important to build theoretical models that predict the effects of variation in a trait. Therefore the mechanism underlying behavioral syndromes will determine the effect on evolution or population dynamics. For example, when dispersal depends on both the individual's personality and the ecological context, the phenotypic composition of potential dispersers (both mean phenotype and the mix of behavioral types), which, in turn, can have important effects on the dynamics of spatially structured populations (Cote, Clobert, Brodin, Fogarty, & Sih, 2010). For example dispersal and aggression can be correlated (Duckworth, 2006) and lead to a wave of range expansion, with highly aggressive individuals arriving expanding a population's range while also displacing competitors (Duckworth, 2009). The very real possibility that a mix of behavioural types may change population dynamics highlights the necessity to understand behavioral syndromes and their impact on higher-level processes.

Closing remarks

This thesis demonstrates that while we have learnt a lot about behavioral syndromes, the study of behavioral syndromes is just a subset of behavioral reaction norms. The study of behavioral syndromes has developed considerably (Andrew Sih et al., 2004) but it opens up new areas of study (Nussey et al., 2007; Wolf & Weissing, 2010; Cote et al., 2010). Including behavioral reaction norms, the continued study of proximate mechanisms genetics, maternal effects, and an individual's state can result in

long term stability of behavior and whether we expect coalescent trait complexes or not. While behavioral ecologists do not fully understand the mechanisms generating behavioral syndromes, we continue to move forward.

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