

# Separate and combined effects of nutrition during juvenile and sexual development on female life-history trajectories: the thrifty phenotype in a cockroach

Emma L. B. Barrett, John Hunt, Allen J. Moore  
and Patricia J. Moore\*

*Centre for Ecology and Conservation, School of Biosciences, University of Exeter,  
Cornwall Campus, Penryn, Cornwall TR10 9EZ, UK*

We have yet to understand fully how conditions during different periods of development interact to influence life-history structure. Can the negative effects of poor juvenile nutrition be overcome by a good adult diet, or are life-history strategies set by early experience? Here, we tested the influence and interaction of different nutritional quality during juvenile and sexual development on female resource allocation physiology, life history and courtship behaviour in the cockroach, *Nauphoeta cinerea*. Nymphs were raised on either a good-quality or poor-quality diet. After adult eclosion, females were either switched to the opposite diet or remained on their original diet. We assessed mating behaviour and lifetime reproductive success for half of the females from each treatment. We evaluated reproductive investment, somatic investment and resource reallocation from reproduction to the soma via oocyte apoptosis in the remaining females. We found that poor juvenile conditions resulted in a fat phenotype with slow juvenile growth and short reproductive lifespan that could not be retrieved with a change in diet. Good juvenile conditions resulted in the converse, but again fixed, phenotype in adulthood. Thus, juvenile nutrition sets adult patterns of resource allocation.

**Keywords:** compensation; condition; growth; life history; lifespan; thrifty phenotype

## 1. INTRODUCTION

In a changing environment, plasticity allows organisms to respond adaptively to existing conditions. There are limits, however, as plasticity has to trade-off with life-history constraints. Furthermore, current life-history traits may trade-off against other past, current or future traits (Beckerman *et al.* 2002; Taborsky 2006; Boggs 2009). It is therefore important to consider the entire life history of an organism when examining potential plasticity and its limits.

The effect of poor early life conditions on fitness and adult traits are well documented and include: reduced size and increased age at maturity (Abrams *et al.* 1996), smaller energy reserves (Birkhead *et al.* 1999), inferior competitive ability (Hunt *et al.* 2005), decreased immunocompetence (Birkhead *et al.* 1999) and increased stress related pathology (Lummaa & Clutton-Brock 2002; Ozanne *et al.* 2004). Less clear is the extent to which organisms can overcome a poor early start (e.g. Blount *et al.* 2006). The response to early life conditions and the capacity for plasticity are known to be highly variable between species (Metcalf & Monaghan 2001; Ali *et al.* 2003; Dmitriew & Rowe 2006; Monaghan 2008). Further, although the adult response to early life conditions is well characterized in vertebrates (Lindström 1999), less work has been done to elucidate its effects within other animals (Nylin & Gotthard 1998; Ali *et al.*

2003; Dmitriew & Rowe 2006). Defining how life-history traits respond to variation in environments across developmental stages in a variety of taxa is needed to increase our understanding of when there can be plasticity in life-history trade-offs (Boggs 2009).

When considering how early experiences trade-off with later traits, the life cycle of the study species may be important. For example, the nutritional niches of juveniles and adults differ dramatically for many insect species, with a period of complete tissue remodelling associated with entering the adult niche (e.g. holometabolous insects such as flies, beetles, butterflies or hetero-hemimetabolous insects such as dragonflies, mayflies). For these species, conditions experienced as juveniles may not be informative of adult conditions, and thus juveniles may be predicted to curtail development in order to benefit from improved adult conditions. Studies investigating the effects of early life nutrition on life-history traits in insects in which metamorphosis is accompanied by a change in niche show that periods of food restriction throughout juvenile development are associated with a smaller adult body size (Blanckenhorn 1998; Boggs & Freeman 2005), reduced nymphal and adult fat stores (Dmitriew & Rowe 2005; Stoks *et al.* 2006) and reduced adult starvation resistance (Gotthard *et al.* 1994; Dmitriew & Rowe 2006).

In contrast, the conditions experienced as juveniles are a good indicator of the adult environment in species where juveniles and adults share a common niche (pauro-hemimetabolous insects such as cockroaches, earwigs, grasshoppers and true bugs). The increased

\*Author for correspondence (p.j.moore@exeter.ac.uk).

information available as juveniles regarding the quality of the adult environment leads to the prediction that poor conditions may lengthen development so that individuals can accumulate resources for use in adult activities such as reproduction. However, greater predictability of the adult environment may also result in reduced ability of adults to respond to a change in diet quality (Velasco & Walter 1993). However, there are few studies on the effects of early life nutrition on life-history plasticity in pauro-hemimetabolous insects to test these predictions.

Here, we tested the hypothesis that a pauro-hemimetabolous life history leads to reduced plasticity under environmental variability by examining the relative effects of diet quality at two key demographic developmental stages, juvenile development and sexual maturation, on the life history of female cockroaches of *Nauphoeta cinerea*. Previous studies on *N. cinerea* have shown that females evaluate food availability during sexual maturation and use this information to alter adaptively reproductive strategy (Barrett *et al.* 2009). Individuals were either maintained on the initial juvenile diet or switched to the opposite diet for the period of sexual maturation prior to mating. We found that when food quality changed upon adult emergence, females followed the same allocation patterns as determined by juvenile conditions and females of poor juvenile backgrounds were less able to compensate than those that had been reared in good conditions. Our findings give initial evidence that insects that share a common niche throughout their life history have opposite life-history priorities to those that change niche, and that resource allocation pathways appear to be constrained by juvenile conditions, limiting the animal's ability to respond to new conditions.

## 2. MATERIAL AND METHODS

### (a) *Animal husbandry and experimental design*

We manipulated diet quality in two life-history stages; juvenile development (from first instar to adult eclosion) and sexual maturation (from eclosion to 18 days, Barrett *et al.* 2009). Juvenile and sexual maturation diet was either a poor-quality unbalanced diet (P), or a good-quality balanced diet (G). Thus, we had four diet manipulation combinations (PP, PG, GG, GP,  $n = 150$  for each). The good-quality diet consisted of 50 per cent fish food (Kusuri Super Growth, 63% protein, 8.1% fat, 0.69% carbohydrate) and 50 per cent oatmeal (Mornflake, 16.9% protein, 11% fat, 22% carbohydrates), whereas the poor-quality diet contained 100 per cent fish food. Little is known about the nutritional ecology of *N. cinerea*. Many cockroach species are omnivores with diets rich in nitrogen, store protein and prefer high carbohydrate diets (Cochran 1985; Cohen 2001; Jones & Raubenheimer 2001; Kopanic *et al.* 2001; Raubenheimer & Jones 2006). Further, high-protein diets can result in increased mortality and morbidity (Cochran 1985; Jones & Raubenheimer 2001; Boersma & Elser 2006; Raubenheimer & Jones 2006).

We produced the diets using oatmeal and high-protein fish food bound together with water, or fish food only in water. The mixture was forced into 0.25 cm holes in a 30 × 30 cm piece of Perspex mounted on an MDF board of the same size, and baked into pellets at 70°C. The most substantial differences between the two diets are in the quantities of protein and carbohydrates. However, fish food and

oatmeal differ in other respects (e.g. vitamins and minerals, oils of marine and vegetable origin, Zajitschek *et al.* 2008). This means that we did not exclusively manipulate protein or carbohydrate content *per se*, thus we refer to the diets as good (G) and poor (P) quality.

We maintained mass colonies and all experimental animals of *N. cinerea* in incubators under standard rearing conditions of 28°C with a 12/12 photoperiod with ad libitum access to water and our standard food source (rat & mouse expanded diet; B & K Universal Ltd., Hull, UK). We isolated experimental individuals as first instar nymphs, sourced directly from the large mass colonies, and assigned nymphs randomly between the four diet manipulation treatments. We collected 150 first instar nymphs for each diet treatment. There was no significant difference in the size of the nymphs allocated to each treatment (ANOVA,  $F_{1,596} = 0.575$ ,  $p = 0.632$ ). At this stage, nymphs cannot be sexed, consequently some nymphs were males (approx. 1 : 1 sex ratio), and some nymphs also died during development, thus resulting in a different number of females available for our sexual maturation diet manipulation (PP = 57, PG = 57, GP = 75, and GG = 76).

### (b) *Female reproductive and somatic condition*

We measured mass and pronotum length (as a proxy for size) at adult eclosion of all females. We measured mass again at 18 days post-adult eclosion (end of the sexual maturation period), at which point we ended the diet manipulation. At 18 days post-adult eclosion, we dissected approximately half of the females from each of the four treatment groups to assess ovarian development state and size of fat stores. This involved dissecting the ovaries and the fat body from each female. We immediately assayed one ovary, chosen at random, for levels of apoptosis using the Vybrant Apoptosis Assay Kit No. 4 (Molecular Probes, Eugene, OR) as described by Moore & Sharma (2005). Apoptosis assays were used to indicate whether resources were being reabsorbed from oocytes (Barrett *et al.* 2008). We used the remaining ovary and the fat body for measuring dry biomass. We obtained dry mass by weighing 2 × 3 cm strips of foil that had been dried in a drying oven for 12 h to remove residual moisture using a UMX2 ultra-microbalance (Mettler-Toledo Ltd. Leicester, UK), mounting the dissected organs on the foil, allowing specimens to desiccate in a drying oven for 48 h at 70°C, and then weighing both the foil and the dried organ. The foil mass was deducted from the total mass to calculate the dry organ mass.

### (c) *Female receptivity, courtship and reproductive success*

We used the remaining adult females from each of the four treatment groups in mating trials at 18 days to assess fecundity and longevity. At 18 days, all females are sexually receptive, even under poor nutritional conditions (Barrett *et al.* 2009). We placed each experimental female into a 17 × 12 × 6.5 cm clear plastic mating arena with a randomly assigned virgin 10-day-old male. We observed and recorded courtship and mating behaviour using the methodology of Clark *et al.* (1997). Most receptive females respond to male courtship display within minutes. We observed all pairs until the start of copulation, or for 20 min if copulation did not occur. We allowed courtship to continue for 20 min rather than the 5 min shown to be sufficient to indicate long-term unwillingness to mate (Moore 1990), owing to

the potential effect of dietary stress on female receptivity. We measured male willingness to court experimental females as the time between approach and the initiation of courtship. We measured female choosiness as time from initiation of courtship to start of copulation. In *N. cinerea*, female mate choice depends on an absolute threshold rather than relative differences among males (Moore & Moore 1988). Thus, courtship speed is an indicator of mate preference (Clark *et al.* 1997). We returned mated females back to individual containers. All females were provided with ad libitum food (rat chow) and water and returned to standard rearing conditions following mate trials. *Nauphoeta cinerea* is ovoviviparous and gives live birth, and we checked females daily for parturition of offspring. We removed clutches from the female on the day of birth and counted the number of offspring born. After each clutch was removed, we returned females to their container to await the birth of the next clutch. We observed females until their death, which we recorded. We analysed development time, adult longevity and total lifespan using a Kaplan–Meier survival analysis and tested for significant differences between diet manipulations using the Cox Regression Tarone–Ware log rank test.

### 3. RESULTS

#### (a) Effect of juvenile and sexual maturation diets on growth and survival

The quality of diet during juvenile development, but not during sexual maturation, affected the distribution of time spent at different developmental stages, but not overall survivorship (figure 1). Nymphs raised on the poor-quality diet were more likely to die prior to eclosion than nymphs raised on the good-quality diet (Tyrone–Ware  $\chi^2 = 40.59$ , d.f. = 1,  $p < 0.001$ ). Of those that survived to adult eclosion, nymphs that were fed the poor-quality diet took longer to develop into adults than nymphs raised on a good diet. For these individuals, the time spent between isolation as first instar nymph and adult eclosion was significantly affected by juvenile diet (figure 1a; Tyrone–Ware  $\chi^2 = 61.117$ , d.f. = 1,  $p < 0.001$ ). Juvenile diet quality (Tyrone–Ware  $\chi^2 = 24.649$ , d.f. = 1,  $p < 0.001$ ), but not the quality of diet during sexual maturation (Tyrone–Ware  $\chi^2 = 0.094$ , d.f. = 1,  $p = 0.759$ ) also affected female adult survival (figure 1b). Juvenile diet irreversibly influenced adult lifespan; females fed the poor-quality juvenile diet had a short adult lifespan, and females fed the good-quality juvenile diet had a long adult lifespan. However, there were no overall differences in the total recorded lifespan between females that survived to adulthood in all four treatment groups (figure 1c; Tyrone–Ware  $\chi^2 = 0.078$ , d.f. = 3,  $p = 0.994$ ). This is because females fed the poor-quality juvenile diet had prolonged juvenile development and reduced adult lifespan, whereas females fed the good-quality diet had shorter juvenile development and a longer adult lifespan. Overall, this resulted in the same lifespan lived in different ways.

#### (b) Effect of juvenile and sexual maturation diets on somatic condition

The quality of diet during juvenile development had a significant effect on somatic condition (figure 2). Poor-quality diet during juvenile development resulted in a significantly higher body mass at adult eclosion

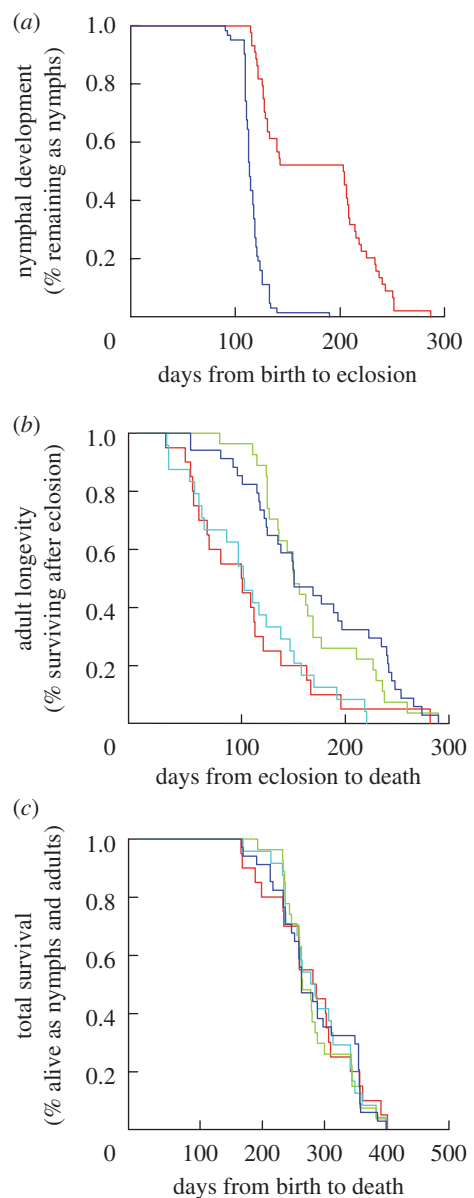


Figure 1. (a) Nymphs that were fed the poor-quality juvenile diets took longer to eclose to adults than females reared on good-quality diets. Red line, poor; blue line, good. (b) Adult lifespan was determined by the juvenile, but not sexual maturation diet; those reared on poor diets had shorter life-spans than those that were reared on good diets. Red line, PP; light blue line, PG; green line, GP; dark blue line, GG. (c) However, there was no overall difference in the lifespan of the females that survived to adulthood, as the decreased adult lifespan was cancelled by the increase in juvenile development.

(figure 2a;  $F_{1,262} = 14.556$ ,  $p < 0.001$ ). The quality of diet during juvenile development had a lesser effect on pronotum length, which approached, but did not reach, statistical significance ( $F_{1,260} = 3.415$ ,  $p = 0.066$ ). Using the residuals of mass at eclosion against pronotum width, nymphs raised on a poor-quality diet during juvenile development eclosed as adults with a greater mass for their size (measured as pronotum length), and thus in better condition, than those raised on a good-quality diet ( $F_{1,207} = 41.261$ ,  $p < 0.001$ ).

Female body mass changed between eclosion and the end of sexual maturation and that change depended on

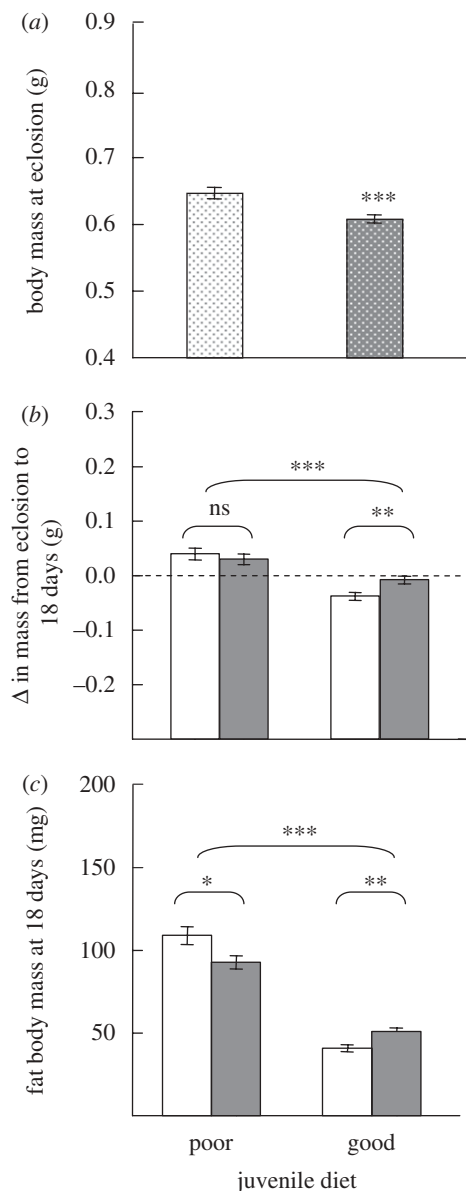


Figure 2. (a) Nymphs that were fed the poor-quality juvenile diet eclosed at a larger mass than those reared on the good-quality diet. (b) Females reared on poor juvenile diets gained mass overall during the sexual maturation period, and those reared on good juvenile diets lost mass. Females from both juvenile diets that switched for the sexual maturation diet lost mass relative to those that did not change diets. (c) This mass change is mirrored by dry fat body mass of females after the sexual maturation period. Significant differences indicated by (\*\*\*) when  $p < 0.001$ , (\*\*) when  $p < 0.01$ , (\*) when  $0.05 > p > 0.01$ , and (ns) when  $p > 0.05$ . Error bars represent  $\pm 1$  s.e. of the mean. White bar, poor adult diet; grey bar, good adult diet.

both diet quality during juvenile development and an interaction between diets during juvenile development and sexual maturation (figure 2b). Adults that had experienced poor-quality juvenile diets as nymphs continued to gain mass during sexual maturation, while those that had experienced good-quality juvenile diets lost mass ( $F_{1,207} = 51.937$ ,  $p < 0.001$ ). We found no direct effects of quality of diet during sexual maturation on mass ( $F_{1,262} = 2.147$ ,  $p = 0.144$ ), but there was a significant interaction between juvenile and sexual maturation diets ( $F_{1,262} = 4.618$ ,  $p = 0.033$ ). Females that had a

good-quality juvenile diet that were switched to a poor juvenile diet lost more mass than females that continued to be fed the good-quality diet ( $F_{1,119} = 8.923$ ,  $p = 0.003$ ).

The quality of diet during juvenile development, but not during sexual maturation, affected fat body stores at sexual maturity (figure 2c). Females that had experienced poor-quality juvenile diets had significantly larger fat bodies than those that had experienced good-quality juvenile diets ( $F_{1,133} = 284.607$ ,  $p < 0.001$ ). There was no effect of diet quality during sexual maturation ( $F_{1,133} = 0.946$ ,  $p = 0.333$ ), but there was a significant interaction between diet quality during juvenile development and sexual maturation ( $F_{1,133} = 16.402$ ,  $p < 0.001$ ). Females from both juvenile diet groups that switched diet for sexual maturation lost fat body mass relative to the females that experienced the same sexual maturation diet as they had been fed as juveniles (figure 2c, Poor juvenile diet,  $F_{1,53} = 6.338$ ,  $p = 0.015$ ; Good juvenile diet,  $F_{1,80} = 10.509$ ,  $p = 0.002$ ).

#### (c) Effect of juvenile and sexual maturation diets on reproductive physiology

The quality of diet during both juvenile development and sexual maturation had a significant effect on ovarian physiology (figure 3). Poor-quality juvenile diet resulted in smaller ovaries (figure 3b;  $F_{1,131} = 25.312$ ,  $p < 0.001$ ). However, good-quality diet during sexual maturation led to an increase in ovarian mass in individuals from both juvenile diet treatments ( $F_{1,131} = 42.652$ ,  $p < 0.001$ ), and the magnitude of the increase depended on the diet experienced as a nymph (juvenile\*sexual maturation  $F_{1,131} = 6.076$ ,  $p = 0.015$ ). The observed increase in ovarian mass in females that had, at some point, experienced the good-quality diet was not related to an increase in ovariole number. Ovariole number was unaffected by the nutritional environment during either juvenile development or sexual maturation (figure 3a; juvenile diet  $F_{1,130} = 1.972$ ,  $p = 0.163$ , sexual maturation diet  $F_{1,130} = 1.058$ ,  $p = 0.306$ , juvenile\*sexual maturation  $F_{1,130} = 0.014$ ,  $p = 0.908$ ), therefore changes in ovarian mass were related to changes in oocyte size, rather than number. Poor-quality juvenile diet resulted in lower levels of ovarian apoptosis (figure 3c;  $F_{1,130} = 11.636$ ,  $p < 0.001$ ). Good-quality diet during sexual maturation did not directly affect levels of ovarian apoptosis ( $F_{1,130} = 3.287$ ,  $p < 0.072$ ), but there was a significant interaction between diet quality during juvenile development and sexual maturation ( $F_{1,130} = 7.012$ ,  $p = 0.009$ ), with females that had experienced poor-quality diet during juvenile development showing an increase in levels of apoptosis with good-quality diet during sexual maturation ( $F_{1,53} = 9.406$ ,  $p = 0.003$ ).

#### (d) Effect of juvenile and sexual maturation diets on mating behaviour and reproductive success

Diet quality during juvenile development, but not sexual maturation, affected female willingness to mate and female choosiness. Diet quality affected overall willingness to mate ( $R \times C$  test of independence  $G_{adj} = 13.276$ , d.f. = 3,  $p = 0.004$ ). After 20 min, 27 per cent (6/22) of the females reared on the poor-quality diet at juvenile



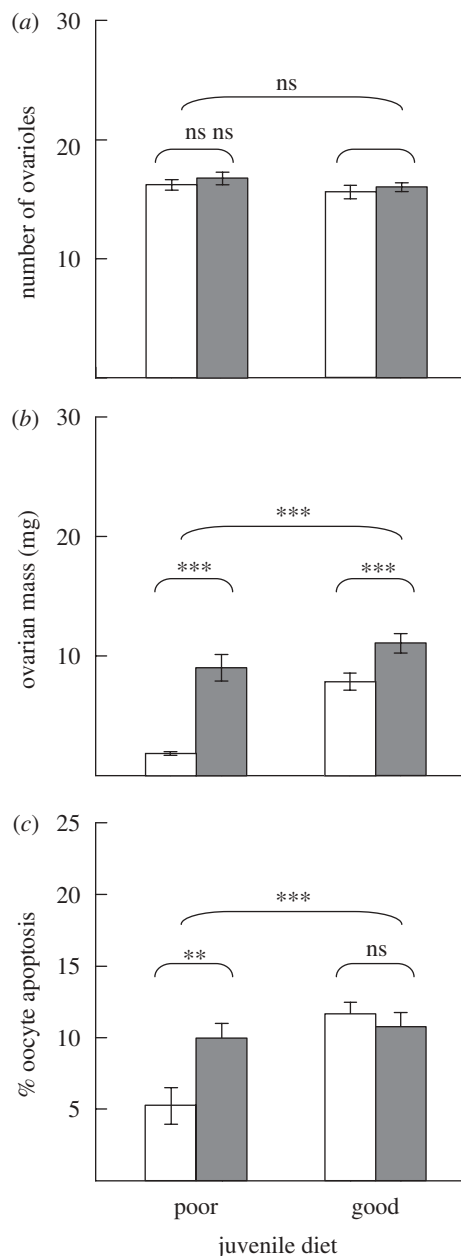


Figure 3. (a) Juvenile diet did not have a significant effect on ovariole number. (b) Females that had, at some point, received the good-quality diet had larger ovaries than those that had not, where ovaries remained immature. (c) Females reared on the good-quality juvenile diet had higher levels of oocyte apoptosis than those that were reared on the poor-quality diet. Significant differences indicated by (\*\*\*) when  $p < 0.001$ , (\*\*) when  $p < 0.01$ , (\*) when  $0.05 > p > 0.01$ , and (ns) when  $p > 0.05$ . Error bars represent  $\pm 1$  s.e. of the mean. White bar, poor adult diet; grey bar, good adult diet.

and sexual maturation stages mated while 57 per cent (14/23) of the females that experienced poor diet during juvenile development and good-quality diet during sexual maturation mated, 62 per cent (22/35) of the females that experienced good diet during juvenile development followed by poor sexual maturation diet quality mated, and 77 per cent (21/27) of females that experienced good-quality diet throughout mated.

Males were equally willing to court females from all treatment groups. There was no effect of female diet quality during juvenile development ( $F_{1,97} = 0.084$ ,  $p = 0.360$ ) or during sexual maturation ( $F_{1,97} = 0.446$ ,

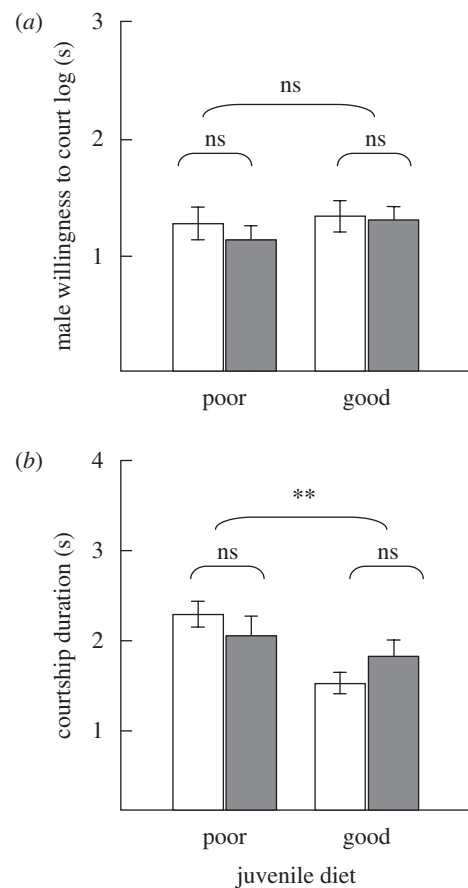


Figure 4. (a) Male courtship effort was not effected female diet regime. (b) Females reared on poor juvenile diets required more courtship from males than those reared on good-quality juvenile diets. (c) Significant differences indicated by (\*\*\*) when  $p < 0.001$ , (\*\*) when  $p < 0.01$ , (\*) when  $0.05 > p > 0.01$ , and (ns) when  $p > 0.05$ . Error bars represent  $\pm 1$  s.e. of the mean. White bar, poor adult diet; grey bar, good adult diet.

$p = 0.506$ ) on the time taken for males to initiate courtship (figure 4a). There was no significant interaction between juvenile and sexual maturation diet quality on male willingness to court ( $F_{1,97} = 0.181$ ,  $p = 0.672$ ). Of the females that responded to male courtship display, there was a significant effect of diet quality on female choosiness. Females that experienced a poor-quality diet during juvenile development required more courtship effort to accept a copulation than females that experienced a good-quality juvenile diet ( $F_{1,49} = 8.190$ ,  $p = 0.006$ ). There was no effect of diet quality during sexual maturation ( $F_{1,49} = 0.035$ ,  $p = 0.852$ ) and no interaction between diet quality during juvenile development and sexual maturation ( $F_{1,49} = 2.385$ ,  $p = 0.129$ ) on willingness to copulate.

Because so few females that had experienced a poor-quality diet during juvenile development mated, we were only able to analyse the effect of juvenile diet quality on the ability of females to produce offspring following mating. Females that experienced a poor-quality diet during juvenile development were not only less willing to mate, they were also less likely to produce offspring following mating ( $R \times C$  test of independence  $G_{\text{adj}} = 9.916$ , d.f. = 1,  $p = 0.002$ ). Of the 20 females that experienced a poor-quality juvenile diet that mated, only 7 produced any offspring. In contrast, 33 out of the 43 females that

experienced a good-quality juvenile diet and mated produced offspring.

#### 4. DISCUSSION

In insects in which metamorphosis results in both a change in morphology and a change in ecological niche, poor juvenile diet is known to result in early reproduction, smaller adult body size and reduced fat stores. We find that the effects of poor juvenile conditions in a species in which juveniles and adults use a common niche results in slow juvenile growth, large fat stores, and no reduction in body size. Moreover, although the sum of juvenile development time and adult lifespan was the same in all the females that survived to adulthood, females reared on poor juvenile diets had protracted juvenile development, followed by a short adult lifespan, regardless of the quality of diet during sexual maturation. Females with a good diet during juvenile development, on the other hand, developed to adult quickly and lived longer as adults. Therefore, our data suggest that the resource allocation pathways in *N. cinerea* are established during juvenile development, and constrain adult plasticity, providing initial evidence that insects that share a common niche throughout their life history may have different life-history priorities from those that change niche. If plasticity to environmental change has an inverse relationship with environmental predictability, this would mean pauro-hemimetabolous insects, like *N. cinerea*, are less able to compensate for a poor start in life.

##### (a) Effect of juvenile and sexual maturation diets on growth and survival

Female lifespan did not differ among treatments, but the demographic composition did depend on juvenile conditions. Females fed the poor-quality juvenile diet had prolonged juvenile development and reduced adult lifespan, and females fed the good-quality diet had rapid juvenile development and a long adult lifespan. One hypothesis is that the extension of the juvenile period under the poor diet is to compensate for the suboptimal nutritional income by extending the period of growth to maintain the adult body size. Large adult body size is commonly associated with increased fecundity (e.g. Roff 1992; Taborsky 2006). However, in *N. cinerea*, there is no evidence that body size is correlated with fecundity (Moore *et al.* 2001, unpublished results) while fecundity may well depend on adult lifespan (Moore *et al.* 2003). The advantage, if any, of this reallocation is unclear. Developmental constraints may mean that body size is canalized, and females must reach a threshold size to mature; *N. cinerea* females are larger than males and take longer to develop (Barrett *et al.* 2009).

##### (b) Effect of juvenile and sexual maturation diets on somatic condition

Traditional life-history theory assumes that juvenile growth is strongly influenced by environmental factors like food and temperature (Roff 1992), and individuals grow at the rate that the environment permits. Our results may fit this constrained phenotype model, as females fed a poorer juvenile diet have slower growth and decreased fitness. Yet, the picture is more complicated because females reared on poor-quality diets had much larger fat

stores than those reared on good diets. This suggests that females reared on poor juvenile diets are of better 'condition' (residual mass for size, Tomkins *et al.* 2004), but have lower fitness, measured as reproduction and longevity.

It is known that in other taxa in which juveniles and adults share a niche that some organisms are able to adjust the optimal rate for growth given the risk of nutritional deprivation in adulthood (Abrams *et al.* 1996; Gotthard 2000). Hence, females that develop in poor juvenile environments have reduced growth rate and store excess energy as insurance against further decrease in nutritional income. Likewise, females reared in good juvenile environments may increase the growth rate, but without the insurance of such great resource capital against starvation (Gotthard *et al.* 1994). The lack of plasticity to a metabolic trajectory dictated by the juvenile environment may be a developmental constraint as plasticity may be costly when predictability is high. Studies in mammals (Bertram & Hanson 2001), including correlations in humans (Hales & Barker 2001; Lindsay & Bennett 2001), show that poor early life nutritional conditions can lead to the adoption of a 'thrifty phenotype' (Hales & Barker 2001), programmed to perform best in poor-resource environments by storing food as fat and maintaining high blood glucose levels. This phenotype can become maladaptive under good conditions (Gluckman *et al.* 2005; Monaghan 2008), and poor juvenile diet followed by excess as adults can lead to a higher susceptibility to metabolic syndromes (Hales & Barker 2001; Ozanne *et al.* 2004). It is known that insects can be susceptible to metabolic syndromes (Schilder & Marden 2006), and as we fed cockroaches a standard diet after mating it is possible that the poor phenotype is 'mismatched' to the adult environment, and the constraint in ability to respond to current conditions may result in reduced fitness.

An alternative hypothesis is that females raised in poor juvenile conditions may be fat but malnourished as a consequence of compensatory feeding. If individuals require certain amounts of one nutritional component for adequate metabolic function, but concentration within the available diet is low, individuals may increase their food intake to obtain adequate proportions of the vital nutrients (Lee *et al.* 2008; Boggs 2009), and excess nutrients may be stored in the fat body (Cochran 1985). Accumulation of excess nutrients, particularly proteins, resulting from hyperphagia may also result in a build-up of toxic products, such ammonia and sodium urate (Cochran 1985), and high-protein diets are known to decrease lifespan in *Drosophila melanogaster* (Lee *et al.* 2008). Hence, hyperphagia may reduce quality in seemingly high-condition individuals owing to 'hidden' malnutrition and toxicity of excess nutrients interfering with normal physiological functioning.

We do not think that hyperphagia fully explains our results. We might predict that hyperphagia stimulated by a limiting nutrient would be reduced once females switch from poor to good-quality diet. Or that hyperphagia would be stimulated in adults that were switched from good- to poor-quality diet. However, females fed a poor-quality juvenile diet continued to invest resources in storage and gained mass. Females fed the good-quality juvenile diet also continued to invest resources in

reproduction and lost mass. Thus, these resource allocation trajectories were not responsive to a change in nutritional income. Ultimately, further work that explicitly tracks nutrients within the cockroach is required to decipher between hyperphagia and thrifty phenotype explanations.

**(c) Effect of juvenile and sexual maturation diets on reproductive physiology and mating behaviour**

We found that the females that were fed the poor diet in both periods did not invest in oocyte maturation, were not receptive to mates and continued to prioritize fat storage. This is perplexing as it is predicted that in animals that mature oocytes as adults, such as in *N. cinerea*, adult nutrients should have primacy in allocation to reproduction regardless of the quality of the diet (Boggs 1997*a,b*). However, it is becoming increasingly clear that trade-offs are complicated both by the quantity and quality of resources available during different life-history stages (Lee *et al.* 2008; Boggs 2009). Our data suggest the adult behavioural phenotype of *N. cinerea* was also dictated by juvenile conditions. When juveniles and adults share a common environment, females that experience poor conditions as juveniles may be unwilling to gamble their capital on a reproductive attempt. Among the females that *were* receptive, those that experienced the poor-quality diet as juveniles were more choosy, perhaps indicating that they would only risk reproduction with a high-quality male. Further work is required to investigate the effect of early life environment on age at sexual maturity, and the behavioural and life-history effects, especially in terms of lifetime reproductive success.

**(d) Conclusion**

Studies of life-history trade-offs across developmental stages are increasingly common. This work must include representatives from multiple taxa with alternative life-history strategies if we are to be able to generalize. Reproductive physiology can act as a mediator between environmental signals across a lifetime, but while some groups of organisms can respond adaptively to changing conditions, others, as shown in this study, may be constrained by past experience. It appears that paurohemimetabolous insects may be more representative of organisms where juveniles and adults share a common environment.

This work was supported by an NERC graduate postgraduate fellowship to ELBB, an NERC grant to JH and AJM and a Leverhulme grant to PJM and AJM. Thanks to Nick Bennett, Tommaso Pizzari and two anonymous reviewers for their constructive comments that greatly improved the manuscript.

**REFERENCES**

Abrams, J. M., Leimar, O., Nylin, S. & Wiklund, C. 1996 The effect of flexible growth rates on optimal sizes and development times in a seasonal environment. *Am. Nat.* **147**, 381–395. (doi:10.1086/285857)

Ali, M., Nicieza, A. G. & Wootton, R. J. 2003 Compensatory growth in fishes: a response to growth depression. *Fish Fish.* **4**, 147–190. (doi:10.1046/j.1467-2979.2003.00120.x)

Barrett, E. L. B., Preziosi, R. G., Moore, A. J. & Moore, P. J. 2008 Effects of mating delay and nutritional signals on resource recycling in a cyclically breeding cockroach. *J. Insect Physiol.* **54**, 25–31. (doi:10.1016/j.jinsphys.2007.07.008)

Barrett, E. L. B., Moore, A. J. & Moore, P. J. 2009 Diet and social conditions during sexual maturation have unpredictable influences on female life history trade-offs. *J. Evol. Biol.* **22**, 571–581. (doi:10.1111/j.1420-9101.2008.01671.x)

Beckerman, A., Benton, T. G., Ranta, E., Kaitala, V. & Lundberg, P. 2002 Population dynamic consequences of delayed life history effects. *Trends Ecol. Evol.* **17**, 263–269. (doi:10.1016/S0169-5347(02)02469-2)

Bertram, C. E. & Hanson, M. A. 2001 Animal models and programming of the metabolic syndrome. *Br. Med. Bull.* **60**, 103–121. (doi:10.1093/bmb/60.1.103)

Birkhead, T. R., Fletcher, F. & Pellatt, E. J. 1999 Nestling diet, secondary sexual traits and fitness in the zebra finch. *Proc. R. Soc. Lond. B* **266**, 385–390. (doi:10.1093/bmb/60.1.103)

Blanckenhorn, W. U. 1998 Adaptive phenotypic plasticity in growth, development, and body size in the yellow dung fly. *Evolution* **52**, 1394–1407. (doi:10.2307/2411309)

Blount, J. D., Metcalfe, N. B., Arnold, K. E., Surai, P. & Monaghan, P. 2006 Effects of neonatal nutrition on adult reproduction in a passerine bird. *Ibis* **148**, 509–514. (doi:10.1111/j.1474-919X.2006.00554.x)

Boersma, M. & Elser, J. J. 2006 Too much of a good thing: on stoichiometrically balanced diets and maximal growth. *Ecology* **87**, 1325–1330. (doi:10.1890/0012-9658(2006)87[1325:TMOAGT]2.0.CO;2)

Boggs, C. L. 1997*a* Dynamics of reproductive allocation from juvenile and adult feeding: radiotracer studies. *Ecology* **78**, 192–202. (doi:10.2307/2265989)

Boggs, C. L. 1997*b* Reproductive allocation from reserves and income in butterfly species with differing adult diets. *Ecology* **78**, 181–191. (doi:10.2307/2265988)

Boggs, C. L. 2009 Understanding insect life histories and senescence through a resource allocation lens. *Funct. Ecol.* **23**, 27–37. (doi:10.1111/j.1365-2435.2008.01527.x)

Boggs, C. L. & Freeman, K. D. 2005 Larval food limitation in butterflies: effects on adult resource allocation and fitness. *Oecologia* **144**, 353–361. (doi:10.1007/s00442-005-0076-6)

Clark, D. C., DeBano, S. J. & Moore, A. J. 1997 The influence of environmental quality on sexual selection in *Nauphoeta cinerea* (Dictyoptera: Blaberidae). *Behav. Ecol.* **8**, 46–55. (doi:10.1093/beheco/8.1.46)

Cochran, D. G. 1985 Nitrogen excretion in cockroaches. *Annu. Rev. Entomol.* **30**, 29–49. (doi:10.1146/annurev.en.30.010185.000333)

Cohen, R. W. 2001 Diet balancing in the cockroach *Rhyarobia maderae*: Does serotonin regulate this behavior? *J. Insect Behav.* **14**, 99–111. (doi:10.1023/A:1007805814388)

Dmitriew, C. & Rowe, L. 2005 Resource limitation, predation risk and compensatory growth in a damselfly. *Oecologia* **142**, 150–154. (doi:10.1007/s00442-004-1712-2)

Dmitriew, C. & Rowe, L. 2006 Effects of early resource limitation and compensatory growth on lifetime fitness in the ladybird beetle (*Harmonia axyridis*). *J. Evol. Biol.* **20**, 1298–1310. (doi:10.1111/j.1420-9101.2007.01349.x)

Gluckman, P., Hanson, M. A., Spencer, H. G. & Bateson, P. 2005 Environmental influences during development and their later consequences for health and disease: implications for the interpretation of empirical studies.

- Proc. R. Soc. B* **272**, 671–677. (doi:10.1098/rspb.2004.3001)
- Gotthard, K. 2000 Increased risk of predation as a cost of high growth rate: an experimental test in a butterfly. *J. Anim. Ecol.* **69**, 896–902. (doi:10.1046/j.1365-2656.2000.00432.x)
- Gotthard, K., Nylin, S. & Wiklund, C. 1994 Adaptive variation in growth rate: life history costs and consequences in the speckled wood butterfly, *Pararge aegeria*. *Oecologia* **99**, 281–289. (doi:10.1007/BF00627740)
- Hales, C. N. & Barker, D. J. P. 2001 The thrifty phenotype hypothesis. *Br. Med. Bull.* **60**, 5–20. (doi:10.1046/j.1365-2656.2000.00432.x)
- Hunt, J., Brooks, R. & Jennions, M. D. 2005 Female mate choice as a condition-dependent life-history trait. *Am. Nat.* **166**, 79–92. (doi:10.1086/430672)
- Jones, S. A. & Raubenheimer, D. 2001 Nutritional regulation in nymphs of the German cockroach, *Blattella germanica*. *J. Insect Physiol.* **47**, 1169–1180. (doi:10.1016/S0022-1910(01)00098-1)
- Kopanic Jr, R. J., Holbrook, G. L., Sevala, V. & Schal, C. 2001 An adaptive benefit of facultative coprophagy in the German cockroach *Blattella germanica*. *Ecol. Entomol.* **26**, 154–162. (doi:10.1046/j.1365-2311.2001.00316.x)
- Lee, K. L., Simpson, S. J., Clissold, F. J., Brooks, R., Ballard, J. W. O., Taylor, P. W., Soran, N. & Raubenheimer, D. 2008 Lifespan and reproduction in *Drosophila*: new insights from nutritional geometry. *Proc. Natl Acad. Sci. USA* **105**, 2498–2503. (doi:10.1073/pnas.0710787105)
- Lindsay, R. S. & Bennett, P. H. 2001 Type 2 diabetes, the thrifty phenotype—an overview. *Br. Med. Bull.* **60**, 21–32. (doi:10.1093/bmb/60.1.21)
- Lindström, J. 1999 Early development and fitness in birds and mammals. *Trends Ecol. Evol.* **14**, 343–348. (doi:10.1016/S0169-5347(99)01639-0)
- Lummaa, V. & Clutton-Brock, T. H. 2002 Early development, survival and reproduction in humans. *Trends Ecol. Evol.* **17**, 141–147. (doi:10.1016/S0169-5347(01)02414-4)
- Metcalf, N. B. & Monaghan, P. 2001 Compensation for a bad start: grow now, pay later? *Trends Ecol. Evol.* **16**, 254–260. (doi:10.1016/S0169-5347(01)02124-3)
- Monaghan, P. 2008 Early growth conditions, phenotypic development and environmental changes. *Phil. Trans. R. Soc. B* **363**, 1635–1645. (doi:10.1098/rstb.2007.0011)
- Moore, A. J. 1990 The inheritance of social dominance, mating behaviour and attractiveness to mates in male *Nauphoeta cinerea*. *Anim. Behav.* **39**, 388–397. (doi:10.1016/S0003-3472(05)80886-3)
- Moore, A. J. & Moore, P. J. 1988 Female strategy during mate choice: threshold assessment. *Evolution* **42**, 387–391. (doi:10.2307/2409241)
- Moore, P. J. & Sharma, S. 2005 A delay in age at first mating results in the loss of future reproductive potential via apoptosis. *Evol. Dev.* **7**, 216–222. (doi:10.1111/j.1525-142X.2005.05024.x)
- Moore, A. J., Gowaty, P. A. & Moore, P. J. 2003 Females avoid manipulative males and live longer. *J. Evol. Biol.* **16**, 523–530. (doi:10.1046/j.1420-9101.2003.00527.x)
- Nylin, S. & Gotthard, K. 1998 Plasticity in life-history traits. *Annu. Rev. Entomol.* **43**, 63–83. (doi:10.1146/annurev.ento.43.1.63)
- Ozanne, S. E., Fernandez-Twinn & Hales, C. N. 2004 Fetal growth and adult diseases. *Semin. Perinatol.* **28**, 81–87. (doi:10.1053/j.semperi.2003.10.015)
- Raubenheimer, D. & Jones, S. A. 2006 Nutritional imbalance in an extreme generalist omnivore: tolerance and recovery through complementary food selection. *Anim. Behav.* **71**, 1253–1262. (doi:10.1016/j.anbehav.2005.07.024)
- Roff, D. A. 1992 *The evolution of life histories: theory and analysis*. New York, NY: Chapman and Hall.
- Schilder, R. J. & Marden, J. H. 2006 Metabolic syndrome and obesity in an insect. *Proc. Natl Acad. Sci. USA* **103**, 18 805–18 809. (doi:10.1073/pnas.0603156103)
- Stoks, R., De Block, M. & McPeck, M. A. 2006 Physiological costs of compensatory growth in a damselfly. *Ecology* **87**, 1566–1574. (doi:10.1890/0012-9658(2006)87[1566:PCOCGI]2.0.CO;2)
- Taborsky, B. 2006 The influence of juvenile and adult environments on life-history trajectories. *Proc. R. Soc. B* **273**, 741–750. (doi:10.1098/rspb.2005.3347)
- Tomkins, J. L., Radwan, J., Kotiaho, J. S. & Tregenza, T. 2004 Genic capture and resolving the lek paradox. *Trends Ecol. Evol.* **19**, 323–324. (doi:10.1016/j.tree.2004.03.029)
- Velasco, L. R. I. & Walter, G. H. 1993 Potential of host switching in *Nezara viridula* (Hemiptera: Pentatomidae) to enhance survival and reproduction. *Environ. Entomol.* **22**, 326–333.
- Zajitschek, F., Hunt, J., Jennions, M. D., Hall, M. D. & Brooks, R. C. 2008 Effects of juvenile and adult diet on ageing and reproductive effort of male and female black field crickets *Teleogryllus commodus*. *Funct. Ecol.* **23**, 602–611. (doi:10.1111/j.1365-2435.2008.01520.x)