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Immediate and delayed life history effects caused by food deprivation early in life in a short-lived lizard

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Abstract

Detailed studies of the mechanisms driving life history effects of food availability are of prime importance to understand the evolution of phenotypic plasticity and the capacity of organisms to produce better adapted phenotypes. Food availability may influence life history trajectories through three nonexclusive mechanisms: (i) immediate and long-lasting effects on individual quality, and indirect delayed effects on (ii) intracohort and (iii) intercohort interactions. Using the common lizard (*Zootoca vivipara*), we tested whether a food deprivation during the two-first months of life influence life history (growth, survival, reproduction) and performance traits (immunocompetence, locomotor performances) until adulthood. We investigated the underlying mechanisms and their possible interactions by manipulating jointly food availability in a birth cohort and in cohorts of older conspecifics. Food deprivation had direct immediate negative effects on growth but positive long-lasting effects on immunocompetence. Food deprivation had also indirect delayed effects on growth, body size, early survival and reproduction mediated by an interaction between its direct effects on individual quality and its delayed effects on the intensity of intercohort social interactions combined with density dependence on body size. These results demonstrate that interactions between direct and socially mediated effects of past environments influence life history evolution in size-structured and stage-structured populations.

Introduction

Food availability early in life is a major factor of life history variation that determines how individuals allocate energy among competing demands for growth, immunity or maturation (Metcalfe & Monaghan, 2001; Taborsky, 2006). In particular, food deprivation during development has both immediate effects on juvenile survival and growth and delayed effects on adult body size, survival and reproductive performances (e.g. Lindström, 1999; Madsen & Shine, 2000; Beckerman *et al.*, 2003). Thus, detailed studies of the mechanisms driving immediate and delayed effects of food availability are of prime importance to understand the evolution of plastic life history strategies and the capacity of organisms to produce better adapted phenotypes (Monaghan, 2008).

Currently, three potential mechanisms of delayed life history effects have been identified. First, food availability can have direct, long-lasting effects on individual quality, including body size, body condition and immunity (Birkhead *et al.*, 1999; Taborsky, 2006). An extreme form of long-lasting effects arises from 'silver spoon effects', when favourable early conditions lead to consistent fitness advantages throughout life (Grafen, 1988). Such silver spoon effects will affect the life history trajectories of entire birth cohorts of individuals and lead to significant cohort effects (Lindström, 1999). For example, Madsen & Shine (2000) demonstrated that a high abundance of rats during the birth year results in cohorts of consistently larger water pythons. Second, food availability may change the number and body size

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of juveniles, and thus alter social interactions within a birth cohort (Beckerman *et al*., 2003). For example, poor nutritional conditions early in life may decrease juvenile survival and, therefore, lead to weaker density dependence at the adult stage (e.g. in a fish, van de Wolfshaar *et al*., 2008). Third, food availability can also influence social interactions between juveniles and older individuals. Competition, cannibalism or inhibition of sexual maturation between cohorts may in turn influence growth and maturation strategies, as demonstrated in fishes (Claessen *et al*., 2000; Szabo, 2002; Aday *et al*., 2006).

Delayed life history effects of food availability experienced early in life are now documented in numerous animal species (e.g. Madsen & Shine, 2000; Beckerman *et al*., 2003; Le Galliard *et al*., 2005). Unfortunately, few studies have investigated the delayed effects of environmental variation on life history trajectories through intracohort and intercohort interactions. Detailed laboratory experiments with soil mites indicate that rearing conditions during early development influence not only individual quality but also density dependence at the adult stage (Beckerman *et al*., 2003). Asymmetric interactions between cohorts are also commonly seen in nature where they often result from size-dependent competition or predation (e.g. in fishes Claessen *et al*., 2000; in lizards Pafilis *et al*., 2009). Yet despite the strong effects of food deprivation early in life on growth trajectories and, therefore, size-dependent behaviours (Monaghan, 2008), the influence of food availability on intercohort interactions remains to be investigated. In fact, little is known about how direct effects of food availability early in life and indirect effects mediated by social interactions combine to shape life history trajectories (Beckerman *et al*., 2003). This is unfortunate because cohort interactions can strongly influence the dynamics of structured populations and the evolution of life history traits (Claessen *et al*., 2000; Claessen & Dieckmann, 2002; De Roos *et al*., 2003).

Here, we report a study of the immediate and delayed effects of food availability on life history variation in the common lizard (*Zootoca vivipara* Jacquin 1787), a short-lived reptile (3–4 years life expectancy) with a plastic life history (Marquis *et al*., 2008). Typical immediate responses to malnutrition in this species include structural growth repression and reduced energy storage in body fat reserves (Avery, 1971; Le Galliard *et al*., 2005). Long-lasting direct effects of food deprivation on survival and reproduction may, therefore, be mediated by silver spoon effects on body size and stored energy (Massot *et al*., 1992; Le Galliard *et al*., 2005; Fig. 1a). However, these direct effects may be buffered if individuals can compensate for a bad start when conditions subsequently improve (Birkhead *et al*., 1999; Metcalfe & Monaghan, 2001). Animals with a continuous growth such as common lizards could in fact maintain the ability to compensate for poor early conditions over their entire life. Nevertheless, such compensatory responses may be associated with ecological and physiological costs (e.g. increase of predation risk or of metabolic rate, Anholt & Werner, 1998; Criscuolo *et al*., 2008) that could reduce survival and/or reproduction (Fig. 1a).

Natural populations of the common lizard are characterized by substantial size variation, asymmetric competition between juveniles and older individuals, and strong density dependence (Massot *et al*., 1992). This implies that indirect effects involving density dependence within a cohort and intercohort interactions may influence life history plasticity to food availability early in life (Fig. 1a). First, high survival early in life in a favourable environment may lead to stronger density dependence and reduced performances at the adult stage. Second, asymmetric competition between cohorts may occur when food availability is limited and the niche overlapping between cohorts is substantial. Food-deprived older conspecifics should, therefore, compete more strongly with juveniles than fully fed older conspecifics if they increase their food intake and/or prey on juveniles (Avery, 1971; Pafilis *et al*., 2009). In addition, large and fast-growing juveniles should be more sensitive to competition with older conspecifics than small and slow-growing juveniles because of a larger niche overlapping (Keren-Rotem *et al*., 2006). Asymmetric competition with older individuals should reduce body growth at the juvenile stage, size at maturation and reproductive outputs and may also reduce survival when social interactions are agonistic (Claessen *et al*., 2000; Pafilis *et al*., 2009). Thus, direct and indirect effects of early food conditions can interact to shape life history trajectories of common lizards (Fig. 1a).

We tested whether life history effects of food availability involved direct effects on individual quality and/or indirect effects by manipulating food availability during approximately 2 months in the laboratory for an entire birth cohort of offspring and for cohorts of older individuals (Fig. 1b). Offspring from each food treatment were released in outdoor enclosures with food-deprived or fully fed cohorts of older lizards in a fully crossed factorial design. Experimental populations were monitored during 2 years and cohort densities were recorded. We compared the life histories of fully fed and food-deprived offspring until adulthood according to their social environment. Several life history traits (growth rates, body size, survival and reproductive performances) and two performance traits (immunocompetence and locomotor performances) were measured in males and females to evaluate the sensitivity of various demographic traits to food availability (Fig. 1b). This experimental design enabled us to examine (i) direct immediate and delayed effects of early food conditions on individual quality, (ii) indirect effects by intercohort interactions and delayed density dependence within a cohort and (iii) interactions between direct and indirect effects.

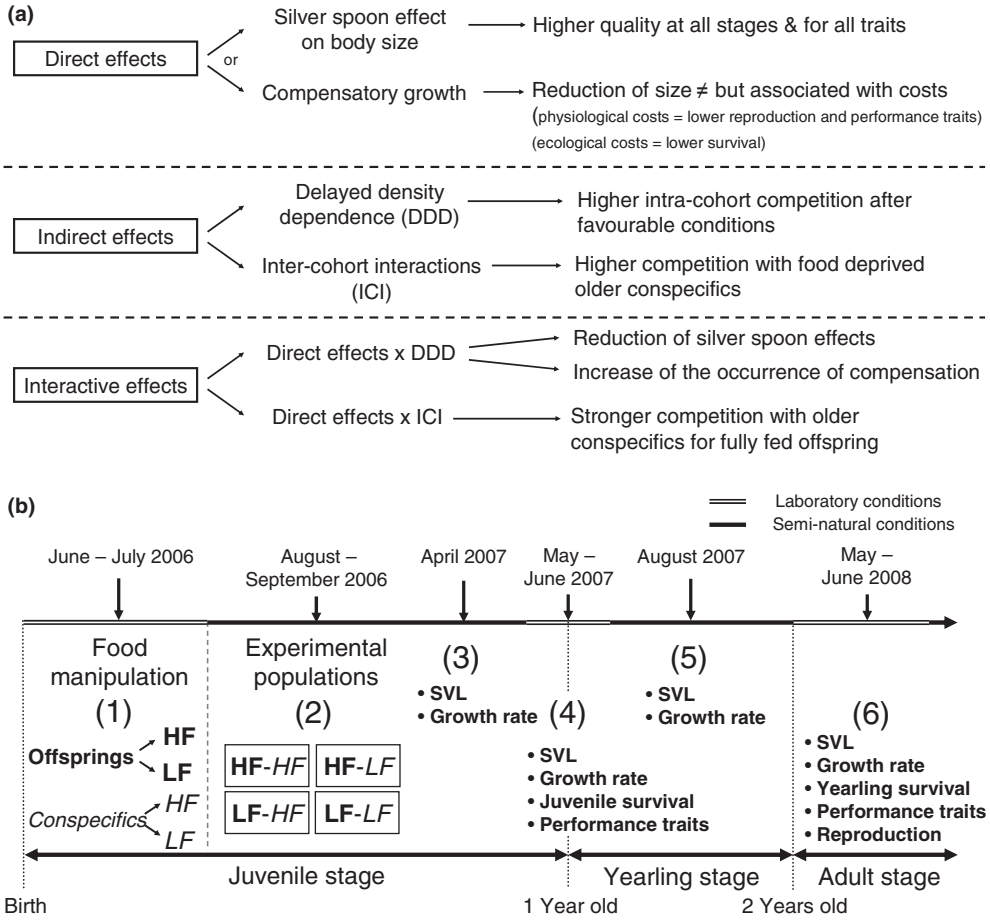

Fig. 1 Predicted effects of food deprivation early in life on life history trajectories in the common lizard (a) and schematic representation of our experimental design (b). (a) Direct life history effects should cause lower performances of food-deprived individuals, possibly buffered by compensatory growth responses throughout life. Such compensatory responses may involve physiological and ecological costs. Indirect effects through delayed-density dependence (DDD) are predicted if fully fed lizards have a higher juvenile survival than food-deprived lizards. Intercohort interactions (ICI) are also predicted if food-deprived conspecifics compete more with juveniles than fully fed conspecifics. Thus, direct effects of early food conditions may interact with delayed density dependence and with intercohort interactions if fully fed offspring are larger and compete more with older conspefics. (b) During step 1, food deprivation was simulated in an entire birth cohort (offspring food treatment) and in cohorts of yearlings and adults (conspecific food treatment). After 50 days of laboratory manipulation (around August–September 2006), juveniles and conspecifics were released in 16 outdoor enclosures according to a fully crossed factorial design (step 2). Life history traits were monitored in the cohort of offspring during four capture sessions carried out in April 2007 (step 3), May–June 2007 (step 4), August 2007 (step 5) and May–June 2008 (step 6). HF, high-food treatment group, LF, low-food treatment group.

Methods

Experimental design

From the 19th of June until the 13th of September 2006, we manipulated the daily food consumption in the laboratory of 460 juvenile lizards for 50 days from their day of birth. Offspring were fed with crickets *Acheta domesticus* obtained from Kreca Inc. (Ermelo, Holland). We used two food treatments, as in our previous studies (Le Galliard *et al.*, 2004, 2005). In the 'high-food' treatment (HF), individuals were fully fed with 100 mg day^{-1} of food, whereas individuals in the 'low food' treatment (LF) were maintained under a food deprivation with a diet of 40 mg day^{-1}. Prey items were weighed prior to feeding and nonconsumed prey was removed each week. Offspring were obtained from 42 gravid females captured in outdoor enclosures (CEREEP, 48°17′N, 2°41′E; n = 209 offspring) and from 58 gravid females captured in two natural populations (Mont Lozère, 44°27′N, 3°44′E; n = 251). However, all lizards were from genetic strains originating from the same geographical area around the Mont Lozère. Immediately after birth, offspring were marked by toe clipping, sexed

by counting ventral scales, measured for snout-vent length (SVL, to the nearest mm) and weighed (to the nearest mg). Clutches of offspring were split into two groups randomly assigned to each food treatment. Offspring food-treatment groups did not differ initially for SVL, body condition (residuals of a linear regression of body mass against body size) or sex ratios (ANOVA, all $P > 0.80$). To identify fathers, we performed microsatellite marker-based paternity analyses following the protocols described by Laloi *et al.* (2004). We used five highly polymorphic microsatellite loci (Lv-3-19, Lv-4-72, Lv-4-alpha, Lv-4-X and Lv-4-115, Boudjemadi *et al.*, 1999) and carried out paternity assignments with a likelihood approach in the Cervus 3.0 software (http://www.fieldgenetics.com/). We could assign fathers to 202 offspring ($n = 21$ fathers and 41 mothers) in litters born from gravid females captured at CEREEP but to no offspring from the natural populations as candidate fathers were not all sampled in natural populations.

At the same time, we also manipulated the daily food consumption of 263 older individuals for 50 days in the laboratory (144 yearlings and 119 adults; adults are ≥ 2 years old and sexually mature individuals, Fig. 1b). We fed lizards with a combination of crickets and moth larvae (*Pyralis farinalis*). Food treatments for yearlings and adults (hereafter named 'conspecific food treatments') were adjusted according to food consumption data from the wild (Avery, 1971). Half of the individuals were fully fed with an average of 200 mg day^{-1} (HF treatment) and the remaining half were maintained under a food deprivation with an average of 100 mg day^{-1} (LF treatment). Animals were captured in outdoor enclosures (CEREEP, $n = 214$) and in two natural populations (Mont Lozère, $n = 49$), and half of the animals of each origin were randomly assigned to each food treatment. The treatment groups did not differ initially for SVL, body condition or sex ratios (ANOVA, all $P > 0.45$) but body condition was significantly higher in the HF treatment after the manipulation ($F_{1,259} = 49.85$, $P < 0.0001$). Food manipulation for adult females started the day after parturition. The number of yearlings and adults starting the experiment each day was adjusted to the number of females to maintain the age and sex structure of each treatment.

At the end of the laboratory manipulation, lizards were measured for body size (SVL) and released into 16 outdoor enclosures each measuring 10 m by 10 m and located in a wet meadow at the CEREEP. We generated populations of lizards by mixing offspring from the same food-treatment group with yearlings and adults from a food-deprived or a fully fed treatment group following a fully crossed factorial design (Fig. 1b). Offspring from the same clutch and food treatment were released into the same enclosure. Four outdoor enclosures were randomly selected for each treatment group, and initial populations had similar age and sex structure (4–5 adult females, three adult males, 4–6 yearling females, 4–5 yearling males, 28–34 juveniles, juvenile sex ratio = 51% of males ± 2 SE). Outdoor enclosures provided lizards with natural food supplies, shelters and basking sites, and were protected by nets to avoid avian predation.

Measurements of life history traits

We monitored populations during four successive capture sessions each lasting 5–20 days to obtain data on growth, body size, survival and female reproductive performances in the cohort of offspring (Fig. 1b). At each recapture, animals were measured for SVL, broken tail (intact vs. recently broken tail, used as an indicator of predation attempts Pafilis *et al.*, 2009) and ectoparasite load (number of ticks Ixodes ricinus around the neck and forelimbs, used to evaluate ectoparasite pressure, Moyer *et al.*, 2002). Growth rates were calculated as the change in SVL divided by the time interval, calculated as the number of days elapsed between the initial measurement and the focal measurement minus the number of days spent in hibernation and emergence (assumed to be from the 1st of October to the 15th of March). Because recapture probabilities are close to one in our experimental system, annual survival probabilities were estimated from recapture data at the juvenile stage (from release in summer 2006 to May 2007) and at the yearling stage (from May 2007 to May 2008). In late spring 2008, gravid females were maintained until parturition in the laboratory to assess their reproductive characteristics. We measured the quantity (total number of eggs) and quality (body size at birth) of the first generation (F1) of offspring born from manipulated females. In late spring 2007, 14 yearling females (8 HF and 6 LF) were obviously gravid upon capture but were not maintained in the laboratory because this would have changed their growth trajectories (Le Galliard, personal observation). We, therefore, could not include reproductive data from 2007 in our analyses.

Measurements of performance traits

We assessed immunocompetence and locomotor performances of manipulated offspring in late spring 2007 and 2008 (Fig. 1b). Immunocompetence was estimated by measuring the delayed inflammatory response to the injection of the mitogen phytohaemagglutinin (PHA), which evaluates acquired T-cell-mediated immunity characterized by a local T-cell proliferation (Tella *et al.*, 2008). This procedure was preferred over alternative methods to measure innate and specific immunity that necessitate substantial blood samples. We injected all individuals ($n = 166$ in 2007 and 90 in 2008) subcutaneously in the right posterior leg with a solution of phosphate-buffered saline (PBS, Sigma, reference D5773) containing 2.5 mg mL^{-1} PHA (Sigma, reference L2880). We inject 0.04 mL in 2007 and 0.08 mL in 2008 to match the size increase in lizards from the yearling to the adult

stage. Just before and 12 h after the injection, we measured the thickness of the right posterior leg to the nearest 0.01 mm with a spessimeter (Mitutoyo ABSOLUTE ID-C1012BS, Japan). The inflammatory response was calculated as the difference in thickness of the leg (mm) between 12 h after and before the injection. A larger value indicates a higher T-cell-mediated immune response (Tella *et al.*, 2008).

We also measured maximal sprint speed and endurance capacity at an optimal body temperature for locomotion (31–33 °C) and in a postabsorption state (after 3 days without food). Sprint speed measurements were made with a 3-metre-long racetrack equipped with 12 pairs of photoreceptor cells, one pair placed every 0.25 m, to register running speed as described by Sorci *et al.* (1997). Maximal sprint speed was calculated from three successive trials separated by a minimum resting time of 30 min. Endurance capacity of the same individuals was measured with a motorized treadmill (Panlab LE 8700, Bioseb, France) as described by Le Galliard *et al.* (2004). Measurements were performed at a running speed of 7–8 ms^{-1} in 2007 and 8–11 ms^{-1} in 2008 to match the size increase in lizards and obtain data at ecologically relevant running speeds (Le Galliard *et al.*, 2004). Pregnancy alters locomotor performances in the common lizard, so we measured the locomotor performances of 43 pregnant females after parturition in 2008.

Statistical analyses

We first analysed immediate effects of food availability on growth rate in SVL and SVL at the end of the experiment (hereafter named 'final SVL') with mixed-effects linear models. Fixed effects included offspring food treatment, sex and their first-order interactions, as well as additive effects of SVL at birth, date of birth and geographical origin of the mother. Clutch identity was included as a random effect influencing both the intercept and the food treatment effect. Next, we used similar models to study the delayed effects of food availability on life history traits (growth rates in SVL, SVL and reproductive traits) and performance traits (immunocompetence, sprint speed and endurance capacity). These mixed-effects linear models also included (i) a conspecific food-treatment effect and its interaction with the offspring food-treatment effect to account for the food manipulation of older conspecifics, (ii) a fixed effect of the number of activity days since release when analysing SVL and (iii) a fixed effect of initial SVL when analysing growth rates to control for decelerating growth curves (Andrews, 1982) and second-order interactions between initial SVL and offspring and conspecific food treatment to test for context-dependent selection on growth rate. Locomotor performances increase with body size in the common lizard (Sorci & Clobert, 1997; Le Galliard *et al.*, 2004). We, therefore, tested separately for the effects of treatments and the effects of body size on maximal sprint speed and endurance capacity. The random part of these models included an effect of enclosure identity, and whenever necessary a clutch identity effect nested in the enclosure effect. Model parameters and test statistics were estimated with a restricted maximum likelihood approach in the *lme* procedure in R 2.9.2 software (http://cran.r-project.org/) as described by Pinheiro & Bates (2000). Minimum adequate models were selected by removing nonsignificant terms in a backward procedure using marginal F tests. Assumptions of normality were fulfilled but Bartlett tests detected variance heterogeneity between treatments and sexes in some analyses. We accounted for heterogeneous variances with a *varIdent* function in *lme* (chapter 5.2 in Pinheiro & Bates, 2000).

We analysed annual survival probabilities at the juvenile and yearling stages with mixed-effects logistic regressions including a logit link and binomial error terms. Models included fixed effects of offspring food treatment, conspecific food treatment, sex and their first-order interactions, as well as additive effects of date of birth and geographical origin and a random enclosure effect. We tested for context-dependent selection on SVL by including in the models second-order interactions between initial SVL centred at its grand mean (linear and quadratic effects), offspring and conspecific food treatment. Model parameters were estimated with a Laplace approximation of the maximum likelihood in *lmer* and fixed effects were tested with Wald Z tests (Bolker *et al.*, 2009). A minimum adequate model was obtained by backward elimination of nonsignificant terms. All estimates are provided with standard errors unless otherwise stated.

Results

Immediate effects

Food deprivation had immediate, negative effects on growth rate in SVL and final SVL (growth rate: $F_{1,357} = 77.77$, $P < 0.0001$; SVL: $F_{1,357} = 77.28$, $P < 0.0001$; Fig. 2a), and variability in both traits was greater in the HF treatment group (growth rate: Bartlett's K-squared = 41.97, d.f. = 1, $P < 0.0001$, variance ratio = 1.59; SVL: Bartlett's K-squared = 42.73, d.f. = 1, $P < 0.0001$, variance ratio = 1.59, Fig. 2a). Sex influenced growth rate and final SVL independently of food treatment (food treatment × sex: all $P > 0.68$; sex: all $P = 0.02$). Growth rate and final SVL also varied significantly between clutches (growth rate: likelihood ratio test (LRT) = 53.85, $P < 0.0001$; SVL: LRT = 55.10, $P < 0.0001$) and, more importantly, growth sensitivity to food availability differed between clutches (growth rate: LRT = 5.95, $P = 0.05$; SVL: LRT = 6.58, $P = 0.04$). This was well illustrated by the crossing familial reaction norms for growth rate (Fig. 2b). We investigated whether this variability could be explained by paternal effects in a subsample of offspring with known paternal lineage

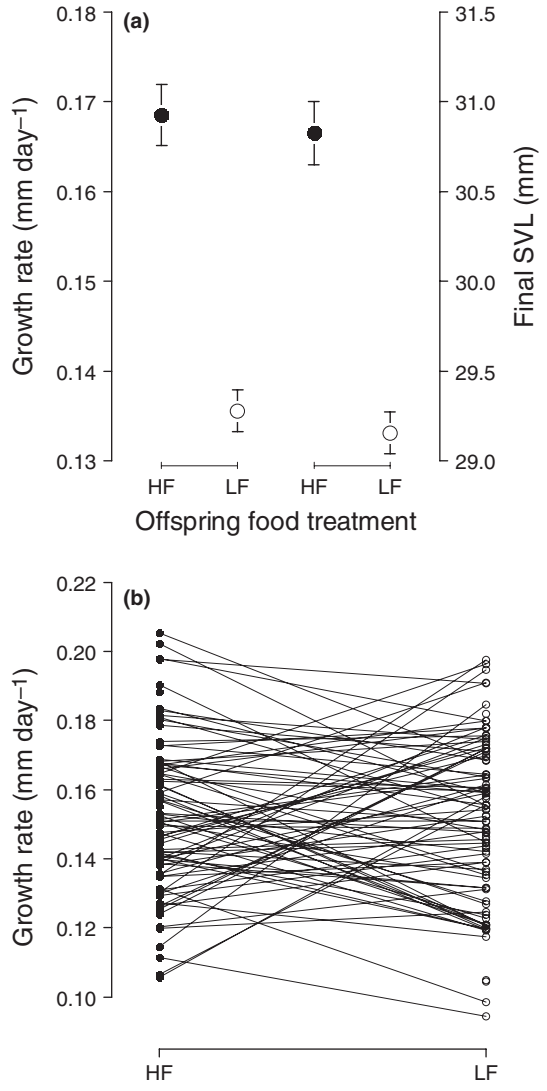

Fig. 2 Immediate effects of offspring food treatment on growth rate and final snout-vent length (SVL). (a) Mean (± SE) growth rate (on the left) and final SVL (on the right) and (b) familial reaction norms for growth rate according to offspring food treatment. (b) Data are given as mean predicted growth rates per family and were obtained from the minimum adequate model selected (see main text).

(n = 202) but this was not the case (LRT, all $P > 0.78$). Furthermore, the growth sensitivity to food treatment of a family was not correlated with mean offspring SVL at birth, birth date or clutch sex ratio (F tests, all $P \geq 0.10$).

Delayed effects on body size

Growth rates from release to adulthood did not differ between offspring food-treatment groups (see the main effect in Table 1). However, growth rate in spring 2007 was affected by a significant interaction between offspring and conspecific food treatment (Table 1) as LF lizards maintained in populations with LF conspecifics grew about 0.1 mm day^{-1} faster than the other lizards (Fig. 3a). Growth rate in summer 2007 was affected by an interaction between conspecific food treatment and SVL in May 2007 (Table 1): growth rate of individuals decreased less importantly with SVL when they were maintained with HF conspecifics (−0.003 ± 0.0007, $F_{1,56}$ = 11.84, P = 0.001) than with LF conspecifics (−0.005 ± 0.0006, $F_{1,70}$ = 55.28, $P < 0.0001$).

Differences in SVL between HF and LF lizards were of the same order of magnitude in April 2007 as after the laboratory manipulation (Fig. 3b) but were only marginally significant because of larger interindividual variation (Table S1 in Supporting Information). From May 2007 onwards, SVL was affected by an interaction between offspring and conspecific food treatment (Table S1). *Post hoc* tests revealed that LF lizards grew to be larger than HF lizards when maintained with LF conspecifics but grew to be smaller when maintained with HF conspecifics (Fig 3b, Table S1). Cohort density did not explain differences in growth rates but partially explained differences in SVL. SVL in May 2008 was negatively correlated with cohort density in August 2007 ($F_{1,14}$ = 10.91, P = 0.005) and treatment effects on SVL were no longer significant when cohort density was taken into account ($F_{1,11}$ = 2.97, P = 0.11). We also found sexual differences in growth rates and SVL after spring 2007 independently of offspring food treatment (Table 1, Table S1).

Delayed effects on survival

Juvenile survival was affected by a significant interaction between offspring and conspecific food treatment (Z = −4.17, $P < 0.0001$). Survival was less than half for HF juveniles as for LF lizards when juveniles were maintained with HF conspecifics (*post hoc* test: Z = 3.61, P = 0.0003) but was slightly longer for HF lizards when juveniles were maintained with LF conspecifics (*post hoc* test: Z = −2.19, P = 0.03, Fig. 4a). Among survivors, the frequency of broken tails was similar between treatment groups in May 2007 (logistic regression: Z = 0.28, P = 0.78) as well as the ectoparasite load (logistic regression: Z = 0.99, P = 0.32). Survival increased with SVL at release up to a plateau, indicating quadratic selection on SVL (SVL = 0.2857 ± 0.054, Z = 4.98, $P < 0.0001$; SVL^2 = −0.0374 ± 0.0141, Z = −2.66, P = 0.008, Fig. 4b).

Yearling survival was similar between treatment groups (offspring × conspecific food treatment: Z = 0.10, P = 0.91; offspring food treatment: Z = −1.23, P = 0.22; Fig. 4c) and was influenced by a marginal interaction between offspring food treatment and a quadratic SVL effect (offspring food treatment × SVL^2: Z = 1.91, P = 0.06). Selection on SVL at the yearling stage was stabilizing around the mean SVL in the HF offspring

Table 1 Analysis of growth rates of lizards (mm day^{-1}) throughout their two-first years of life according to the offspring (offsp.) food treatment, the conspecific (consp.) food treatment, sex, initial snout-vent length (SVL), date of birth and the origin of the mother. Results in bold indicate significant effects. The *F* and *P* values were obtained by backward selection from the full models and likelihood ratio test (LRT) values were obtained for the minimum adequate model selected. Initial SVL refers to the SVL at the beginning of the studied period.

Effect	Release-April 07	April–May 07	May–August 07	August 07–May 08
Fixed effect	$F_{ndf,ddf}$	$F_{ndf,ddf}$	$F_{ndf,ddf}$	$F_{ndf,ddf}$
Offsp. food treatment	$F_{1,13} = 0.05$	$F_{1,12} = 1.96$	$F_{1,13} = 0.12$	$F_{1,14} = 0.22$
Consp. food treatment	$F_{1,14} = 1.25$	$F_{1,12} = 0.44$	$F_{1,14} = 0.53$	$F_{1,13} = 0.02$
Offsp. × consp. food treatment	$F_{1,12} = 0.76$	$F_{1,12} = 10.17^{**}$	$F_{1,12} = 3.05$	$F_{1,12} = 3.61\dagger$
Sex	$F_{1,154} = 0.62$	$F_{1,126} = 70.00^{****}$	$F_{1,128} = 46.25^{****}$	$F_{1,68} = 74.27^{****}$
Offsp. food treatment × sex	$F_{1,151} = 0.00$	$F_{1,119} = 0.63$	$F_{1,124} = 0.08$	$F_{1,63} = 1.21$
Consp. food treatment × sex	$F_{1,153} = 2.66$	$F_{1,120} = 1.33$	$F_{1,123} = 0.06$	$F_{1,62} = 0.18$
Initial SVL (mm)	$F_{1,152} = 1.63$	$F_{1,126} = 26.03^{****}$	$F_{1,128} = 19.49^{****}$	$F_{1,68} = 19.82^{****}$
Offsp. food treatment × SVL	$F_{1,151} = 1.46$	$F_{1,124} = 0.19$	$F_{1,127} = 0.09$	$F_{1,66} = 0.07$
Consp. food treatment × SVL	$F_{1,150} = 0.01$	$F_{1,125} = 1.03$	$F_{1,128} = 5.95^{*}$	$F_{1,67} = 0.58$
Offsp. × consp. food treatment × SVL	$F_{1,149} = 0.10$	$F_{1,123} = 2.68$	$F_{1,126} = 2.88\dagger$	$F_{1,65} = 2.22$
Birth date (days)	$F_{1,155} = 13.83^{***}$	$F_{1,121} = 1.30$	$F_{1,128} = 26.16^{****}$	$F_{1,64} = 1.35$
Origin of the mother	$F_{1,155} = 3.15\dagger$	$F_{1,122} = 1.26$	$F_{1,125} = 0.43$	$F_{1,68} = 6.02^{*}$
Random effect	LRT	LRT	LRT	LRT
Enclosure	16.58^{****}	11.10^{***}	25.00^{****}	12.66^{***}

$\dagger P < 0.10$, $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$, $^{****}P < 0.0001$.

treatment group (SVL = 0.0269 ± 0.0803, $Z = 0.34$, $P = 0.74$; $SVL^2 = -0.0190 \pm 0.0095$, $Z = -2.01$, $P = 0.05$) but no selection on SVL was found in the LF offspring treatment group (SVL: $Z = -0.29$, $P = 0.77$; SVL^2: $Z = 0.22$, $P = 0.83$; Fig. 4d).

Delayed effects on reproductive performances

Fecundity was affected by a significant interaction between offspring and conspecific food treatment ($F_{1,11} = 21.48$, $P = 0.0007$). Females from the HF offspring food-treatment group produced on average three more offspring than LF females when maintained with HF conspecifics, but the reverse was observed when females were maintained with LF conspecifics (Fig. 5a). These treatment effects were importantly explained by differences in female SVL between treatment groups (female SVL: $F_{1,25} = 7.95$, $P = 0.009$; offspring × conspecific food treatment controlling for SVL: $F_{1,11} = 4.35$, $P = 0.06$). In addition, SVL of produced offspring differed significantly between treatment groups after controlling for the trade-off between offspring quantity and quality (offspring × conspecific food treatment: $F_{1,11} = 7.69$, $P = 0.02$; fecundity: $F_{1,14} = 14.89$, $P = 0.002$). For a given fecundity, females from the HF offspring treatment group produced offspring in average of 1–2 mm larger than those produced by LF females when maintained with HF conspecifics, but the reverse was observed when they were maintained with LF conspecifics (Fig. 5b). Again, differences in female SVL between treatment groups explained importantly these treatment effects (offspring × conspecific food treatment: $F_{1,11} = 4.18$, $P = 0.07$; fecundity controlled for female SVL: $F_{1,13} = 15.17$, $P = 0.002$; female SVL: $F_{1,13} = 4.08$, $P = 0.06$). Cohort density measured in May 2008 did not explain variation in reproductive performances (*F* tests, all $P > 0.40$).

We combined data from all demographic traits to estimate the asymptotic growth rate (λ) of an age-structured population parameterized with treatment-specific estimates of each vital rate (see Appendix S1 in Supporting Information). Monte Carlo simulations indicate that λ did not differ from 1 in any treatment group and would not differ between treatment groups if observed differences in fecundity at age 2 are consistent throughout life (Fig. S1A in Supporting Information). However, if differences in fecundity are compensated for after age 2, for example because of growth compensation, the λ of a population of HF offspring maintained with HF conspecifics would be significantly smaller than the λ of other treatment groups (Fig. S1B).

Delayed effects on performances traits

Offspring food treatment had a delayed effect on immunocompetence at the yearling and adult stages (respectively, $F_{1,14} = 5.07$, $P = 0.04$; $F_{1,14} = 4.04$, $P = 0.06$). The inflammatory response was significantly higher in LF than in HF offspring food-treatment groups at the yearling stage (HF = 0.0589 mm ± 0.00657, LF = 0.0805 ± 0.0078) and marginally higher at the adult stage (HF = 0.1076 ± 0.0107, LF = 0.1366 ± 0.0144). Inflammatory response at the yearling stage did not correlate with future survival (logistic regression: $Z = 0.19$, $P = 0.85$), growth (linear regression: $F_{1,63} = 2.85$, $P = 0.10$) or fecundity (linear regression: $F_{1,23} = 0.01$, $P = 0.91$) and with current ectoparasite load (Poisson regression: $Z = 0.40$, $P = 0.69$).

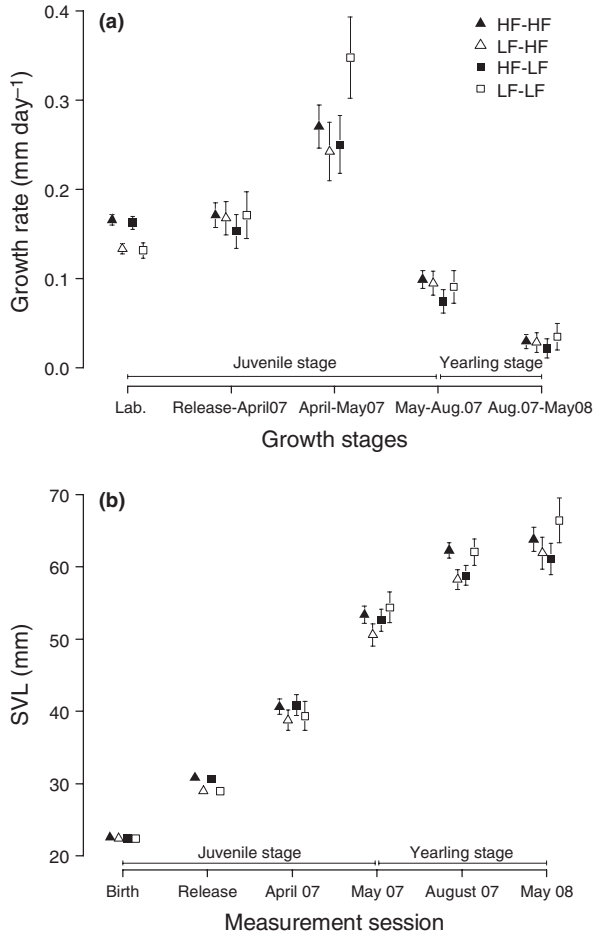

Fig. 3 Growth rates (a) and snout-vent length (SVL) (b) in each treatment group. (a) Growth rates in the laboratory (Lab.: $n = 460$), from release in summer 2006 to April 2007 (Release-April 07: $n = 173$), from April to May 2007 (April–May 07: $n = 144$), from May to August 2007 (May–August 07: $n = 148$) and from August 2007 to May 2008 (August 07–May 08: $n = 87$). (b) SVL at birth, at release, in April, May ($n = 167$) and August 2007 ($n = 152$) and in May 2008 ($n = 90$). (a, b) Estimates were obtained from linear mixed effects models with offspring and conspecific food treatment and their interaction as fixed factors. Black symbols: HF offspring food treatment; white symbols: LF offspring food treatment; triangles: HF conspecific food treatment; squares: LF conspecific food treatment.

Locomotor performances at the yearling and adult stages were not affected by offspring food treatment (all $P > 0.12$) and only maximal sprint speed at the yearling stage increased with body size ($F_{1,44} = 10.42$, $P = 0.0015$, other tests: $P > 0.45$). Males ran faster and were more enduring than females (all $P < 0.02$). Performance traits in 2007 and 2008 were positively correlated (Pearson's product correlation for immunocompetence: $r = 0.20$ [−0.01, 0.41] 95% CI, $P = 0.07$; maximal sprint speed: $r = 0.34$ [0.13, 0.51] 95% CI, $P = 0.002$; endurance capacity: $r = 0.38$ [0.19, 0.55] 95% CI, $P = 0.0003$).

Discussion

Immediate effects of food availability

Spatial and temporal variations in food availability are common in nature and play a major role in life history variation (Metcalfe & Monaghan, 2001). Here, we found that growth and body size during the first weeks of life responded quickly to food deprivation as in our previous study (Le Galliard *et al.*, 2005). However, offspring raised under favourable conditions displayed more residual variation than lizards raised under food deprivation for growth rate and body size in the laboratory but not in the outdoor enclosures. This illustrates a clear example of context-dependent expression of phenotypic variation. Hoffmann & Merilä (1999) explained this reduction in phenotypic variation under unfavourable conditions by a limitation of the genetic potential of organisms – a hypothesis validated for morphometric traits by the meta-analysis of Charmantier & Garant (2005).

In addition, contrary to a previous study involving a smaller number of families (Le Galliard *et al.*, 2005), we found that growth rate sensitivity to food availability varied significantly between families. This variation was substantial as growth did not improve, or even responded negatively to full feeding in some families (see Fig. 2b). Maternal (reviewed in Mousseau & Fox, 1998) and paternal effects (Galloway, 2001) may contribute to changes in the ranking of families across environments. However, such parental effects are unlikely here because no paternal effects on phenotypic plasticity were found and none of the maternal effects on phenotypic plasticity tested were significant. Low genetic correlations across environments have already been demonstrated in the common lizard for survival and growth plasticity along altitudinal and humidity gradients (Sorci *et al.*, 1996; Lorenzon *et al.*, 2001) and are common in the wild (reviewed in Charmantier & Garant, 2005), Altogether, context-dependent expression of phenotypic variation and genotype–environment interactions may contribute to the maintenance of phenotypic plasticity in resource allocation strategies in this species (Hoffmann & Merilä, 1999; Charmantier & Garant, 2005).

Long-lasting effects of food availability on life history traits

We hypothesized that food deprivation early in life should have long-lasting effects on body size and would, therefore, affect size-dependent life history traits such as growth, juvenile survival and reproductive performances (Fig. 1a). If individuals were able to compensate, we expected them to face physiological costs through trade-offs between competing functions as well as ecological costs that may reduce their survival, performances traits or reproduction (Metcalfe & Monaghan, 2001; Fig. 1a).

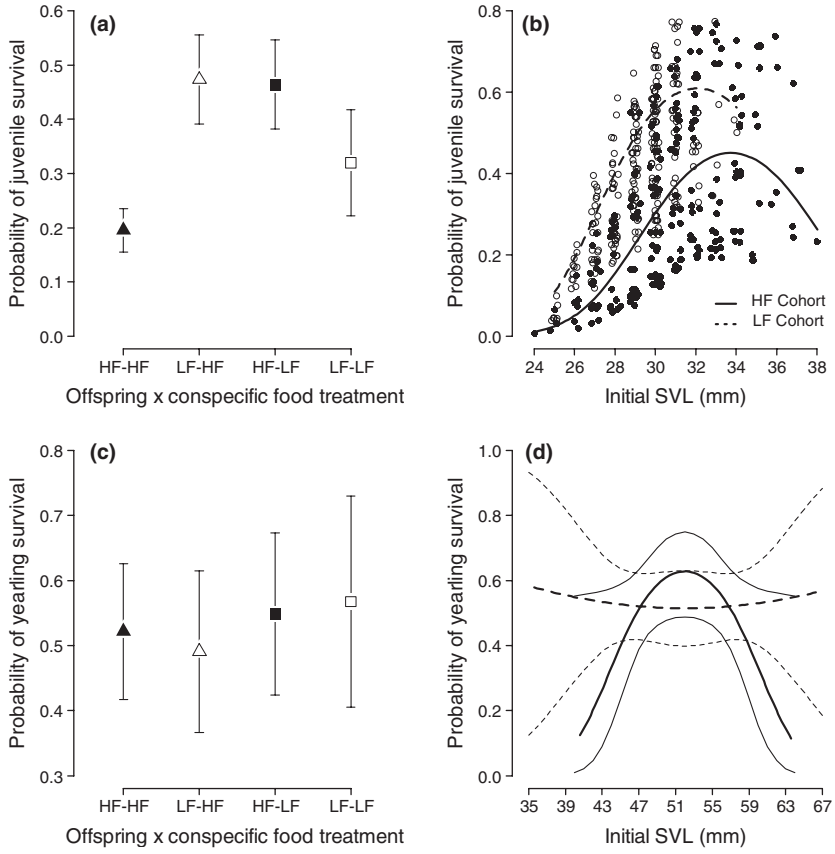

Fig. 4 Annual survival probabilities for treatment groups at the juvenile stage (a) and yearling stage (c), as well as effects of initial snout-vent length on juvenile survival (b, size at release) and yearling survival (d, size in May 2007). (a) The annual survival probability of fully fed juveniles maintained with fully fed older conspecifics was lower than the average annual juvenile survival probability observed in our experimental system from 1999 to 2003 (0.33 [0.23, 0.45] 95% CI, n = 1743 juveniles). (a, c) Estimates were obtained from generalized mixed effects models with offspring and conspecific food treatment and their interaction as fixed factors. (b) Fitted values and predicted curves were obtained from the minimum adequate model (see main text) including an interaction between the quadratic body size effects and offspring food treatment. (d) Predicted curves (and 95% CI) were drawn from the estimates of the minimum adequate model (see main text). Symbols as in Fig. 3.

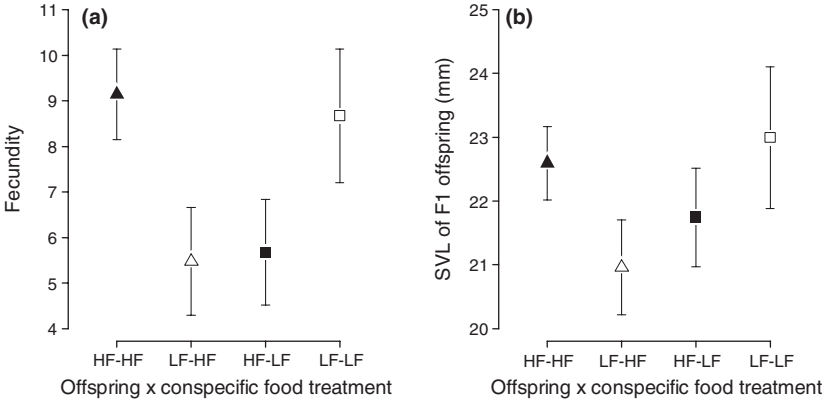

Fig. 5 Female reproductive performances in 2008. (a) Fecundity (n = 43) and (b) snout-vent length of the offspring (n = 212) produced in each treatment group. Estimates were obtained from the minimum adequate models (see main text) and (b) were calculated for a mean fecundity.

We also predicted that these direct effects of food availability could be modulated by indirect effects caused by changes in the intensity of competition within and between cohorts (van de Wolfshaar *et al.*, 2008; Pafilis *et al.*, 2009; Claessen *et al.*, 2000; Fig 1a). Our findings partially support these qualitative predictions.

When food-deprived offspring were maintained with food-deprived conspecifics, they overcompensated for

their bad growth start during the juvenile stage and thus reached a larger body size at adulthood, which enabled them to produce more offspring than fully fed lizards. This growth overcompensation was associated with a juvenile survival that was slightly lower than that for fully fed lizards. In sharp contrast, fully fed lizards released with fully fed yearlings and adults maintained their body size advantage over food-deprived offspring at the juvenile stage and thus reached a larger size at the yearling stage. As expected, the reproductive performances of adult females were highly dependent on their body size (Massot *et al.*, 1992). However, a silver spoon effect existed for the reproductive performances of fully fed females maintained with fully fed conspecifics and these females produced more and larger offspring than food-deprived females despite their similar body size (Taborsky, 2006). At the same time, juvenile survival of fully fed individuals maintained with fully fed conspecifics was less than half that of food-deprived individuals. Thus, life history trajectories were shaped by an interaction between the direct effects of food availability early in life on individual quality and some indirect effects on the intensity of intercohort interactions. However, contrary to our qualitative predictions, fully fed older conspecifics inhibited compensatory responses in juveniles (Fig. 1a). These results suggest that fully fed yearlings and adults with a higher condition (unpublished data) were greater competitors for juveniles than conspecifics that experienced a prolonged food deprivation (Keren-Rotem *et al.*, 2006). In turn, competition with fully fed conspecifics may have selected for higher investment in body growth according to body size in juveniles and could explain why growth rates decelerated more weakly with body size for juveniles maintained with fully fed conspecifics.

Compensatory growth responses to poor early environmental conditions have been demonstrated in several species, where it may be an adaptive mechanism to reduce the fitness costs associated with poor early development (Birkhead *et al.*, 1999; Metcalfe & Monaghan, 2001). Yet the ability to compensate for a bad start varies between and within species (Metcalfe & Monaghan, 2001; Tschirren *et al.*, 2009). In the common lizard, this ability depends on the population structure. Our previous studies have shown that strong competition within a cohort can prevent individuals from compensating for growth restriction at the juvenile and at the yearling stage (Massot *et al.*, 1992; Le Galliard *et al.*, 2005). Here, we demonstrated that food-deprived lizards displayed compensatory growth only when they were maintained with food-deprived older conspecifics. Thus, the ability to compensate can be modulated by intercohort interactions in size-structured and stage-structured populations. Furthermore, despite their fitness advantages, compensatory growth strategies are often associated with short-term costs, such as an increased metabolic rate (Criscuolo *et al.*, 2008) or risks of predation and parasitism (Anholt & Werner, 1998; Crouch & Lubin, 2000), and/or with long-term physiological costs, such as cellular damages (reviewed in Metcalfe & Monaghan, 2001). Here, growth overcompensation for a bad start that led to a higher maturation body size and fecundity was also associated with the immediate cost of reduced juvenile survival. This lower survival was not apparently because of higher predation and parasitic risks as assessed from the proportion of broken tails (Pafilis *et al.*, 2009), ectoparasite load (Moyer *et al.*, 2002) and immune performances (see below). However, these variables were measured only on surviving individuals and may, therefore, not be reliable indicators of causes of mortality at the individual level.

Silver spoon effects from being born under favourable conditions are assumed to be associated with increased lifetime reproductive success (Grafen, 1988; Monaghan, 2008), but our analysis led to a different picture. Here, fully fed lizards benefited from a silver spoon effect on body size and female reproduction when they were maintained with fully fed yearlings and adults. However, these positive effects of early food conditions were associated with a strong negative effect on juvenile survival. As fully fed juveniles did not display higher growth rates than food-deprived lizards, their low juvenile survival cannot be explained by ecological or physiological costs associated with fast growth (Anholt & Werner, 1998; Criscuolo *et al.*, 2008). Alternatively, we predicted that a low juvenile survival could be because of viability selection against larger individuals (Fig. 1a). Indeed, juvenile survival depended on body size and the effect of body size was nonlinear, indicating that some fitness costs counter-balanced the survival benefits of being larger early in life (Fig. 4b). Yet we failed to detect differential natural selection on body size between treatments (offspring × conspecific food treatment × initial SVL (linear and quadratic): all $P < 0.10$). Thus, the low juvenile survival of fully fed offspring cannot be explained by selection against larger individuals. Moreover, we did not find evidence of more aggression or predation attempts as assessed from the proportion of broken tails, and fully fed juveniles maintained with fully fed yearlings and adults did not suffer from a higher parasitic exposure. Thus, fully fed yearlings and adults imposed some undetected mortality factors on fully fed juveniles, which may be driven by behavioural interactions (Webster, 2004).

In summary, life history consequences of food availability were caused by statistical interactions between direct effects on individual quality and indirect effects on intercohort interactions. In addition, intracohort density dependence varied between experimental populations as a consequence of differences in juvenile survival and contributed to maintain body size differences between populations but did not influence yearling survival and reproductive performances. These results suggest that the dynamical effects of fluctuations in food supply should depend on cohort interactions, which could generate

cohort cycles and complex population dynamics (De Roos *et al.*, 2003). Also our analysis of lifetime reproductive success suggests that the fitness benefits and costs of fast growth under favourable conditions early in life depend significantly on cohort interactions. The general picture was one of a lower lifetime reproductive success for fully fed juveniles maintained with fully fed older conspecifics. Thus, cohort interactions may constrain the evolution of rapid growth rates early in life and should be considered in future studies of growth strategies in this and other species with structured populations.

Long-lasting effects of food availability on performance traits

Several laboratory studies investigated long-lasting effects of food deprivation on performance traits and hypothesized that these traits are surrogates of demographic performances in the wild (e.g. Birkhead *et al.*, 1999; Hoi-Leitner *et al.*, 2001; Le Galliard *et al.*, 2004). However, our results show that effects of food deprivation on the performance traits investigated here (locomotor performances and immunocompetence) were independent of the effects on demographic traits. Performance traits were insensitive to social interactions between cohorts and to density-dependent interactions within a cohort. Also locomotor performances were not influenced by food availability experienced early in life and a positive allometric relationship between locomotor performances and body size was significant only for maximal sprint speed at the yearling stage. Thus, factors not tested here, for example maternal genetic effects, the quality and the quantity of muscle or the length of the limbs, were more important determinants of locomotor capacities than body size and food availability early in life (Vanhooydonck *et al.*, 2001; Le Galliard *et al.*, 2004).

In addition, our results clearly indicate long-lasting effects of food deprivation on immunity, measured as the delayed inflammatory response to PHA injection. Other studies have demonstrated that food deprivation during early development has immediate immunosuppressive effects (e.g. Birkhead *et al.*, 1999; Hoi-Leitner *et al.*, 2001). However, individuals may be able to compensate or even overcompensate for these immunosuppressive effects (e.g. in the zebra finch, Birkhead *et al.*, 1999; Tschirren *et al.*, 2009). Here, offspring raised under food deprivation displayed higher inflammatory responses later in life than fully fed offspring but we cannot affirm that they overcompensated as we did not measure immediate effects of food deprivation on immunocompetence. The maintenance of a strong immunity requires substantial energy expenditure and thus should trade-off with growth, fecundity and/or survival (reviewed in Lochmiller & Deerenberg, 2000). This cost of overcompensation was not detected in our experiment until adulthood; however, physiological costs may appear later in life and affect senescence patterns (Lochmiller & Deerenberg, 2000). Alternatively, the maintenance of a strong immunity may be an adaptive response to compensate for the survival costs associated with a reduced early growth as stronger immune responses allows individuals to fight parasites more efficiently (Tella *et al.*, 2008; Cote *et al.*, in press). Yet we found no evidence of direct survival advantages to higher immunocompetence in this study.

Conclusion

Our study demonstrates complex cohort effects of food availability early in life mediated by direct effects on growth, long-lasting effects on immune performances, and an interaction between direct effects on body size and indirect effects on growth, survival and reproduction. The effects of food deprivation on performance traits were clearly independent of those on demographic traits, suggesting that findings concerning performance traits in laboratory studies should be interpreted with caution. In accordance with a previous study (Le Galliard *et al.*, 2005), life history effects did not differ between sexes despite the existence of a sexual dimorphism in growth and body size. In addition, the early environment influenced population growth through variation in juvenile survival mediated by social interactions between cohorts. Interactions between direct and indirect effects of environmental variation may play a major role in the evolution of life history traits within size-structured and stage-structured populations characterized by strong asymmetric competition for food and space between cohorts as in many species of fish (Claessen *et al.*, 2000; Szabo, 2002; Webster, 2004).

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Supporting information

Additional Supporting Information may be found in the online version of this article:
Table S1 Analysis of SVL (mm) of male and female lizards throughout their two-first years of life according to the offspring (offsp.) food treatment, the conspecific (consp.) food treatment, the age (number of activity days since day of birth) and the origin of the mother.
Appendix S1 Methods: projection matrix model.
Figure S1 Predicted asymptomatic growth rate (λ) in each treatment group.

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