doi: 10.1111/j.1420-9101.2006.01094.x

Ontogenic sources of variation in sexual size dimorphism in a viviparous lizard

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Keywords:

body size;
Lacerta vivipara;
maternal effects;
plasticity;
sexual dimorphism;
viviparity.

Abstract

To elucidate the developmental aspects of the evolution of sexual size dimorphism (SSD), an understanding of the sex-specific ontogeny of body size is critical. Here, we evaluate the relative importance of genetic and environmental determinants of SSD in juvenile common lizards (*Lacerta vivipara*). We examined the prenatal and post-natal effects of population density and habitat humidity on SSD, as well as the maternal effects of food availability, corticosterone level, humidity and heat regime during gestation. Analyses indicated strong prenatal and post-natal plasticity in body size *per se* and yielded three main results with respect to SSD. First, SSD in juvenile common lizards matches qualitatively the SSD observed in adults. Secondly, SSD was influenced by none of the prenatal factors investigated here, suggesting poor sex-biased maternal effects on offspring size. Thirdly, SSD was sensitive to post-natal habitat humidity, which positively affected growth rate more strongly in females than in males. Thus, natural variation in SSD in juvenile common lizards appears to be primarily determined by a combination of sex-biased genetic factors and post-natal conditions. We discuss the possibility that viviparity may constrain the evolution of sex-biased maternal effects on offspring size.

Introduction

Sexual size dimorphism (SSD) is common in adult animals and the degree of SSD can vary greatly among taxa and populations (Fairbairn, 1997; Blanckenhorn, 2000). Studies that try to elucidate the ultimate causation of SSD in adults are numerous (Andersson, 1994) and suggest an adaptive divergence in body size between sexes (e.g. Price, 1984; Preziosi & Fairbairn, 2000; Bouteiller-Reuter & Perrin, 2005). Three main selective pressures driving the evolution of SSD have been recognized: (i) natural selection for niche divergence between sexes, (ii) sexual selection that favours larger adult body size in one sex and (iii) fecundity selection that favours larger adult females (Shine, 1989; Fairbairn, 1990). Such sex-specific selection on adult body size leads to ontogenic divergence between the sexes despite the common gene pool shared by males and females (e.g. Kozlowski, 1989; Badyaev, 2002).

Sexual size dimorphism in adults results from fundamental differences in physiology, behaviour and ecology between the sexes during growth (e.g. Stamps, 1993; Stamps & Krishnan, 1997; Haenel & John-Alder, 2002). An understanding of the sex-specific ontogeny of body size is thus critical to elucidate the evolution of SSD. First, sex-specific development processes may be the proximate targets of sex-specific selection on adult body size (Reeve & Fairbairn, 1996). Secondly, similar patterns of SSD among adults of different species may be produced by different ontogenic processes and therefore may not be homologous (Stamps, 1993). For example, a larger adult body size in one sex can result from a larger size at birth, a longer growth period and/or a faster growth rate

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(Kozlowski, 1989; Shine, 1990; Stamps & Krishnan, 1997). Thus, a species-specific examination of the ontogeny of SSD may be necessary (Stamps, 1993; Waltkins, 1996). Finally, mechanisms that control divergent growth in males and females may influence the evolutionary trajectories of SSD. For example, the strong genetic correlation for body size between sexes observed in several species implies a slow genetic response of SSD to selection and may thus constrain the evolution of SSD (Lande, 1980; Merilä *et al.*, 1998). However, adaptive adjustments in SSD to changing environments may be facilitated by phenotypic plasticity (Cooch *et al.*, 1996; Post *et al.*, 1999), as exemplified by the house finch (*Carpodacus mexicanus*). In this species, maternal control of offspring SSD allows the development of a sex-specific body size based on the different selection pressures of the habitat (Badyaev & Martin, 2000) so that reproductive success of each sex can be optimized (Badyaev *et al.*, 2003).

A recent review has summarized the proximate studies of SSD in vertebrates (Badyaev, 2002). This review indicates that ontogenic divergence in body size between the sexes can be produced by a combination of sex-specific genetic and environmental determinants that act upon ontogenic factors such as size at hatching, growth rate and age at maturation (Shine, 1990; Stamps, 1993; Waltkins, 1996). With respect to genetic determinants, SSD may be caused by sex-linked genes involved either directly in development or in the modulation of autosomal developmental genes (Rhen, 2000). For example, differences in gonadal steroid levels between sexes may differentially affect plasma growth hormone, which indirectly stimulates protein synthesis, bone formation, and cell growth and proliferation (Gatford *et al.*, 1998). SSD may also result from sex-specific environmental effects (e.g. LeBlanc *et al.*, 2001), including those experienced during the prenatal period (e.g. Müller *et al.*, 2002). Yet, studies quantifying the genetic, maternal and environmental determinants of variation in SSD remain exceedingly rare.

The paucity of data on proximate causes of variation in SSD is rooted in the difficulties to reliably differentiate between effects of genetic and prenatal or post-natal environmental determinants of SSD. Estimates of genetic variation in SSD can be especially confused by prenatal environmental effects, emphasizing the need for carefully controlled experiments that distinguish between such sources of variation (Clark & Galef, 1995). The several studies that have addressed environmental mediation of SSD found significant effects of season, population density, food supply and parasitism (reviewed by Badyaev, 2002). However, these studies most often rely on a correlative approach, and thus cannot definitively identify environmental factors responsible for variation in SSD or distinguish between prenatal and post-natal influences of the environment. Experimental studies could address both of these issues. For example, manipulations of environmental conditions before parturition could be combined with reciprocal transplants of offspring to distinguish between prenatal and post-natal environmental effects. Experimental studies in vertebrates have already determined several prenatal and post-natal factors important for body size determination *per se* (e.g. in lizards, see Ferguson & Talent, 1993; Niewiarowski & Roosenburg, 1993; Sears & Anguiletta, 2003), but, to our knowledge, only a few of them have tested for effects on SSD (Haenel & John-Alder, 2002). Likewise, although many studies show that maternal effects can affect offspring body size, the extent to which maternal effects may produce sex-biased size differences remains largely unknown (Cordero *et al.*, 2001; Badyaev *et al.*, 2003).

Here, we attempt to evaluate the relative importance of genetic and environmental determinants of SSD in juvenile common lizards (*L. vivipara*). A significant sexual dimorphism in our population of interest has already been documented, with adult females being larger than males by ca. 10% (Massot *et al.*, 1992). Furthermore, this lizard species shows substantial phenotypic plasticity in body size (Sorci *et al.*, 1996), and critical environmental factors that affect body size (population density, humidity, food availability and temperature) have been identified (Massot *et al.*, 1992; Lorenzon *et al.*, 2001; Le Galliard *et al.*, 2005). Studies conducted in different animal species indicate that body size can be influenced by a web of interacting environmental factors, most notably food availability and temperature (Badyaev, 2002). Thus, to account for the possibility that effects on SSD may be produced in a complex way, we addressed the combined effects of several potential prenatal and post-natal factors towards variation in SSD. However, because a previous laboratory experiment has shown that SSD in juvenile common lizards is not affected by post-natal food availability (Le Galliard *et al.*, 2005), we do not further address the effects of this variable here.

We used two different approaches to examine genetic and environmental mediation of SSD in the common lizard. First, we considered variation in SSD among families, study sites and study years to allow for a direct comparison of our results with those found in other vertebrate species examined for sex-specific familial, spatial and yearly variation in body size (Badyaev, 2002). Secondly, we tested for sex-specific sensitivity of body size to population density and habitat humidity, both prenatally and post-natally. We also examined for possible maternal effects by manipulating food availability and corticosterone level of gravid females in the laboratory as well as humidity and heat conditions during gestation. All data used in our analyses originated from a long-term study not originally planned for the investigation of ontogenic sources of SSD. Thus, we were limited in addressing only a predetermined set of factors. However, previous investigations indicate that the

environmental variables examined here are relevant for determination of body size *per se* (Massot *et al.*, 1992; Lorenzon *et al.*, 2001; Meylan & Clobert, 2004) and, thus, may also be relevant in determining variation in SSD. Although we did not directly examine effects of post-natal temperature on offspring SSD, effects of maternal temperature conditions were tested, and post-natal temperature effects were indirectly addressed through investigations of spatial and temporal variability in SSD (Clobert *et al.*, 1994). Therefore, we are confident that our study does not exclude the potential of post-natal temperature effects on SSD. We predict that if SSD is plastic, some of the prenatal and post-natal factors investigated here will affect body size differences between males and females. In contrast, if SSD results mainly from sex-biased genetic factors, we expect no changes in SSD as a consequence of our environmental manipulations.

Materials and methods

Species

The common lizard *L. vivipara* (Jacquin 1787) is a small [50–70 mm adult snout-vent length (SVL)] live-bearing species that inhabits peat bogs and heathlands across Eurasia. We investigated this species in 11 different study sites (see Fig. 1) located in a high-elevation area (ca. 1500 m a.s.l.) in the Cévennes, southern France (44°30′N, 3°45′E), where hibernation lasts from October until April and mating starts in mid-May (see Clobert *et al.*, 1994 for a description of the study area). After 2 months of gestation, parturition occurs over a 3-week period in mid-July. Embryos develop within yolked eggs retained in the oviduct, and a primitive placenta allows nutrient and gas exchange between the mother and the eggs, as well as between the eggs themselves (Panigel, 1956). On average, five eggs are laid (range: 1–13) and offspring hatch out of the thin and transparent shell within 24 h. Upon hatching, offspring require no parental care.

We used SVL as an estimator of body size. SVL has a clear functional significance in females because it relates to the volume of the abdominal cavity, which in turn sets the upper limit on clutch size (Avery, 1975). SVL may also be important for a male's ability to compete for mates (Andersson, 1994); it is a good predictor of male mating success in this species (P. S. Fitze and J.-F. Le Galliard, unpublished data). SVL is also positively correlated with juvenile survival in both sexes (Le Galliard *et al.*, 2004). Furthermore, SVL is a reliable estimate of body size because it is not confounded by tail loss or daily food intake, as is the case for total body length and body mass respectively (Massot *et al.*, 1992).

In all of the experiments outlined below, gravid females were captured on average 1 month before giving birth and were maintained under standardized conditions in the laboratory. Females were housed in individual terraria (25 × 15 × 15 cm), and terraria were checked daily for freshly laid clutches at 9:00 and 14:00 hours (Lorenzon *et al.*, 1999). Immediately after hatching, offspring were measured for SVL, individually marked by toe clipping, and sexed according to their ventral scales, as described by Lecomte *et al.* (1992). This method successfully identifies an individual's sex 96% of the time, as determined from three cohorts of newborns that were later unambiguously sexed at the age of 1 year ($n = 525$, J.-F. Le Galliard, unpublished data). Newborns were released into the field and recaptured at the age of 1 month, at which time they were again measured for SVL. Growth rate was calculated as the increase in SVL

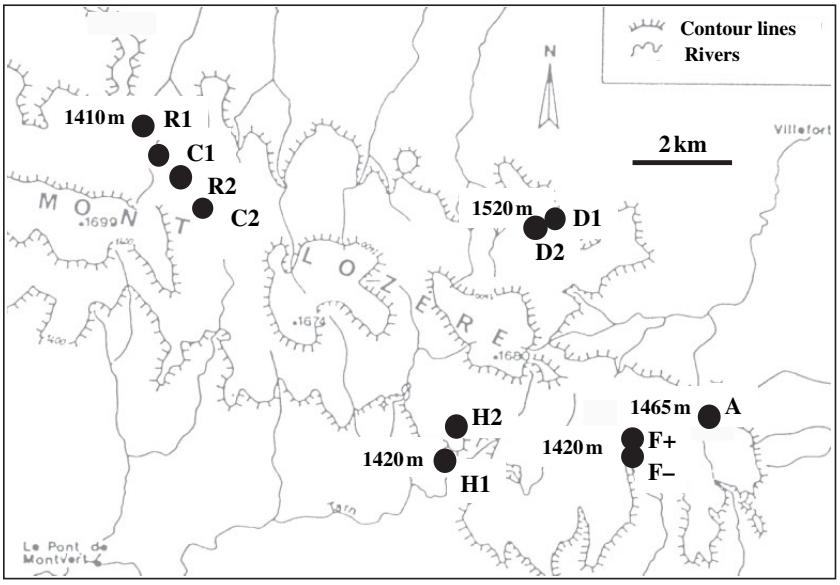

Fig. 1 Spatial location and altitude of the 11 study populations of common lizards in the Mont Lozère area, southern France. Each study site was approximately 0.5 ha in size. Temporal variation in sexual size dimorphism (SSD) and the effects of maternal food availability on offspring SSD were investigated in two neighbouring sites (F+ and F–; co-ordinates: 44°30′N, 3°45′E). Influence of population density and maternal corticosterone level were studied in two reduced-density sites (R1 and R2) and two control sites (C1 and C2). Effects of habitat humidity and maternal gestation conditions (heat and humidity in the laboratory) were tested in two dry sites (D1 and D2) and two humid sites (H1 and H2). Site A was included in the analysis of spatial effects on SSD.

from hatching divided by the number of days between hatching and recapture. We use the terms SSD-h to describe sexual dimorphism in SVL at hatching and SSD-g to describe sexual dimorphism in growth rate during the month after hatching. In our study sites, growth takes place primarily during the two first years of life, and individuals of both sexes reach maturity at the end of these 2 years (Uller *et al.*, 2004). Body size of 2-year-old individuals is positively correlated with their growth rate during the first month of life (Pearson correlation, $r = 0.29$, $P = 0.01$, $n = 72$).

Analysis of environmental variation in SSD

Experiment 1: Maternal food availability

To determine whether maternal food availability affects SSD of offspring, gravid females were captured from two long-term study sites (Massot & Clobert, 1995). These sites, henceforth called F− and F+ (see Fig. 1), are located in the same glade (1420 m a.s.l.). Site F+ has a higher structural diversity of habitat and shows a consistently higher density of lizards than site F− (700 adults ha^{-1} vs. 430 adults ha^{-1}). Approximately 10% of the juveniles identified at hatching migrate between these two sites, suggesting poor genetic isolation (Clobert *et al.*, 1994). However, hatchlings from site F− have a larger body size at birth and F− females lay later (Clobert *et al.*, 1994), indicating that these sites can be considered as two different demographic units.

Between the years 1990 and 2002, a total of 848 gravid females from these two sites were brought into the laboratory (Fig. 2). Females from each site were equally divided into low and high food treatments. Females were provided with two levels of food provisioning during an average period of 1 month before parturition: females were offered one moth larva (*Pyralis farinalis*, average live weight ± SD = 0.189 ± 0.051 g, $n = 30$) either every week in the high food treatment or every 2 weeks in the low food treatment. After giving birth, females were released together with their offspring within 1 m of the original capture site. A total of 3956 juveniles were produced from the females in this experiment. Food effects on SSD were analysed by comparing SSD-h and SSD-g of offspring between low food and high food treatments.

Some of the manipulated females were recaptured during the subsequent year and their food availability was again manipulated (treatments randomly assigned). This allowed us to contrast long-term maternal effects (i.e. how food manipulation on females in year $t - 1$ affects SSD of offspring born in year t) with short-term effects (i.e. how food manipulation on females in the current year affects offspring SSD).

Experiment 2: Population density and maternal corticosterone level

Here, we tested for effects of population density (prenatal and post-natal) and maternal corticosterone level on SSD of offspring (Fig. 2). The four sites in which we manipulated population density were distributed along a 2-km transect (1450 m a.s.l., Fig. 1). The natural population density of the general study area has been found to be stable through time, suggesting that population density might be at the carrying capacity (Massot *et al.*, 1992).

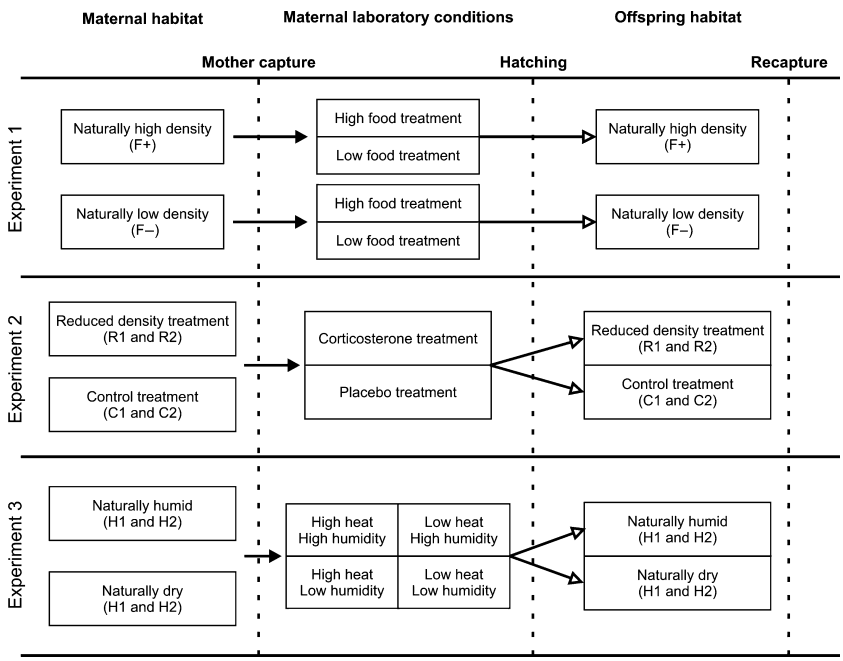

Fig. 2 Schematic representation of the three experiments (1, 2 and 3) utilized to assess ontogenic sources of variation in sexual size dimorphism (SSD) of juvenile common lizards. Prenatal sources of variation were investigated via manipulations of maternal habitat (2 months prior to parturition) and maternal conditions in the laboratory (1 month prior to parturition). Post-natal sources of variation were investigated via the immediate release of hatched offspring back into the field and their recapture at the age of 1 month. Except for Expt 1, offspring were reciprocally transplanted between maternal habitats. Effects on SSD at hatching (SSD-h) were assessed in the laboratory immediately after parturition, and effects on SSD during growth (SSD-g) were determined after 1 month in the field. Solid and open arrows show prenatal and post-natal treatment histories respectively.

We therefore randomly assigned our experimental sites into reduced-density or control treatments. Prior to our manipulations, all sites had similar population density (ca. 1000 individuals ha^{-1}) and population structure (Meylan & Clobert, 2004). In early spring of the years 2000 and 2001, i.e. during the early mating period and ca. 2 months before parturition, we removed 400 individuals from two of these four sites (R1 and R2, see Fig. 1) to produce a reduced-density treatment. The remaining two sites were left undisturbed and served as controls (C1 and C2, see Fig. 1).

One month after our density manipulations, we brought gravid females from all four of these sites into the laboratory (over an average period of 1 month), maintained them in individual terraria, and manipulated their corticosterone level (Fig. 2). This glucocorticoid hormone typically increases in response to high population density, and, at elevated concentrations, redirects behaviour and physiology towards survival (Wingfield & Ramenofsky, 1999; Cote *et al*., in press). A total of 341 females were randomly assigned to either the corticosterone or the control treatment. Plasma corticosterone was delivered through a noninvasive method: a mixture of corticosterone (C2505, Sigma-Aldrich, St. Louis, MO, USA) and sesame seed oil was applied daily to the skin of the back until parturition. Control females received an application of sesame seed oil only. A similar manipulation in a previous study on gravid female common lizards induced a 15-fold increase in plasma corticosterone, which is within the natural range observed for this species (Meylan *et al*., 2003).

Captive experimental females produced a total of 1705 juveniles that were released into the field 3 days after hatching. In order to test for interactions between prenatal effects (population density during early gestation and maternal corticosterone treatment during late gestation) and post-natal effects (population density) of sites into which juveniles were released, siblings from each family were separated randomly into two groups that were released into either a control or a reduced-density site. Mothers were always released at the site of their original capture. In contrast, offspring were never released at their mother's capture site to exclude possible confounding interactions between mothers and their young.

Experiment 3: Habitat humidity and maternal gestation conditions

In this experiment, we tested for the effects of maternal gestation conditions (humidity and heat in the laboratory) and the prenatal and post-natal effects of habitat humidity on offspring SSD (Fig. 2). In the years 1996 and 1997, we removed gravid females 1 month before parturition from four sites differing with respect to their habitat humidity (1420 m a.s.l. for the two humid sites, 1520 m a.s.l. for the two dry sites; Fig. 1). The two humid sites (henceforth called H1 and H2) were located in clearings that flooded in spring and late summer. The two dry sites (henceforth called D1 and D2) were located in heathlands that received water only from rain and dew (Lorenzon *et al*., 2001). The sites were isolated from each other by distance (50 m) and by a dense stand of trees, both of which constitute a strong barrier to population exchanges in the common lizard as demonstrated by Massot *et al.* (1992) and Clobert *et al.* (1994). The first axis generated by a principal component analysis on air humidity, flooded surface area and forest proximity (presence of trees helps to retain humidity) was used to calculate a habitat humidity index for each site (D1: −1.14, D2: −0.48, H1: 0.51 and H2: 1.10).

Gravid females taken from each of these sites were randomly assigned to one of four different gestation conditions in the laboratory prior to giving birth: two levels of heat regime were provided in a crossed design with two levels of humidity in the laboratory (Fig. 2). Maternal heat was provided with lamps either 3 h day^{-1} in the low heat treatment or 6 h day^{-1} in the high heat treatment. Different maternal humidity conditions were produced either by wetting terraria seven times per day in the low humidity treatment or 20 times per day in the high humidity treatment (Lorenzon *et al*., 2001). We verified that the manipulation produced significant differences in air humidity in the terraria (on average, 60.0% and 83.3% in the low and high humidity treatments, respectively). Three days after hatching, siblings from each of the 393 families were separated randomly into two groups that were released into one of the humid or dry field sites. Juveniles were never released into their population of origin. Mothers were released at their original site of capture.

Statistical analyses

We examined effects of all experimental manipulations on size at hatching and growth rate. In the common lizard, larger individuals tend to exhibit a slower growth rate (Massot *et al*., 1992). Thus, we included SVL at hatching as a covariate in models describing growth rates. SVL satisfied conditions of linearity with growth rate in all cases. For each of the three experiments outlined above, we built a full model including the effects of sex, experimental treatment, study year and all of the interactions between these factors. Some manipulations were conducted at the population level (e.g. population density), whereas others were conducted at the individual level (e.g. maternal food availability). To account for these nested factors, we included a random family effect (nested within maternal treatment, year and study site) and a site effect (nested within population treatment) when necessary. In the analysis of the results from Expt 1, we excluded data from 1994 and 1995 either because juveniles were not sexed, or because food treatments in the laboratory were not the same as in other years. In Expt 3, we were only able to utilize a subset of the collected data because 385 of the 1859 juveniles involved

were not sexed at hatching. Nevertheless, our manipulations produced effects on body size *per se* similar to those shown in a previous study involving the full data set (Lorenzon *et al.*, 2001), suggesting that the subset analysed here is representative of the original sample.

We used the LME procedure in R software to run all models (Ihaka & Gentleman, 1996). First, the normality and homogeneous variance of residuals and random effects of all models was verified (Pinheiro & Bates, 2002). Results and discussion of these analyses are presented in the Supplementary Material section. Secondly, we simplified models by backward elimination of nonsignificant terms (starting from higher-order interactions) until a minimum adequate model was achieved. Because all three experiments were conducted independently from each other, the risk of making type I errors was not inflated by multiple testing. We therefore used the critical level $\alpha = 0.05$ in our two-tailed statistical tests. Thirdly, we used the Aikake Criterion Index (AIC) to choose between two alternative ways of describing habitat humidity: a categorical classification of the four sites vs. a habitat humidity index (see description above).

Analysis of spatial and temporal variation

To study natural spatial variation in SSD of juveniles, we used data collected from seven sites surveyed both in 2000 and 2001. The following sites were included (see Fig. 1): F+, F−, R1, C1, R2, C2 and A (a heretofore unmentioned dry site with a low population density in which only SVL at hatching was measured; located at 1465 m a.s.l.). We modelled SVL at hatching and growth rate as a function of sex, site, year and the interactions between these factors. In addition, we examined long-term temporal variation in SSD from 1990 to 2002 within sites F+ and F−. In this analysis, we modelled SVL at hatching and growth rate as a function of sex, year and their interaction, while also including a random family effect.

Analysis of family effects

We conducted full-sib comparisons to study genetic variation in body size (Reusch & Blanckenhorn, 1998). We first modelled SVL at hatching and growth rate as a function of family (random effect), sex (fixed effect) and the interaction between sex and family (random effect) with the MIXED procedure in SAS (Littell *et al.*, 1996). We independently analysed data from the three experiments mentioned above irrespective of the experimental manipulations to which families were assigned (i.e. no maternal effect). However, site and year were included as factors to control for spatial and temporal variations in body size, and SVL at hatching was included as a covariate in the analysis of growth rate. In these analyses, a significant interaction between sex and family would indicate interfamilial variation in SSD and, thus, would suggest genetic variation in SSD. We then conducted separate analyses in males and females to calculate heritabilities (Lynch & Walsh, 1997; Reusch & Blanckenhorn, 1998; Jensen *et al.*, 2003). Variance components were estimated using restricted maximum likelihood estimation procedures (Littell *et al.*, 1996). The total phenotypic variance was partitioned in three components

$$V_P = V_A + V_E + V_R \quad (1)$$

where V_A was the variance among families, V_E was the variance among study sites and years and V_R was the residual variance. The heritability (h^2) of each trait was then calculated as

$$h^2 = 2\frac{V_A}{V_P} \quad (2)$$

Unless otherwise stated, all results are presented as least-square mean values (±SE).

Results

Family effects

We conducted three independent analyses of family effects on SSD-h and SSD-g. We found that females were longer at hatching than males (all $P < 0.0001$) and also tended to grow faster (one significant test out of three). Because the interaction between sex and family effects was only significant in one of three tests for hatching SVL and in one of three tests for growth rate, our data suggest that interfamilial variation in SSD-h and SSD-g is weak and inconsistent. In contrast, size at hatching and offspring growth rate varied strongly among families (five main family effects out of six tests) and showed large heritabilities in both males and females (Table 1). Maternal and environmental effects might have inflated these measures of interfamilial variation. However, even when factoring in such effects (using models described in the next sections), interfamilial variation remained strong both for body size ($P < 0.0001$, Expt 1: $h^2 = 0.52$; Expt 2: $h^2 = 0.46$; Expt 3: $h^2 = 0.42$) and growth rate ($P < 0.001$, Expt 1: $h^2 = 0.48$; Expt 2: $h^2 = 0.30$). However, in Expt 3, interfamilial variation in growth rate was not significant when factoring in maternal and environmental effects ($P = 0.89$).

Spatial variability in SSD

We found that females were longer than males at hatching ($F_{[1,2366]} = 190.63$, $P < 0.0001$, contrast = 0.64 ± 0.22 mm), but no sex-specific differences in growth rate were observed ($F_{[1,96]} = 1.69$, $P = 0.20$). SSD-h and SSD-g did not vary across study sites (Sex × Site: $F_{[6,2359]} = 0.87$, $P = 0.52$, and $F_{[5,90]} = 0.67$, $P = 0.41$, respectively). However, spatial variation in length at hatching and in growth rate was highly

Table 1 Sample size, phenotypic means and variances (V_P), and estimates of variance components and heritability (h^2) of body size in male and female juvenile common lizards.

Traits	*n*	Mean	V_P	V_A (SE)	V_R (SE)	h^2
Females						
SVL at hatching (mm)	1984	22.26	1.1813	0.6582 (0.0430)***	0.3391 (0.0141)	1.11
Growth rate (mm day^{-1})	240	0.128	0.0029	0.0010 (0.0003)**	0.0011 (0.0002)	0.68
Males						
SVL at hatching (mm)	2142	21.55	1.0859	0.5208 (0.0344)***	0.3546 (0.0137)	0.96
Growth rate (mm day^{-1})	298	0.124	0.0032	0.0011 (0.0003)***	0.0011 (0.0002)	0.72

Quantitative genetic estimates were generated with mixed-effects models fitted to male and female traits separately. Variance components include variance among families (V_A) and residual phenotypic variance (V_R, see Eqn 1 in the main text). For ease of illustration, data are given only for Expt 1, but similar results were obtained with Expts 2 and 3 involving a smaller number of families.
$^{**}P < 0.01$, $^{***}P < 0.001$.
SVL, snout-vent length.

significant ($F_{[6,595]} = 35.97$, $P < 0.0001$, and $F_{[5,300]} = 9.79$, $P < 0.0001$, respectively). We found that SVL at hatching was higher in eastern sites of the study area (sites F+, F− and A) than in western sites (sites R1, C1, R2 and C2, see Fig. 1) by an average of 1.22 ± 0.078 mm. In addition, site-dependent temporal variation occurred for both SVL and growth rate (Site × Year: $F_{[6,595]} = 2.77$, $P = 0.01$, and $F_{[5,300]} = 6.20$, $P < 0.0001$, respectively).

Temporal variability in SSD

This analysis again shows that females were longer at hatching than males ($F_{[1,3107]} = 1046.57$, $P < 0.0001$, contrast = 0.67 ± 0.07 mm). Additionally, females grew faster than males ($F_{[1,165]} = 26.32$, $P < 0.0001$, contrast = 0.02 ± 0.0068 mm day^{-1}). Remarkably, differences in body length between males and females both at birth and during growth remained fairly constant across study years (Sex × Year: $F_{[11,3096]} = 0.82$, $P = 0.62$, and $F_{[11,155]} = 1.05$, $P = 0.41$, respectively; see Fig. 3). We found inter-annual variation in length at hatching and in offspring growth rate ($F_{[11,836]} = 28.34$, $P < 0.0001$, and $F_{[11,306]} = 5.84$, $P < 0.0001$, respectively). SVL at hatching did not show any systematic change across years (Fig. 3a), although growth rate steadily increased in the early 1990s and then reached a plateau (Fig. 3b).

Effects of maternal food availability on SSD

In the analysis of effects of maternal food availability, site, and year on SSD-h (3350 observations, 698 family-year combinations, see Fig. 2), we found that females were longer at hatching than males ($F_{[1,2651]} = 928.3$, $P < 0.0001$, contrast = 0.67 ± 0.022 mm, Fig. 4a). However, we found that maternal food availability did not affect SSD-h (Food availability × Sex: $F_{[1,2612]} = 0.886$, $P = 0.347$) and, additionally, had no effect on body size at hatching ($F_{[1,658]} = 0.640$, $P = 0.42$, Fig. 4a). The final model showed year effects ($F_{[9,687]} = 19.99$, $P < 0.0001$) and that juveniles originating from site F− were longer at hatching than those originating from site F+ ($F_{[1,687]} = 4.25$, $P = 0.04$, contrast = 0.139 ± 0.068 mm). However, year, study site and their interaction had no effect on SSD-h (all $P > 0.34$).

To estimate effects of maternal food availability on offspring growth rate, a total of 353 juveniles from 223 families were recaptured at an approximate age of 1 month (39.9 days ± 7.71 SD). The final statistical model indicated substantial yearly variation in growth rate ($F_{[6,216]} = 6.03$, $P < 0.0001$) and a significant SSD-g ($F_{[1,128]} = 14.96$, $P = 0.0002$), with females growing faster than males (contrast = 0.018 ± 0.005 mm day^{-1}). However, as in the previous analysis on SSD-h, we found that year, study site and their interaction had no effect on SSD-g (all $P > 0.25$). Furthermore, maternal food availability had no effect on either SSD-g ($F_{[1,127]} = 1.16$, $P = 0.28$) or growth rate ($F_{[1,215]} = 0.01$, $P = 0.92$, Fig. 4b).

Comparisons of short-term and long-term effects of maternal food availability on offspring SSD were based on 112 recaptured adult females that gave birth to 597 juveniles during the second year of manipulations. Because some combinations of treatments and years had few observations, we only tested for effects of site, year, current maternal food availability, maternal food availability of the previous year, and interactions between sex and food availability treatments up to the third order. Again, we found that females were larger than males at hatching ($F_{[1,484]} = 198.7$, $P < 0.0001$). However, we observed no change in SSD-h with current ($F_{[1,482]} = 0.73$, $P = 0.39$) or past maternal food availability ($F_{[1,483]} = 1.55$, $P = 0.21$). SVL at hatching varied among years ($F_{[8,102]} = 3.16$, $P = 0.003$) and sites ($F_{[1,102]} = 8.90$, $P = 0.004$), but body length of offspring was not affected by current ($F_{[1,100]} = 1.29$, $P = 0.26$) or past ($F_{[1,101]} = 2.40$, $P = 0.12$) maternal food availability. There were too few recaptures (44 juveniles from 25 families) to evaluate the contribution of past maternal food availability on offspring growth rate.

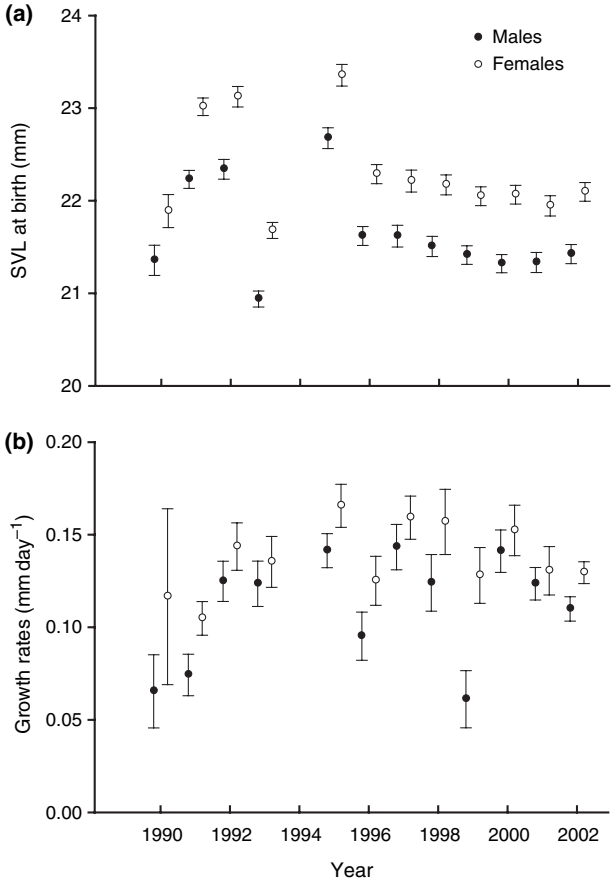

Fig. 3 Temporal variation in sexual size dimorphism (SSD) of common lizards at birth (a) and in association with growth (b). Averages (±SE) are illustrated according to sex and year. Even though male and females were examined within a similar time period, symbols are staggered for purposes of presentation.

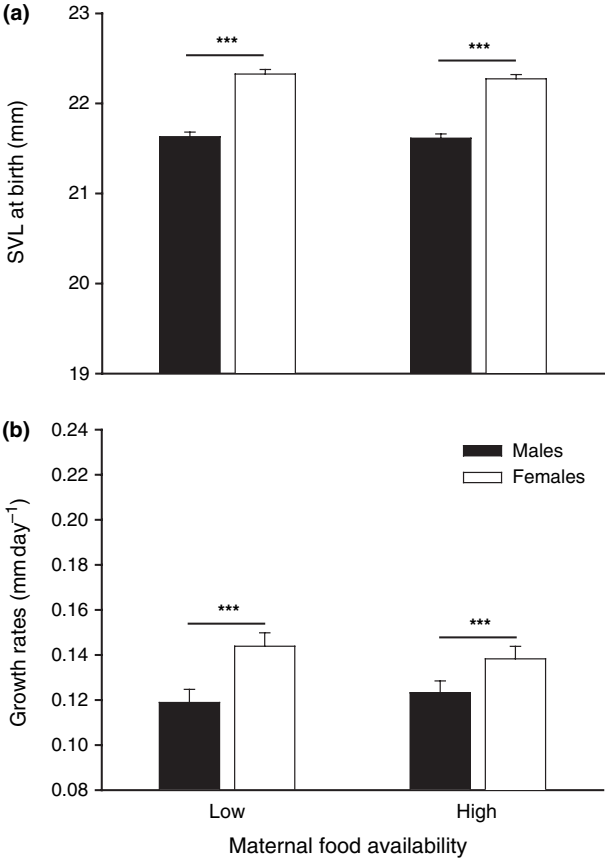

Fig. 4 Effects of maternal food availability on sexual size dimorphism (SSD) of common lizards at birth (a) and in association with growth (b). Averages (±SE) are illustrated according to sex and maternal food availability conditions. Least-square mean values were obtained with a model including year and site effects (***$P < 0.0001$).

Effects of density manipulation and maternal corticosterone level on SSD

In the analysis of the effects of prenatal population density and maternal corticosterone level on SSD-h (1705 observations, 367 families, see Fig. 2), we again found that females were significantly larger than males at hatching ($F_{[1,1337]} = 101.3$, $P < 0.0001$, contrast = 0.65 ± 0.064 mm). However, the degree of this difference was not influenced by maternal corticosterone levels ($F_{[1,1336]} = 0.10$, $P = 0.75$, Fig. 5a) or by prenatal population density (Prenatal density × Sex: $F_{[1,1335]} = 2.93$, $P = 0.09$; Prenatal density × Sex × Year: $F_{[1,1334]} = 0.93$, $P = 0.34$, see Fig. 5b). Offspring of females treated with corticosterone were smaller ($F_{[1,361]} = 109.06$, $P < 0.0001$) and the effects of prenatal population density on body length depended on the study year (Prenatal density × Year: $F_{[1,361]} = 10.54$, $P = 0.001$). A reduction of the prenatal population density tended to negatively affect body length at hatching in 2000, while it had a significant positive effect in 2001 (Fig. 5b).

A total of 332 juveniles were recaptured at the age of 42.2 days (±5.90 SD) for the determination of prenatal and post-natal effects of population density and maternal effects of corticosterone level on growth rate. We found three main results. First, SSD-g was affected by an interaction between the prenatal and post-natal effects of population density (Post-natal density × Prenatal density × Sex: $F_{[1,125]} = 5.84$, $P = 0.02$): females grew faster than males if exposed to reduced-density both prenatally and post-natally (Tukey adjusted contrast, $P = 0.02$), but not otherwise (all $P > 0.05$, Fig. 5d). However, prenatal density alone (Prenatal density × Sex: $F_{[1,125]} = 2.51$, $P = 0.11$) and post-natal density alone (Post-natal density × Sex: $F_{[1,125]} = 0.27$, $P = 0.60$) did not influence SSD-g. Secondly, maternal corticosterone treatment had a significant effect on offspring growth rate ($F_{[1,193]} = 14.18$, $P = 0.0002$) that did not depend on their sex

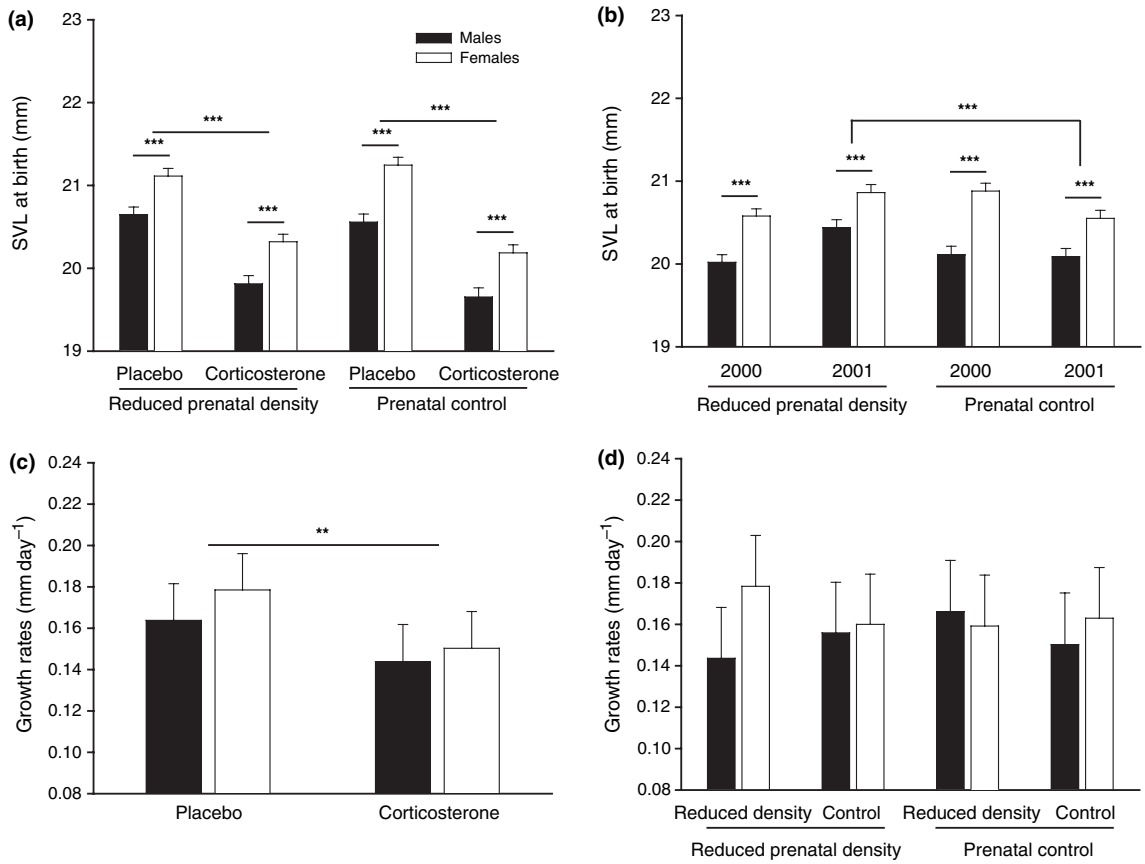

Fig. 5 Effects of population density (prenatal and post-natal) and maternal corticosterone levels on sexual size dimorphism (SSD) in juvenile common lizards. (a) Average snout-vent length (SVL) at hatching (±SE) according to sex, prenatal density and maternal corticosterone treatment. (b) Average SVL at hatching (±SE) according to sex, prenatal density and year. (c) Average growth rate (±SE) according to sex and maternal corticosterone treatment. (d) Average growth rate (±SE) according to sex, post-natal density and prenatal density. Data are least-square mean values of the selected models described in the main text (**$P < 0.01$, ***$P < 0.0001$).

($F_{[1,125]} = 0.18$, $P = 0.67$). Growth rate was 0.024 ± 0.006 mm day^{-1} lower in offspring from corticosterone-treated females than in offspring from placebo females (Fig. 5c). Thirdly, effects of post-natal population density on growth rate depended on study year (Post-natal density × Year: $F_{[1,125]} = 5.70$, $P = 0.02$). Experimental reduction of post-natal population density did not affect growth in 2000 (contrast = -0.008 ± 0.034 mm day^{-1}), but tended to increase growth in 2001 (contrast = 0.017 ± 0.033 mm day^{-1}).

Effects of habitat humidity and maternal gestation conditions on SSD

In the analysis of prenatal effects of habitat humidity and maternal effects (heat and humidity in the laboratory) on SSD-h (1474 observations, 353 families, see Fig. 2), a model incorporating habitat humidity as a categorical variable fit the data better than a model incorporating the continuous habitat humidity score (ΔAIC = −17.1). We thus described differences in habitat humidity using a categorical variable. We found that female offspring were again longer at hatching than males ($F_{[1,1120]} = 318.2$, $P < 0.0001$, contrast = 0.694 ± 0.039 mm). However, SSD-h was not affected by prenatal habitat humidity (Prenatal habitat humidity × Sex: $F_{[3,1116]} = 1.01$, $P = 0.39$, see Fig. 6a), maternal heat conditions, maternal humidity conditions, or any combination of these factors (all $P > 0.12$). In contrast, we found that hatchling body size *per se* depended on prenatal habitat humidity (Prenatal habitat humidity: $F_{[3,336]} = 2.82$, $P = 0.04$, see Fig. 6a) and on the interaction between prenatal habitat humidity conditions, maternal humidity and maternal heat conditions in the laboratory (Prenatal habitat humidity × Maternal humidity × Maternal heat: $F_{[3,336]} = 4.18$, $P = 0.006$). The latter indicates that effects of maternal gestation conditions in the laboratory depended on prenatal habitat humidity (maternal site).

We recaptured 214 juveniles from 146 families (average age: 38.9 days ± 8.36 SD) to test effects of prenatal

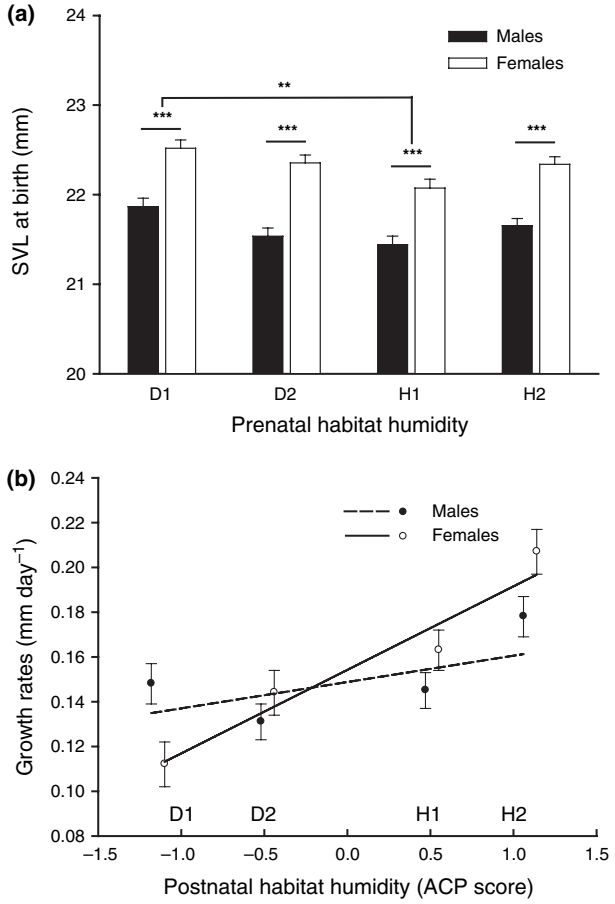

Fig. 6 Effects of habitat humidity on sexual size dimorphism (SSD) at birth (a) and SSD during growth (b). (a) Average snout-vent length (SVL) at hatching according to sex and prenatal habitat humidity. Prenatal habitats are D1 and D2 (dry sites 1 and 2), and H1 and H2 (humid sites 1 and 2). (b) Relationship between post-natal habitat humidity and average growth rate in male and female juveniles. Symbols are staggered for purposes of presentation. Data are least-square mean values (±SE) of the selected models described in the main text (**$P < 0.01$, ***$P < 0.0001$).

habitat humidity, maternal heat and maternal humidity in the laboratory, post-natal habitat humidity, and their first-order interactions on SSD-g. A model incorporating prenatal and post-natal habitat humidity as a continuous score fit the data better than a model incorporating a categorical classification (ΔAIC = −30.5). Thus, we described differences in habitat humidity using a continuous score and found that post-natal habitat humidity influenced SSD-g. Growth rate increased with post-natal habitat humidity ($F_{[1,63]} = 37.49$, $P < 0.0001$) and growth rate was affected to a greater degree by post-natal habitat humidity in females than in males (Post-natal habitat humidity × Sex: $F_{[1,63]} = 6.55$, $P = 0.013$; contrast between slopes = 0.018 ± 0.007, see Fig. 6b). As a consequence of this sex-specific sensitivity to post-natal habitat humidity, there was a reversal in SSD-g from wet to dry sites. As found in our previous analyses, females grew faster than males in H2, the wettest site. In contrast, males grew faster than females in D1, the driest site (Fig. 6b). In addition, we found that growth rate of offspring depended on maternal heat regime ($F_{[1,143]} = 10.68$, $P = 0.001$). Growth rate for juveniles was higher if mothers were provided with high heat during gestation (contrast = 0.020 ± 0.006 mm day^{-1}). However, neither maternal conditions experienced during gestation (heat and humidity in the laboratory) nor prenatal habitat humidity influenced SSD-g (all $P > 0.30$).

Discussion

Table 2 summarizes effects of the examined ontogenic factors on body size in male and female common lizards. As previously observed in other vertebrate species, body size *per se* was sensitive to both prenatal and post-natal factors. Also, temporal, spatial and interfamilial variation of body size in this species was

Table 2 Effects of examined ontogenic factors on body size in juvenile common lizards.

Sources of variation	Size at hatching *per se*	SSD-h	Growth rate *per se*	SSD-g
Family				
Expt 1	+	+	+	−
Expt 2	+	−	+	−
Expt 3	+	−	−	+
Year				
Long-term survey	+	−	+	−
Origin site				
Large-scale survey	+	−	+	−
Prenatal habitat humidity	+	−	−	−
Prenatal effects				
Population density	+*	−	−	+†
Maternal food availability	−	−	−	−
Maternal corticosterone level	+	−	+	−
Maternal humidity	+	−	−	−
Maternal heat	+	−	+	−
Post-natal effects				
Population density	n.a.	n.a.	+*	+†
Habitat humidity	n.a.	n.a.	+	+
Food availability‡	n.a.	n.a.	+	−

We report both the influential (+) and noninfluential (−) factors. A factor was considered as influential when it significantly affected body size as a main effect or when it was involved in a significant interaction with another factor (see main text).
*Interaction between effects of population density and study year.
†Only the interaction between prenatal and post-natal effects of population density was significant. No main effects of prenatal or post-natal density on SSD-g.
‡Post-natal effects of food availability are described in Le Galliard *et al.* (2005).
SSD, sexual size dimorphism; n.a., not available.

strong and of similar magnitude to the variation observed in natural populations of other vertebrates (Badyaev, 2002). With respect to SSD, we found that SSD at hatching (SSD-h) was always significant, with females being longer than males by ca. 0.6–0.7 mm. Sexual dimorphism in growth rate (SSD-g) was also often detected: female juveniles grew faster than males by ca. 0.3–0.6 mm per month. Thus, SSD-h and SSD-g in juvenile common lizards match qualitatively the SSD observed in adults. Because SSD in juveniles was not affected by most investigated prenatal and post-natal factors, and did not vary systematically between study years or families, size differences between sexes appear to be rather rigidly fixed (Table 2). Only post-natal habitat humidity showed a sex-specific effect on growth rate. In the following sections, we discuss whether these ontogenic patterns in body size represent adaptations or constraints.

Ontogenic basis of body size

Body size at hatching varied significantly among families, even when critical prenatal factors known to influence body size in the common lizard were controlled for. Such a large variation among families may suggest a strong genetic polymorphism, although it could also be due to some prenatal factors that were not taken into account in our analyses. Furthermore, we found significant prenatal effects of maternal heat regime and corticosterone level on offspring body size (Lorenzon *et al*., 2001; Meylan & Clobert, 2004), but no effects of maternal food availability. The absence of this effect could be due to the design of our manipulation, where food availability was modified only during the last month of gestation, i.e., after vitellogenin synthesis (Gavaud, 1986). Taken together, our results indicate that both genetic and short-term maternal effects can influence body size at hatching (Table 2). However, we suggest that more detailed quantitative genetic estimates be obtained from breeding experiments (Lynch & Walsh, 1997) to unambiguously assess the genetic basis of variation in body size.

Short-term maternal effects, i.e. heat regime and corticosterone level, influenced not only offspring body size, but also offspring growth rate. Both factors may have produced changes through organizational effects on development (Dufty *et al*., 2002). Growth rate was also influenced post-natally: offspring growth rate was higher in humid post-natal habitats and in reduced-density sites, although the latter depended on study year. Finally, interfamilial variation in growth rate was weaker than that for body size at hatching and, when several prenatal factors were controlled for, was not significant in Expt 3 (see Table 2). Thus, natural variation in growth rate seems to be influenced strongly by maternal and post-natal factors, and more weakly by genetic effects.

Prenatal effects on SSD

In contrast to the strong prenatal effects evidenced for body size *per se*, the key prenatal factors investigated here did not affect SSD of offspring (Table 2, Fig. 2). Although an interaction between prenatal and post-natal population density on SSD-g was found (see Fig. 5d), we do not count this as a relevant effect. First, experimental reduction of density increased offspring growth rate only during the second study year and, thus, may have interacted with other environmental factors we could not control for. Secondly, the reciprocal transplant experiment was intended to disentangle prenatal and post-natal effects of population density on offspring body size rather than test for an interaction between prenatal and post-natal density. The natural population density of *L. vivipara* is fairly stable over time (Massot *et al*., 1992), such that the densities experienced by mothers during gestation and, later, by their offspring are highly correlated.

Although we found no evidence for prenatal effects on SSD in the common lizard, studies conducted in other species suggest at least two mechanisms by which maternal effects might be produced: (i) through sex-dependent maternal allocation of metabolites or androgens (e.g. Cordero *et al*., 2001; Müller *et al*., 2002), or (ii) through modification of clutch sex ratio such that sex-specific secretion of androgens by embryos result in altered local androgen concentrations (e.g. Clark & Galef, 1995). Such mechanisms for maternal control of SSD may be highly relevant for the viviparous common lizard. First, mineral and hormonal exchanges between mother and eggs and, also, between eggs themselves are possible in this species (Dufaure & Hubert, 1961). Secondly, clutch sex ratio in the common lizard can be adjusted, suggesting that the potential for maternal control of local androgen concentrations exists (Uller *et al*., 2004). Furthermore, previous studies in lizards have shown that maternal allocation of metabolites and androgens may be mechanisms whereby variation in body size *per se* is produced. For example, in the oviparous *Uta stransburiana*, the amount of yolk deposited into eggs affects hatchling body size (Sinervo, 1999). In the common lizard, maternal corticosterone level and sex-specific androgen interactions between eggs influence size at hatching (Uller & Olsson, 2003; Uller *et al*., 2004). These mechanisms may be extended to produce effects also on SSD.

Despite the potential for maternal control of offspring SSD, our results indicate that early development of SSD in the common lizard is robust against changes in the prenatal environment. The absence of maternal effects on offspring SSD cannot be explained by a shortcoming in our experimental design: (i) most prenatal factors investigated here induced strong effects on body size *per se* (see Table 2), (ii) we manipulated a number of prenatal variables that showed the potential to be

relevant for SSD, including short-term factors (e.g. corticosterone levels during late gestation) and long-term factors (e.g. habitat humidity) and (iii) sample sizes of our experiments were large and should have been sufficient to detect significant effects. Thus, we propose two explanations for the poor sex-biased maternal effects on offspring size observed in this study. First, strong selection for the developmental stability of physiological processes shared between the sexes may occur at early ontogenetic stages (Badyaev, 2002). Secondly, although viviparity in the common lizard may provide the potential for maternal control of SSD (see Discussion above), it may also constrain maternal effects. In comparison with oviparous species that yolk eggs in a sequence, e.g. birds (Pike & Petrie, 2003), sex-specific investment of metabolites and androgens may be difficult in species that yolk all of their follicles simultaneously, such as the common lizard (T. Uller, personal communication). Furthermore, viviparous species may be less efficient in differentially allocating androgens between sexes due to hormonal leakage between eggs, as steroids originally sequestered within one egg are likely to influence neighbouring eggs during embryogenesis (Uller *et al.*, 2004).

Post-natal effects on SSD

In contrast to lack of maternal effects, we found that offspring SSD could be influenced post-natally: post-hatching habitat humidity produced a significantly faster growth rate in females than in males (Table 2, Fig. 2). Differences in habitat humidity between study plots were associated with differences in altitude, vegetation cover, temperature, population density and food availability (Lorenzon *et al.*, 2001). Thus, environmental factors other than post-natal habitat humidity may have contributed to SSD-g. However, a manipulation of air humidity in a controlled laboratory study produced effects on body size similar to those observed here (Lorenzon *et al.*, 1999), suggesting that post-natal habitat humidity in itself is capable of producing the observed sex-specific effects. Furthermore, even though average air temperature may vary between our study sites because of differences in elevation, slope and habitat structure (M. Massot, personal observation), we found no significant spatial variability in SSD. In addition, Chamaillé-Jammes *et al.*, 2006 have reported that a significant increase in air temperature during the 1990s in our main study site paralleled the change in growth rate, but we show no concomitant effects of this warming on SSD (see Fig. 3b). This suggests that post-natal temperature effects on SSD are weak or inconsistent. Finally, analyses indicate no effects of post-natal population density on SSD-g (see Table 2), and an independent laboratory study found no influence of food availability (Le Galliard *et al.*, 2005). Taken together, these results argue that changes in SSD-g are caused by post-natal habitat humidity *per se*, rather than by associated factors.

In lizards, habitat dryness increases evaporative water loss that occurs mainly in association with basking and foraging activities. In the face of water shortage, lizards reduce the time invested in these behaviours, which in turn decreases metabolic rate and nutritional intake and, as a consequence, growth (Lorenzon *et al.*, 1999; Sears & Anguiletta, 2003). Thus, the observed negative correlation between growth and habitat dryness might be the consequence of a trade-off between behaviours associated with the conservation of water when compared with those involved in thermoregulation and foraging. Because post-natal habitat humidity affected growth more strongly in females than in males, sex-specific differences may exist in effects of habitat humidity on water loss. In lizards, rate of evaporative water loss increases in proportion to body area. Mean body area of juvenile common lizards is approximately 5% higher in females than in males, and adult females also have a larger body area than males (measurements on standardized digitalized pictures, $F_{[1,27]} = 20.96$, $P < 0.0001$, $R^2 = 0.43$, $n = 29$; female/male ratio = 1.18). This suggests that water loss in female common lizards may be more sensitive to dry conditions. Thus, the observed sex-specific plasticity in growth relating to post-natal habitat humidity may be adaptive because it helps conserve water in the sex more susceptible to water loss, i.e. in females.

Whereas the plasticity observed here may reflect sex-specific adaptations, we found no evidence for population-specific adaptations: the reaction norms of growth rate in relation to post-natal habitat humidity were similar between juveniles born of mothers originating from dry sites and juveniles born of mothers originating from wet sites. Such poor differences between maternal habitats suggest the absence of genetic adaptations against water loss in drier sites. Genetic adaptations at the population level in the lizards studied here may be prevented by gene flow (Scheiner, 1998) from the wet sites that surrounds the dry sites in our study area (M. Massot, personal observation). Future studies that contrast habitats separated by greater dispersal barriers are therefore needed to address the potential for genetic adaptations to habitat humidity.

Conclusion

Female juvenile common lizards were consistently larger and grew faster than males over a wide range of manipulated environmental conditions, thereby suggesting that natural variation in SSD is primarily determined by sex-biased genetic factors. Furthermore, SSD measured in the field was similar to that previously observed in the laboratory under controlled feeding conditions (Le Galliard *et al.*, 2005). Thus, genetic factors may operate on differences in how individuals of each sex allocate resources to growth. The weak and inconsistent interfamilial variation in SSD we observed further indicates a strong genetic correlation between sexes and suggest that any genetic response of SSD to selection would be slow.

Sexual size dimorphism was not affected by any of the prenatal factors investigated here, leading us to suggest that viviparity might constrain the evolution of sex-biased maternal control of offspring SSD. To test this hypothesis, differential effects of the mode of reproduction (viviparity vs. oviparity) on the ontogeny of sexual differences in body size must be evaluated. The common lizard is one of the few lizard species that exhibits both oviparous and viviparous populations (Surget-Groba *et al*., 2001) and should provide an ideal model in which to test our prediction.

In contrast to maternal factors, post-natal environmental conditions produced effects on SSD: decreased post-natal habitat humidity caused a stronger reduction in growth in females than in males. This phenotypic plasticity may be the reason why a thorough comparative analysis of SSD in lizards could only explain a small proportion of the variation in SSD by phylogeny and sex-specific selection on body size (Cox *et al*., 2003). In many vertebrate species, post-natal plasticity in SSD arises as a consequence of differential energy demands between sexes, with the larger and faster-growing sex being more susceptible to the environment (Clutton-Brock *et al*., 1985). However, male and female juvenile common lizards have been shown to have similar energetic requirements (Le Galliard *et al*., 2005). Thus, we propose that effects of post-natal habitat humidity on SSD are most probably mediated by adaptive sex-specific behavioural responses to water limitation. However, future experiments should more precisely address the effects of habitat humidity on behaviour, water loss and lifetime reproductive success within each sex.

Acknowledgments

The authors are grateful to W. Blanckenhorn and one anonymous reviewer for their constructive comments. We also thank P. Lorenzon for sharing the data of Expt 3, P. Fitze and J. Cote for providing us with their pictures of lizards, and T. Uller for stimulating discussion on sex-biased maternal allocation. Financial support was received from research grants provided by the French Ministère de l'Education Nationale, de la Recherche et des Technologies, the Action Concertée Incitative 'Invasions biologiques' of the French Ministère de l'Aménagement du Territoire et de l'Environnement, and the program 'Observatoire de Recherche en Environnement' of the French Ministère de l'Education Nationale, de la Recherche et des Technologies.

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Supplementary Material

The following supplementary material is available for this article online:

Appendix S1 Heteroscedasticity modelling.

This material is available as part of the online article from http://www.blackwell-synergy.com.

Received 21 September 2005; revised 2 November 2005; accepted 7 November 2005