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Mother's timing and duration of corticosterone exposure modulate offspring size and natal dispersal in the common lizard (*Lacerta vivipara*)

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Abstract

Although multiple condition dependence in dispersal is common, the proximate mechanisms that integrate information from multiple sources remain largely unknown. In the common lizard (*Lacerta vivipara*), earlier studies have shown that maternal plasma corticosterone level interacts with maternal phenotype to affect offspring phenotype and dispersal strategy, and may reflect the mother's external and/or internal environment. However, the mechanism by which these two types of environmental information are integrated has not been identified. We explored the possibility that the timing and duration of the corticosterone signal are the key factors by which the message is modulated. We found that the timing of corticosterone exposure affects the juvenile phenotype: an exposure to corticosterone early in development has negative effects on juvenile size, weight, and body condition, that can nevertheless be restored in the case of a prolonged exposure. The duration of corticosterone exposure affects the dispersal strategy, although the precise effect depends on the sex ratio of the clutch. That is, in female-biased clutches, a prolonged exposure during gestation promotes philopatry of juveniles, while a short exposure enhances their dispersal, a result that is consistent with kin competition theory. Therefore, our results demonstrate that while corticosterone titer may signal a mother's external and/or internal environment to her developing young, differences in the timing and duration of this endocrine signal produce offspring with specialized phenotypes that exhibit different dispersal strategies.

Keywords: Corticosterone; Maternal effects; External and internal environments; Dispersal

Introduction

Dispersal is an interesting life-history trait because it involves a decision (i.e., to stay or to leave) at the individual level that has consequences up to the population and meta-population scale (Clobert et al., 2001). Causes of dispersal (e.g., habitat variability, intraspecific competition, inbreeding avoidance) are numerous (Clobert et al., 2004; Johnson and Gaines, 1990), and dispersal is often influenced by multiple factors, that interact to produce complex dispersal responses (Dobson and Jones, 1985; Gandon and Michalakis, 2001; Massot et al., 2002; Perrin and Goudet, 2001). To assess variation in their local habitat, individuals use various cues that describe the condition of their social and non-social environments (Ims and Hjermann, 2001). Indeed, it has been demonstrated theoretically that condition-dependent dispersal is superior to fixed dispersal responses in most environments (McPeek and Holt, 1992).

Prenatal factors (Mousseau and Fox, 1998) may play a role in the pre-adaptation of offspring to a particular type of dispersal behavior because they can affect multiple juvenile characteristics simultaneously and lead to the production of specialized phenotypes, allowing for maternal control over offspring dispersal rate (Murren et al., 2001). Such early maternal influences may shape dispersal strategies in response to environmental cues that are not directly available to the individual during embryonic development.

Maternal hormones, especially steroid hormones, may deeply affect offspring phenotype and fitness (Dufty et al., 2002; Groothuis et al., 2005). Steroid hormones modify brain organization during development (Nelson, 1994), which affects physiology and behavior throughout an individual's life

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(Groothuis et al., 2005; Schwabl, 1996). Corticosterone is a steroid hormone involved in the general stress response in vertebrates (Axelrod and Reisine, 1984), but it is known to have many pleiotropic effects. Indeed, corticosterone has been shown to affect brain organization (Gould and Tanapat, 1999; Matthews, 2000), distribution and density of steroids receptors (Szuran et al., 2000), immune responsiveness (Rubolini et al., 2005; Svensson et al., 2002), energy metabolism (Harvey et al., 1984; Hayward and Wingfield, 2004; Sinervo and de Nardo, 1996), and many behaviors (e.g., foraging, Astheimer et al., 1992; locomotion, Belthoff and Dufty, 1995; risk avoidance, De Fraipont et al., 2000; and territoriality, De Nardo and Sinervo, 1994). Corticosterone thus appears to be a general signal that conveys many different messages depending on the neuroendocrine context (Orchinik, 1998), and it could play a role in the adaptive plasticity of natal dispersal (Dufty and Belthoff, 2001; Dufty et al., 2002).

In the common lizard (*Lacerta vivipara*), prenatal corticosterone exposure affects natal dispersal, alone (De Fraipont et al., 2000) or through interactions with maternal age or physical condition (Meylan et al., 2002, 2004). The results of these studies are consistent with the idea that increased levels of corticosterone in old females (or females in poor condition) convey a message about the females' internal environment that reflects their low probability of survival. Natal philopatry increased in the young of mothers with elevated corticosterone levels because the probability of mother–offspring competition was reduced. The opposite trend was observed for offspring of young or healthy females, in which an increased level of corticosterone was thought to reflect poor environmental conditions (Meylan et al., 2002, 2004).

The above experiments demonstrate that prenatal effects of corticosterone on natal dispersal are condition-dependent, but the mechanism by which the message is modulated is poorly understood. For instance, physiological interactions with other hormones may be important in modulating the action of corticosterone (Meylan et al., 2004). Another, more likely, explanation is that variation in signal duration or timing results in peak hormone secretions during specific developmental stages that could convey different information about the female's internal and external environments, and could direct embryonic development along different ontogenetic pathways. In other species, poor body condition due to poor health is characterized by chronically elevated baseline corticosterone levels (Wingfield and Ramenofsky, 1999), while degradation of environmental conditions is characterized by an acute, more ephemeral elevation in corticosterone level. Therefore, we predicted that chronic corticosterone exposure would decrease the extent of offspring dispersal, as the probability of mother–offspring conflict would be reduced. On the other hand, we expected that brief corticosterone exposure would increase offspring dispersal, in response to the perceived degradation of environmental conditions. Furthermore, in previous experiments, the exposure level of females to corticosterone varied, depending on individual capture and parturition date. Therefore, it is possible that interactions between female body condition and the timing and/or duration of the corticosterone exposure induced variation in the dispersal response of offspring. To test these hypotheses, we artificially increased corticosterone levels in gravid females during the last month of gestation. Corticosterone treatments varied in length (2 or 4 weeks), and in developmental timing (first 2 weeks or last 2 weeks) such that each female within a treatment group received the same level of corticosterone. We followed the effects of the different hormonal treatments on female reproduction and offspring natal dispersal.

Methods

The species

L. vivipara is a small (adult snout–vent length ranges from 50 to 70 mm), live-bearing lacertid lizard, found throughout Europe and Asia. Our study population is located on Mont Lozère in southern France (44°39′N, 3°45′E, altitude 1455 m). In this population, males emerge from hibernation in mid-April, followed by yearlings, and adult females in mid-May. Mating occurs at female emergence, and gestation lasts for 2 months. A primitive chorioallantoic placenta allows respiratory and water exchange between mother and embryos during pregnancy (Panigel, 1956). Parturition starts in July and lasts for 2–3 weeks, and average clutch size is five soft-shelled eggs (range 1–12). Offspring hatch 1–2 h after the eggs are laid and are immediately independent of their mother. Juvenile dispersal occurs within the first 10 days of life (Léna et al., 1998), and the active season ends in late September. A more detailed description of *L. vivipara* life history can be found in Massot et al. (1992) and Clobert et al. (1994).

Corticosterone treatment

In June 2002, 130 gravid females were captured and kept in the laboratory until parturition. Females were housed in plastic terraria containing damp soil and a shelter; water was provided *ad libitum*. One larva of *Pyralis farinalis* was offered every week. The females were exposed to natural daylight and were heated 6 h/day with an electric bulb. All animals were treated in accordance with the *National Institutes of Health Guide for Care and Use of Laboratory Animals*.

Corticosterone levels in gravid females were artificially elevated following the non-invasive method of Knapp and Moore (1997), as modified for the common lizard by Meylan et al. (2002). Corticosterone was delivered transdermally on the back of the animal using a mixture of the steroid hormone and sesame oil. Corticosterone (Sigma C2505) was diluted in commercial sesame oil to a concentration of 3 μg/1 μL, and 4.5 μL of this solution were administered daily. The high concentration of lipids in lizard skin enables lipophilic molecules such as steroid hormones to cross the skin readily (Mason, 1992). This method leads to a five- to ten-fold increase of plasma corticosterone levels in the common lizard (mean basal corticosterone plasma level in females 21.64 ng/mL; mean corticosterone level in females experiencing transdermal treatment 281.9 ng/mL; Meylan et al., 2003). Such an increase keeps those levels within the species' natural range and corresponds to the range of a natural response to acute stress in reptiles (Cote et al., 2006).

Different corticosterone treatments were applied to each of the three groups of females. These treatments varied in terms of the duration and timing of the corticosterone application: 1) an early treatment, with daily corticosterone application during the first 2 weeks (June 15–June 30, $n=25$) followed by daily application of sesame oil during the next 2 weeks; 2) a late treatment, with sesame oil application during the first 2 weeks, and corticosterone application during the next 2 weeks (July 1st–July 15, $n=24$); 3) a chronic treatment, with corticosterone application during all 4 weeks (June 15–July 15, $n=26$). To control for the potential stressful effects of a daily manipulation, another group of females was treated only with sesame oil during all 4 weeks (June 15–July 15, $n=25$). Finally, an additional control group was not manipulated ($n=30$). As the increase in plasma corticosterone level induced by the transdermal application is much higher than the increase observed in response to prolonged captivity or handling stress (around 40 ng/mL, Dauphin-Villemant and Xavier, 1987), our

experimental corticosterone treatments are likely to mimic severe stress, and thus generate different responses than in the control groups.

We did not measure plasma corticosterone levels in the various treatment groups, because blood sampling during gestation can cause abortion. Similarly, we also did not try to measure corticosterone after parturition, because parturition is associated with a peak of corticosterone secretion in the common lizard (Dauphin-Villemant and Xavier, 1986), which would have confounded any treatment effects on corticosterone plasma levels in the various groups. Instead, we relied on the results of Meylan et al. (2003), who previously determined that our corticosterone treatment strongly increases plasma corticosterone levels in females and that females treated with sesame oil have plasma levels similar to those found in untreated females. The dynamics of corticosterone elevation and metabolism have not been analyzed either, for the same reasons. However, in an earlier study, Dauphin-Villemant et al. (1990) have demonstrated that the metabolism rate of corticosterone was higher during gestation than in other periods, and that the half-life of corticosterone in gravid females was around 25 min. Therefore, because this rapid diminution of corticosterone plasma concentration reflects a high degradation rate in this species, we can safely assume that the physiological distinction between different treatments was really effective.

There was no overlap between parturition and hormonal treatments (the first clutch was laid on July 16th, the last on August 2nd), and treatment had no effect on parturition date ($F_{4,80}=0.3$, $p=0.87$). However, parturition date was affected by clutch size ($F_{1,80}=3.77$, $p=0.056$). Consequently, females were distributed randomly among treatment groups with regard to their gestation stage, and the effect of treatment timing did not interact with gestation stage (i.e., the "late" treatment was indeed applied later in gestation on average than the "early" treatment).

Measurement of natal dispersal

A total of 414 juveniles were obtained from all females. At birth, offspring were measured (snout–vent length or SVL), weighed and sexed by counting ventral scales (Lecomte et al., 1992). We followed an experimental procedure to obtain a direct measure of natal dispersal and to keep a large sample size. Juvenile dispersal was studied through an experimental design involving wooden enclosures (7.5 m×1.2 m), each consisting of four boxes connected by small holes that allowed movement of juveniles, but not adults, throughout the structure (see De Fraipont et al., 2000; Léna et al., 1998; Meylan et al., 2002, for a similar approach). Mother and offspring were introduced into the first box 4 days after parturition, and juveniles were allowed to move about for 5 days. Location of juveniles in the different boxes was recorded everyday. Juveniles that stayed in the initial box where their mother was present were considered "residents". Juveniles that left the initial box were considered "dispersers". Juveniles that left the first box never returned to it. This protocol provides good estimates of natal dispersal and is similar in timing (Clobert et al., 1994) and in dispersal rate to what is found in natural conditions (De Fraipont et al., 2000; Léna et al., 1998; Meylan et al., 2002).

After the experiment, mothers and offspring were released into their natural population, at the mother's capture point.

Statistical analysis

Body condition was calculated as the residual of the relationship between body mass and snout–vent length (for females $F_{1,128}=369.83$, $p<0.0001$, $r^2=0.75$; for juveniles $F_{1,412}=173.45$, $p<0.0001$, $r^2=0.30$).

The effects of hormonal treatment on female and juvenile characteristics were investigated as follows:

- The effect of handling stress was tested by comparing the un-manipulated females with females treated with sesame oil. If daily handling induced significant stress, these two groups should differ with regard to some of the variables analysed.
- The general effect of corticosterone was tested by comparing all females treated with corticosterone (LG-CORT, E-CORT and LT-CORT being pooled into the CORT group) to control females (CONTROL and OIL being pooled into the NON-CORT group). If the exposure to corticosterone per se, at any time in gestation, was responsible for certain effects on female reproductive success, the groups exposed to corticosterone should share common features in comparison to the control groups.
- The effect of treatment duration was tested by comparing females treated for 2 weeks (E-CORT and LT-CORT being pooled into the SHORT-CORT group) with females treated for 4 weeks (LG-CORT). If the duration of corticosterone elevation is indeed characteristic of different types of stress (a long signal being related to major internal stress with long-term consequences on female survival; a short signal being rather related to temporary internal or environmental stress with few consequences on female survival), an adaptive phenotypic plasticity of reproduction should generate different responses of female and/or juvenile characters depending on treatment duration.
- The effect of treatment timing was tested by comparing females treated the first 2 weeks (E-CORT) with females treated the last 2 weeks (LT-CORT). If corticosterone has major organisational effects at precise stages of embryonic development, these two groups should differ in some of the variables analysed, and in particular in variables describing juvenile phenotype.

We used a Bonferroni correction to account for the effect of these multiple tests, and the significance threshold of our statistical results was therefore lowered to 0.0125.

We also analyzed all treatments separately, and tested for their respective effects, but this did not modify the main results. We present only the results of the pooling method described above.

Female characteristics

Parturition date and clutch size were analyzed with an analysis of variance (GLM procedure, SAS Institute). For binomial variables (mortality, clutch hatching success, clutch sex-ratio), we used a logistic regression analysis (GENMOD procedure, SAS Institute). We tested the effects of treatment, female SVL, and female body condition. *F*-tests (GLM procedure) and likelihood ratio tests (χ^2 values, GENMOD procedure) were used to assess the significance of effects.

Juvenile characteristics

Analysis of data involving siblings raises a statistical difficulty, as siblings cannot be considered as independent statistical units (Massot et al., 1994). Therefore, to analyze the effect of hormonal treatment and maternal characteristics on juvenile morphology, we used mixed models (MIXED procedure, SAS Institute) including the mother as a random effect, and maternal SVL and body condition as covariates. For the analysis of dispersal rate, we used a mixed logistic regression analysis (GLIMMIX procedure, SAS Institute). We tested the effects of treatment, maternal SVL, maternal body condition, sex, and clutch sex-ratio on morphology, and the effects of treatment, maternal SVL, maternal body condition, clutch sex-ratio, sex, juvenile SVL and body condition on dispersal. Estimations and test statistics were calculated with a restricted maximum likelihood approach. *F*-tests were used to assess the significance of fixed effects. Random family effects always accounted for a significant part of variance (all $p<0.0005$), and thus were always included in models for juvenile characteristics.

We used type III sum of squares in all the above analyses. We started with a general model including all the potential effects and their interactions (up to three-way interactions). We then dropped the non-significant effects (backward selection), starting with the most complex interaction terms. Only the results of the final model are reported. The *p*-values for the non-significant effects of treatment are the *p*-values for a reduced model with no interactions, and with all other non-significant effects removed.

Results

Mean values for female and clutch characteristics, and least square mean values for juvenile characteristics are listed in Table 1.

Table 1
Means of females and clutch characteristics, least square means of juvenile characteristics and standard errors in the different treatment groups

Treatment group variable	CONT	OIL	LG-CORT	E-CORT	LT-CORT
Female mortality	0.03 (0.033)	0.08 (0.056)	0.11 (0.065)	0.16 (0.074)	0.12 (0.069)
Clutch size	3.96 (0.482)	3.13 (0.598)	3.25 (0.573)	3.48 (0.592)	3.3 (0.478)
Clutch hatching success	0.8 (0.075)	0.56 (0.102)	0.61 (0.098)	0.52 (0.102)	0.75 (0.090)
Clutch sex-ratio	0.45 (0.055)	0.47 (0.052)	0.42 (0.057)	0.52 (0.048)	0.46 (0.059)
Juvenile SVL (cm)	1.946 (0.015)	1.928 (0.019)	1.900 (0.018)	1.866 (0.020)	1.946 (0.017)
Juvenile weight (g)	0.173 (0.0039)	0.164 (0.0051)	0.157 (0.0048)	0.151 (0.0053)	0.172 (0.0046)
Juvenile body condition index	0.0059 (0.030)	−0.0017 (0.0039)	−0.0046 (0.0037)	−0.0071 (0.0040)	0.0053 (0.0035)
Juvenile dispersal rate	−0.148 (0.349)	−0.448 (0.448)	−0.274 (0.436)	−0.917 (0.479)	−1.090 (0.422)

CONT: un-manipulated control, OIL: sesame oil control treatment, LG-CORT: long corticosterone exposure, E-CORT: early corticosterone exposure, LT-CORT: late corticosterone exposure.

There was no difference between the two control groups either in female reproductive success (female mortality $\chi^2_1=0.38$, $p=0.54$; clutch size $F_{1,48}=0.4$, $p=0.53$; clutch hatching success $\chi^2_1=3.48$, $p=0.06$, clutch sex-ratio $\chi^2_1=0.00$, $p=0.95$, $n=55$), or in juvenile characteristics (SVL $F_{1,35}=0.69$, $p=0.41$; body mass $F_{1,35}=2.80$, $p=0.10$; body condition $F_{1,35}=3.67$, $p=0.063$; dispersal rate $F_{1,35}=0.16$, $p=0.69$). Handling did not significantly affect any variable studied in the experiment.

Female mortality was only weakly related to female body condition ($\chi^2_1=3.96$, $p=0.047$, $n=130$), females with low body condition having a higher mortality rate. Clutch size and clutch hatching success depended on both female SVL and body condition, large females or females with high body condition having larger clutches and higher clutch hatching success (for clutch size: effect of SVL $F_{1,116}=15.07$, $\boldsymbol{p=0.0001}$, effect of body condition $F_{1,116}=18.86$, $\boldsymbol{p<0.0001}$; for clutch hatching success: effect of SVL $\chi^2_1=7.69$, $\boldsymbol{p=0.006}$; effect of body condition $\chi^2_1=7.86$, $\boldsymbol{p=0.005}$, $n=130$). However, female reproductive success was not affected by corticosterone exposure (female mortality $\chi^2_1=1.78$, $p=0.18$; clutch size $F_{1,116}=0.01$, $p=0.94$; clutch hatching success $\chi^2_1=0.17$, $p=0.68$, clutch sex-ratio $\chi^2_1=0.02$, $p=0.87$, $n=130$), by treatment duration (female mortality $\chi^2_1=0.38$, $p=0.54$; clutch size $F_{1,64}=0.01$, $p=0.90$; clutch hatching success $\chi^2_1=0.00$, $p=0.96$, clutch sex-ratio $\chi^2_1=0.18$, $p=0.67$, $n=75$), or by treatment timing (female mortality $\chi^2_1=0.04$, $p=0.85$; clutch size $F_{1,40}=0.05$, $p=0.82$; clutch hatching success $\chi^2_1=3.11$, $p=0.078$, clutch sex-ratio $\chi^2_1=0.03$, $p=0.83$, $n=49$).

Juvenile SVL and body condition were affected by sex (SVL $F_{1,328}=49.91$, $\boldsymbol{p<0.0001}$; body condition $F_{1,328}=43.80$, $\boldsymbol{p<0.0001}$): juvenile females were larger and had a lower body condition than juvenile males. Juvenile morphology was not affected by corticosterone exposure (SVL $F_{1,83}=3.62$, $p=0.060$; weight $F_{1,83}=3.51$, $p=0.060$; body condition $F_{1,83}=1.98$, $p=0.163$), or by treatment duration (SVL $F_{1,45}=0.18$, $p=0.67$; weight $F_{1,45}=20.81$, $p=0.37$; body condition $F_{1,45}=0.74$, $p=0.39$). However, juvenile snout–vent length and juvenile body mass, but not juvenile body condition, were significantly affected by treatment timing (SVL $F_{1,29}=10.08$, $\boldsymbol{p=0.003}$; body mass $F_{1,29}=7.36$, $\boldsymbol{p=0.011}$, body condition $F_{1,29}=3.44$, $p=0.074$). Juveniles exposed to corticosterone early in development were smaller and lighter than juveniles exposed to corticosterone later in development (Fig. 1).

Natal dispersal was not affected by corticosterone exposure ($F_{1,82}=1.98$, $p=0.16$) or by treatment timing ($F_{1,28}=0.25$, $p=0.62$), alone or in interaction, but it was affected by the interaction between treatment duration and clutch sex-ratio ($F_{1,180}=13.6$, $\boldsymbol{p=0.0003}$). Offspring from females briefly exposed to corticosterone treatment dispersed more when the

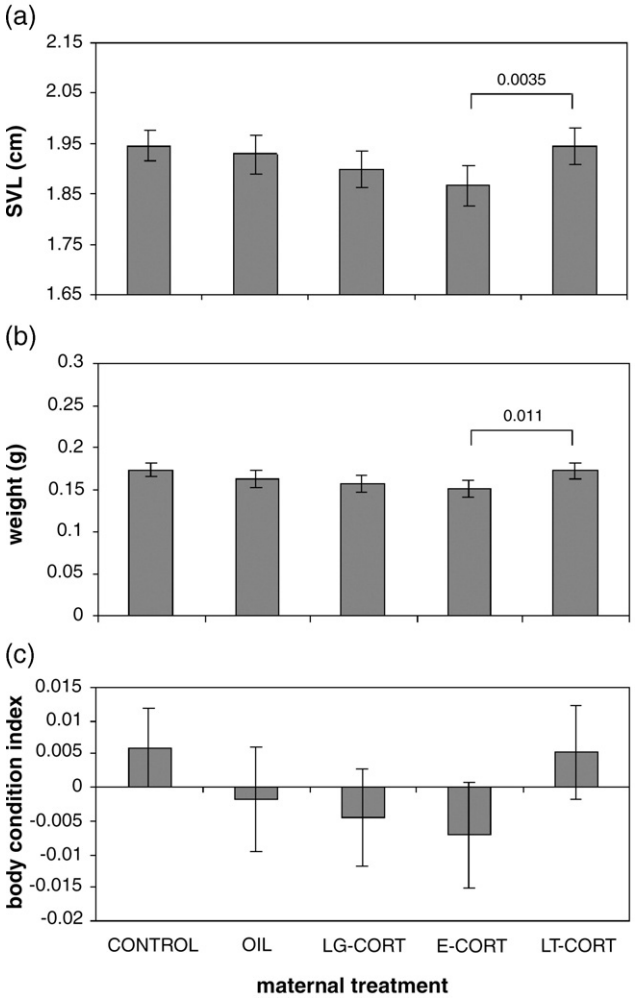

Fig. 1. Effect of maternal treatment on juvenile morphology (LG-CORT: long corticosterone exposure, E-CORT: early corticosterone exposure, LT-CORT: late corticosterone exposure, OIL: sesame oil control treatment, CONT; un-manipulated control). (a) Snout–vent length. (b) Weight. (c) Body condition. Error bars are 95% confidence interval (±2 S.E.).

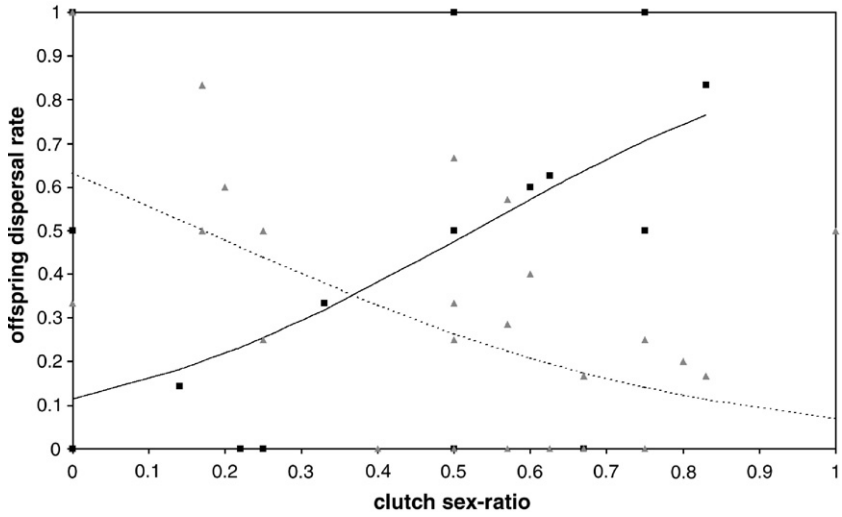

Fig. 2. Model fits for the effect of clutch sex-ratio (proportion of males) on dispersal rate in the case of a long (solid line, black squares: means per female) or a brief (dashed line, white triangles: means per female) period of corticosterone exposure. The interaction between treatment duration and clutch sex-ratio is also significant at the family level ($\chi^2_1=4.17$, $p=0.0411$, $n=47$).

male/female sex-ratio of their clutch was low (i.e., in female-biased clutches; $F_{1,118}=9.92$, $\boldsymbol{p=0.002}$), whereas the opposite trend was found for offspring from mothers chronically exposed to corticosterone although this trend was not significant alone ($F_{1,62}=3.22$, $p=0.078$) (Fig. 2).

When analyses were conducted at the family level, the same results were found: corticosterone exposure and treatment timing had no effect on natal dispersal (for corticosterone exposure $\chi^2_1=0.82$, $p=0.36$, $n=85$; for treatment timing $\chi^2_1=0.02$, $p=0.90$, $n=31$). However, natal dispersal was affected by the interaction between treatment duration and clutch sex-ratio ($\chi^2_1=4.17$, $p=0.0411$, $n=47$, Fig. 2).

Discussion

The stress induced by handling affected neither the reproductive success of females nor the dispersal behavior of their offspring. This is consistent with earlier results, which demonstrated that corticosterone levels decline after a few days of captivity (Dauphin-Villemant and Xavier, 1987) in this species. Corticosterone itself did not affect female mortality, clutch hatching success, clutch size, or clutch sex-ratio. This also confirms earlier results showing that, except for effects on juvenile morphology, corticosterone has little effect on female reproductive success (Meylan et al., 2004).

In our study, the timing of hormonal treatment affected juvenile morphology, in that exposure to corticosterone early in development resulted in smaller and lighter offspring. Furthermore, natal dispersal was influenced by the duration of corticosterone exposure, depending on clutch sex-ratio. Offspring of briefly exposed mothers dispersed more when the proportion of females in the clutch increased, whereas offspring of chronically exposed mothers tended to disperse more if the proportion of females in the clutch was low, although the latter trend was not significant.

Corticosterone and juvenile morphology

The negative effects of early developmental exposure to corticosterone may be a physiological constraint or an adaptive response to stress. The effects of embryonic exposure to glucocorticoids are usually interpreted as detrimental (Romero, 2004; Saino et al., 2005; Sapolsky et al., 2000; Wingfield and Ramenofsky, 1999). However, in most cases, these negative effects are consequences of chronic stress (Kitaysky et al., 2003; Rubolini et al., 2005; Sapolsky et al., 2000). In our study, these effects were not associated with chronic stress (long corticosterone treatment) but with acute, daily corticosterone elevation (short corticosterone treatment), and were reversible, as juveniles from mothers treated chronically with corticosterone were not morphologically different from juveniles from control mothers. Therefore, exposure to corticosterone late in development compensated for the negative effects of an early exposure. If corticosterone exposure early in development has counter-adaptive effects on morphology, such effects also should be present in juveniles from chronically treated mothers. Since those juveniles do not differ from juveniles from control mothers, the effects of early corticosterone exposure on juvenile morphology are more likely to be a part of an adaptive developmental strategy in response to specific conditions. This effect on morphology has been shown not to decrease individual fitness since juveniles from corticosterone-treated mothers, although smaller and with a lower body condition, did not have lower survival than juveniles from control females, and survival was even increased for juvenile males (Meylan and Clobert, 2005).

A large size does not always confer a competitive advantage, and a small size can be selected for in some environments (Lorenzon et al., 2001); for instance, it may decrease predation or metabolic rate (Groothuis et al., 2005; Hayward and Wingfield, 2004). In our experiment, small juveniles were produced when maternal corticosterone was elevated at least

several weeks before parturition. The administration of corticosterone later on has what is tantamount to a stimulatory effect on offspring size. If a small size is an adaptive response to specific conditions, then it should correlate with some environmental cues acting during this temporal window in embryonic development, but not at other times. Also note that small offspring body size is not a response to a chronic degradation of female internal condition since we should have observed an even greater reduction in size for offspring of females exposed to the longest duration of hormonal treatment. Therefore, the timing of corticosterone administration appears to be part of the mechanism by which information about the external environment is conveyed through maternal effects.

Corticosterone and dispersal

In our experiment, offspring dispersal was not affected by corticosterone exposure *per* se, but by the duration of exposure, in interaction with clutch sex-ratio. Although clutch sex-ratio in the control groups had no effect on offspring dispersal ($\chi^2_1=0.02$, $p=0.89$, $n=187$), it seemed to affect offspring dispersal within the context of maternal stress, with opposite effects, depending on the duration of the stress signal. Therefore, the duration of corticosterone exposure is important in shaping the dispersal response that we have previously observed (De Fraipont et al., 2000; Meylan et al., 2002, 2004). Natal dispersal in the common lizard was previously shown to respond to kin competition (Le Galliard et al., 2003; Léna et al., 1998; Meylan et al., 2004; Ronce et al., 1998) and, in particular, to mother–daughter competition (Le Galliard et al., 2003; Ronce et al., 1998). Indeed, the strong response of juvenile dispersal to clutch sex-ratio we found in the brief corticosterone exposure treatment group can be explained by kin competition theory (Lambin et al., 2001). Dispersal behavior strongly increased when the number of females in the clutch increased. If brief corticosterone exposure signals the female's external environment, as proposed above, then the mother–daughter conflict should have intensified as the number of daughters increased, and this was indicated by the rise in dispersal rate. This response might even be exacerbated if the brief corticosterone exposure treatment is characteristic of environmental degradation.

On the other hand, we predicted that females in poor health should produce philopatric individuals. Only chronic corticosterone elevation, indicating poor female body condition (senescence, Ronce et al., 1998; parasitic load, Sorci et al., 1994) and, therefore, poor survival prospects, was expected to reduce offspring dispersal, although a direct relationship between indication increased corticosterone titers and poor health is still to be demonstrated in the common lizard. Our results are consistent with such a relationship, albeit in interaction with clutch sex-ratio. When the mother's probability of survival is low, the best dispersal strategy for females offspring is philopatry, which is indeed the strategy adopted most often by offspring in female-biased clutches. Females in male-biased clutches may be of lower quality (e.g., "masculinized" phenotype, Uller and Olsson, 2003; lower fecundity, Uller et al., 2004), and forced to adopt the non-optimal strategy to limit their costs of competition with better quality individuals. Consequently, physiological information about the female's internal and external environment could be based on the duration of the corticosterone signal. This does not mean that other factors are not involved in this process, but the variation in the signal duration can be sufficient to produce contrasting responses. We propose two interpretations of the differences in natal dispersal rate in response to a short or a long prenatal corticosterone exposure, but things are likely to be more complex. Indeed, a brief increase in corticosterone plasma level might convey different messages, depending on the timing of elevation, resulting in the production of different juvenile phenotypes. Moreover, contrasting information about the female's internal and external environment must be acquired and integrated during development by offspring to produce an optimized strategy in response to the different sources of information. Variations in corticosterone plasma level during gestation are thus likely to convey many different messages depending on the pattern of secretion, and might modify embryonic development in subtle ways in response to many disparate selective forces.

Corticosterone and offspring phenotype

Maternal exposure to corticosterone affects offspring phenotype at several levels (morphology, Meylan et al., 2002, this study; behavior, Belliure et al., 2004; De Fraipont et al., 2000; Meylan et al., 2002, 2004, this study; physiology, Belliure et al., 2004). However, elevated maternal plasma corticosterone levels are not directly transmitted to embryos, as the placenta plays a strong buffering role in hormonal transfers (Painter et al., 2002; Painter and Moore, 2005). Nevertheless, small quantities of corticosterone might have been transmitted to the embryos in response to a strong elevation of maternal plasma levels (Painter et al., 2002). Such a limited transfer may indeed affect embryonic development, as strong organizational effects of corticosterone have been documented (Nelson, 1994; Schwabl, 1996). Moreover, maternal corticosterone has been implicated in the regulation of trans-placental water movements at key stages of embryonic development (Dauphin-Villemant and Xavier, 1986), which are essential to the completion of development (Massot et al., 1992). Indeed, it appears that juvenile morphology and behaviour are strongly modified by an increase in maternal corticosterone levels, and juvenile neurogenesis is also affected when measured right after birth (Y. Voituron, personal communication). Both processes may thus interact in vivo to shape the developing phenotype at different levels, so that variation in quantity, duration and timing of the corticosterone signal in combination with other factors will modulate ontogenetic trajectories to produce phenotypes adapted to their future environment. Additional experiments will be needed to determine precisely which type of information is carried by corticosterone-dependent maternal effects, and what other prenatal physiological factors are involved in determining adaptive dispersal strategies.

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