

How does corticosterone affect parental behaviour and reproductive success? A study of prolactin in black-legged kittiwakes

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Summary

1. An emergency life-history stage is expressed in breeding vertebrates when the immediate survival is threatened by poor energetic conditions (i.e. allostatic overload). This emergency life-history stage shifts energy investment away from reproduction and redirects it toward immediate survival.
2. In birds, this emergency life-history stage is promoted by a release of the stress hormone corticosterone. However, how corticosterone reduces the expression of parental cares remains to be clarified. One hypothesis is that the release of corticosterone may also affect prolactin levels, a pituitary hormone widely involved in regulating parental behaviours.
3. We tested this hypothesis by experimentally increasing corticosterone levels of chick-rearing black-legged kittiwakes (*Rissa tridactyla*) over a 2-day period and by monitoring prolactin levels over an 8-day period. We also investigated whether this hormonal manipulation affected nest attendance, the motivation to come back to the nest after a short-term stress and breeding success.
4. Corticosterone treatment resulted in a significant increase in plasma corticosterone levels during the first 2 days, which returned to pre-treatment values at day 3. This short-term corticosterone increase was accompanied by a 30% decrease in prolactin levels. Prolactin levels were reduced in a progressive and persistent manner and did not return to their initial levels when corticosterone levels returned to pre-treatment levels. Moreover, although corticosterone levels had returned to pre-treatment values, low prolactin levels were associated with a reduced nest attendance and a greater latency to come back to the nest after a short-term stress. This hormonal treatment also significantly reduced breeding success.
5. This experimental treatment strongly supports the idea that the secretion of these two hormones might be mechanistically linked. Thus, we showed that even a relatively short-term increase in corticosterone levels can durably affect plasma prolactin levels. Therefore, the well-established suppressive action of corticosterone on parental behaviour is probably mediated and reinforced through an effect on prolactin levels. This study highlights the need to consider the potential synergistic effects of these two hormones when studying on the hormonal basis of parental decisions.

Key-words: corticosterone, kittiwake, parental care, prolactin, seabird, stress

Introduction

The breeding season is a demanding period because individuals have to share the available energy their own energetic requirements and those of reproduction. During this period, life-

history theory predicts that individuals should optimally allocate the energy and the time available for self-maintenance and reproductive demands to maximize their fitness (Stearns 1992). Accordingly, allocation decisions can be dramatically influenced by several parameters which affect the fitness yield of parental effort, such as environmental conditions (food availability: Weimerskirch *et al.* 2001; predation risk: Fontaine & Martin 2006; climatic conditions: Fisher *et al.* 2004) or parent's energetic state (McNamara & Houston 1996). Thus,

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an emergency life-history stage is expressed in breeding vertebrates when environmental conditions dramatically deteriorate or the parent's energetic state reaches a low threshold. This emergency life-history stage redirects the individual away from the breeding stage so that it can cope with the perturbation and survive in the best condition possible (Wingfield *et al.* 1998; Wingfield & Kitaysky 2002; Wingfield 2003).

At the proximate level, the activation of this emergency life-history stage is known to be mediated by several neuroendocrine mechanisms (Wingfield *et al.* 1998; Wingfield & Kitaysky 2002; Wingfield 2003). Specifically, suppression or reduction of parental effort has been shown to be associated with the activation of the hypothalamo-pituitary-adrenal (HPA) axis and by the resulting secretion of glucocorticosteroid hormones (reviewed by Landys *et al.* 2006). For instance, elevated corticosterone levels have been reported to result in reduced brood provisioning and even to trigger nest desertion in bird parents (Silverin 1986; Wingfield & Kitaysky 2002; Love *et al.* 2004). However, exactly how elevated corticosterone levels may trigger reduced brood provisioning and abandonment of the nest remains to be clarified (Wingfield & Kitaysky 2002).

One hypothesis is that corticosterone affects other endocrine mechanisms which mechanistically govern the expression of parental cares. In this context, the pituitary hormone prolactin is of particular interest. First, this hormone is involved in the initiation and maintenance of avian parental (reviewed by Buntin 1996, Sockman *et al.* 2006) and alloparental behaviour (Vleck *et al.* 1991; Schoech *et al.* 1996; Khan *et al.* 2001, Angelier *et al.* 2006). Thus, elevated prolactin levels enhance the expression of incubation behaviour, nest provisioning, nest defence and brood guarding (Buntin *et al.* 1991; Wang & Buntin 1999). Second, recent studies have shown that circulating plasma prolactin levels decrease in response to acute stress in breeding birds (Maney *et al.* 1999; Chastel *et al.* 2005; Angelier *et al.* 2007a; Verreault *et al.* 2008), and that, as for the extensively studied corticosterone, the magnitude of the prolactin response to stress can be modulated according to parental effort (Chastel *et al.* 2005; Angelier *et al.* 2007a). Moreover, several studies support the idea that prolactin secretion is also affected by prolonged stress (i.e. type I allostatic overload, McEwen & Wingfield 2003) and energetic constraints (e.g. poor environmental conditions or long-term fasting, Cherel *et al.* 1994; Delehanty *et al.* 1997; Criscuolo *et al.* 2002; Groscolas *et al.* 2008). Because a reduction or suppression of parental investment is accompanied by an increase in corticosterone levels and a decrease in prolactin levels, the secretion of these two hormones might be mechanistically linked.

However, mechanistic relationships between these two hormones appear complex since correlations between baseline or stress-induced corticosterone and prolactin levels have been reported in some cases (Chastel *et al.* 2005; Angelier *et al.* 2007b, 2009) whereas prolactin and corticosterone appeared uncoupled or stage/condition-related in other studies (Chastel *et al.* 2005; Criscuolo *et al.* 2006; Angelier



Fig. 1. A black-legged kittiwake parent caring for its two nestlings at a breeding colony in Kongsfjord, Svalbard. Photo by Céline-Clément Chastel[†].

et al. 2007a; Groscolas *et al.* 2008). The mechanistic relationship between these two hormones is still not clearly understood in breeding wild birds. Thus, experimental examination of the influence of elevated corticosterone levels on prolactin levels in free-living birds is necessary.

In this study, we experimentally test the hypothesis that the maintenance of elevated corticosterone over a prolonged period can affect prolactin levels, and consequently the expression of parental behaviour in a free-living bird species, the black-legged kittiwake (*Rissa tridactyla*, Fig. 1). We experimentally elevated corticosterone levels over a 2-day period to mirror prolonged energy constraints (type I allostatic overload, McEwen & Wingfield 2003) and the activation of an emergency life-history stage (Wingfield *et al.* 1998). Then, we monitored how it affected (i) prolactin levels, (ii) nest attendance, and (iii) breeding success. First, we examined whether our manipulation of corticosterone levels affected circulating prolactin levels. If elevated corticosterone levels have a direct inhibitory effect on prolactin synthesis, secretion or metabolism, we predict that prolactin levels will be reduced in corticosterone-implanted birds. Moreover, we aimed to examine whether prolactin levels return to pre-treatment levels when the allostatic overload ends, that is, when corticosterone levels return to pre-treatment levels. In addition to neuroendocrine aspects, prolactin levels are also likely to be regulated by a behavioural component since tactile and visual stimuli from the nest, the egg(s) or the offspring(s) are known to increase prolactin secretion in birds (Sharp *et al.* 1988; Book 1991; El Halawani *et al.* 1980). If prolactin secretion is only and directly influenced by elevated corticosterone levels, prolactin levels should return to pre-treatment levels when corticosterone levels have returned to pre-treatment levels. However, prolactin levels could remain low during a prolonged period if nest attendance and thus exposure to stimuli have been reduced when circulating corticosterone levels were experimentally increased.

Second, we examined whether this experimental increase of corticosterone levels affects the time devoted to guarding the

brood in the nest. Although increased corticosterone levels have been shown to reduce nest attendance in some specific cases (Silverin 1986; Kitaysky *et al.* 2001; Wingfield & Kitaysky 2002; Angelier *et al.* 2007c), previous studies have brought evidence that reduced nest attendance may mechanistically result from decreased prolactin levels (Buntin *et al.* 1991; Wang & Buntin 1999). If our experimental increase of corticosterone levels reduces prolactin levels in kittiwake parents, we predict that corticosterone-implanted birds will spend less time guarding the brood in the nest. Specifically, we predict that nest attendance will be reduced when prolactin levels are low, that is potentially even when corticosterone levels have returned to pre-treatment levels.

Finally, we tested whether this experimental increase of corticosterone levels affects breeding success. According to previous studies (reviewed by Landys *et al.* 2006), we predict that corticosterone-implanted birds will have a reduced breeding success. By examining the relationships between corticosterone, prolactin and reproductive behaviour, we then discuss how corticosterone can affect reproductive performance and parental investment.

Materials and methods

STUDY AREA AND BIRDS

Our study was conducted between 23 July and 7 August 2005 in a colony of black-legged kittiwakes (mid chick-rearing period to late chick-rearing period) at Kongsfjorden, Svalbard, Norway (78°54'N, 12°13' E). Black-legged kittiwakes are colonial seabirds that breed on cliffs throughout the northern parts of the Pacific and Atlantic, including the Barents Sea region up to the Svalbard Archipelago (Fig. 1). Although females expend slightly more energy than males during the chick-rearing period (Jodice *et al.* 2006), chick-rearing roles and hormonal levels (corticosterone and prolactin) are largely similar between males and females in kittiwakes (Kitaysky *et al.* 1999; Moe *et al.* 2002; Chastel *et al.* 2005).

EXPERIMENTAL MANIPULATION OF CORTICOSTERONE LEVELS

In the colony, 54 parents were captured at their nest with a noose at the end of a 5-m fishing rod. Immediately after capture, a blood sample was collected within 3 min to determine baseline corticosterone and prolactin levels ($n = 47$, in 7 birds blood sampling was difficult and handling time exceeded the one recommended to assess baseline corticosterone and prolactin levels, Romero & Reed 2005; Chastel *et al.* 2005). Blood samples were collected from the alar vein with a 1-ml heparinized syringe and a 25-gauge needle. All birds were then weighed to the nearest 2 g using a Pesola spring balance and their skull length (head + bill) was measured to the nearest 0.5 mm. Immediately after taking these measurements, each bird was subcutaneously implanted between the shoulders with two 25 mm silastic tubes (internal diameter 1.47 mm, external diameter 1.96 mm, Dow Corning, Michigan) either filled with crystallized corticosterone (C2505, Sigma Chemical Co., St. Louis, MO, $n = 31$ corticosterone-implanted birds) or empty ($n = 23$ controls). Knowing that corticosterone cannot diffuse through silastic, we cut both ends of the silastic tube to allow rapid release of CORT over a limited period. Our aim

was to increase plasma corticosterone levels significantly but temporarily (2 days, see Angelier *et al.* 2007c) and within physiological range (stress-induced levels, Kitaysky *et al.* 1999; Chastel *et al.* 2005; Angelier *et al.* 2007d). We used body mass and the skull length to calculate an index of body condition at day 0, that is, at the time of implantation (thereafter called 'initial body condition'). Because residuals from an ordinary least squares linear regression of body mass against body size can lead to spurious results (Green 2001), we calculated residual indices of condition by using the residuals from a reduced major axis regression of body mass against skull length (Green 2001).

SUBSEQUENT BLOOD SAMPLES

Birds were then recaptured opportunistically when they were present at the colony. Specifically, birds were recaptured in 1, 2, 3 and 8 days after the implantation (sample sizes were 16, 13, 30 and 19, respectively). Days of sampling were selected according to previous studies conducted on this species: corticosterone levels were increased 2 days after corticosterone implantation, and then, they returned to pre-implantation levels (Kitaysky *et al.* 2001; Angelier *et al.* 2007c). Thus, birds were sampled at days 1 and 2 to check that our corticosterone manipulation was effective, and they were then sampled at days 3 and 8 to check that corticosterone levels returned to pre-implantation levels. Immediately after capture, kittiwake parents were blood sampled as described above. In a few cases, the amount of plasma available was not sufficient to assay both corticosterone and prolactin. Consequently, baseline prolactin levels at 1, 2, 3 and 8 days after the implantation were available for 13, 12, 29 and 17 birds, respectively.

NEST ATTENDANCE AND BREEDING SUCCESS

At the time of implantation, birds were individually marked with white PVC plastic bands engraved with a unique three-digit code and fitted on the bird's tarsus, for identification from a distance. They were also marked with a spot painted on their head to facilitate individual identification. In order to monitor the nest attendance of a sub-sample of kittiwakes ($n = 36$, 18 corticosterone-implanted kittiwakes and 18 controls), their presence at the nest was observed using binoculars twice a day during an 8-day period following the implantation by. The first observation of the nest was conducted 12 h after the implantation, and the time elapsed between two subsequent observations was approximately 12 h. Nest attendance was then calculated for two periods that were selected according to the expected differences in corticosterone levels between the corticosterone-implanted birds and the controls (period A: from 0.5 to 2 days after implantation; period B: from 3 to 8 days after implantation; see Results section; Fig. 2). Previous studies have shown that corticosterone levels are elevated in the 2 days following corticosterone implantation (period A) and then return to pre-implantation levels (period B, Angelier *et al.* 2007c). These two periods will allow testing of how prolactin levels and nest attendance are affected by an experimental increase of corticosterone levels. Two birds lost their last chick during period B. Consequently, their nest attendance during period B was estimated from day 3 after implantation to the last day of the chick's presence in the nest. In a previous study, we recorded nest attendance in detail over a 2-day period by using activity loggers (Angelier *et al.* 2007c), and we also monitored the presence of these equipped birds at the nest twice a day by visual observations of the nest. These two measurements of nest attendance were highly correlated ($n = 22$, $\chi^2 = 7.29$, d.f. = 1, $P = 0.007$, $r = 0.56$) demonstrating

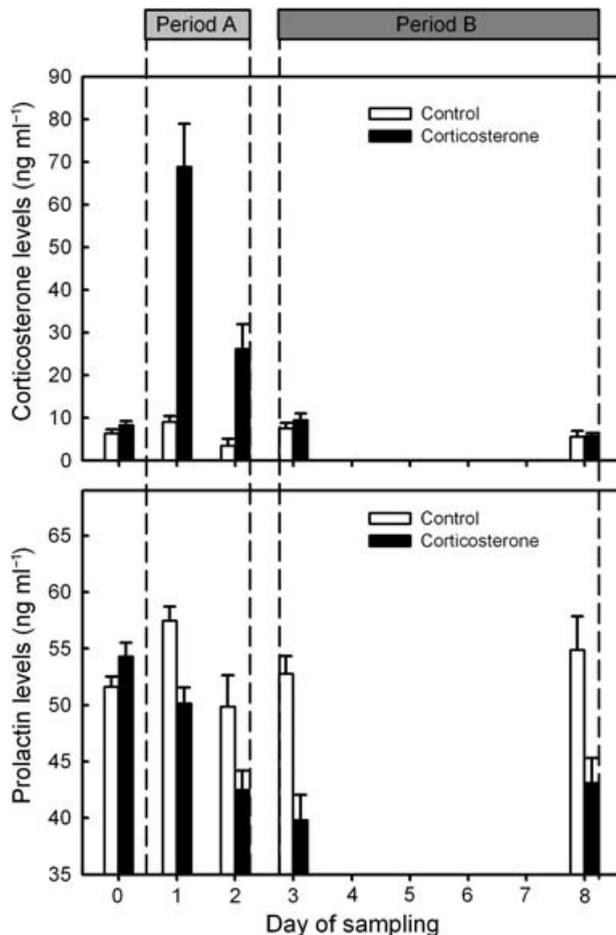


Fig. 2. Effects of corticosterone implants on baseline corticosterone and prolactin levels in black-legged kittiwakes (mean \pm SE). Corticosterone levels of corticosterone-implanted birds were significantly increased during the period A (day 1 and day 2), but returned to low initial levels during the period B (day 3 and day 8). Prolactin levels of corticosterone implanted birds were significantly reduced during both periods (day 1, day 2, day 3 and day 8). Corticosterone and prolactin levels of controls did not vary over the study period.

that two visual observations of the nest per day provides a reliable estimate of nest attendance.

Moreover, we monitored the willingness of birds to return quickly to their nest and to resume parental activities after a disturbance (capture and handling). We monitored this behaviour in a sub-sample of the birds captured and blood sampled for hormone levels at day 3 ($n = 11$, 6 corticosterone-implanted kittiwakes and 5 controls). We chose to monitor this behaviour at day 3 during period B when corticosterone levels had returned to pre-treatment levels (Angelier *et al.* 2007c), to test whether our corticosterone manipulation had a long-term effect on the willingness of birds to resume parental activities after a disturbance. After capture and sampling procedures, birds were released in the vicinity of the colony, and we measured the time duration (in minutes) between their release and their return to the nest (called hereafter 'absence duration').

The number of chicks in the nest was checked at the time of implantation (day 0) and then two and eight days later (days 2 and 8). This was done by using a mirror at the end of a 4 m fishing rod. We were able to determine whether a bird lost its chick(s) during

period A and again during period B. However, a few nests were not easily accessible, and their number of chicks was, therefore, not known with certainty ($n = 12$). They were excluded from the analysis of the probability of losing a chick (final sample size, $n = 42$).

HORMONE ASSAYS

Blood samples were centrifuged, the plasma was decanted, and then both blood and plasma were stored at -20°C before analysis. All laboratory analyses were performed at the Centre d'Etudes Biologiques de Chizé (CEBC). DNA was extracted from the red blood cells and then the sex of the individuals was determined through PCR amplification of the CHD gene following standard procedures (Fridolfsson & Ellegren 1999). Plasma concentrations of corticosterone were determined following methods described in Lormée *et al.* (2003). Since blood samples were collected within 3 min of capture, the corticosterone and prolactin levels were considered to reflect baseline levels (Romero & Reed 2005; Chastel *et al.* 2005). Concentrations of prolactin were determined with the remaining plasma by a heterologous radioimmunoassay as detailed and validated for this species (Chastel *et al.* 2005). The intra-assay variations were 7.7% for corticosterone and 8.0% for prolactin. The minimal detectable corticosterone and prolactin levels were 0.5 and 6 ng mL^{-1} , respectively, and no samples fell below these limits.

STATISTICAL ANALYSES

All analyses were performed with SAS statistical software (SAS Institute Inc., 2000). We used Generalized Linear Models (GLM), Generalized Linear Mixed Models (GLMM) and maximum likelihood technique (Burnham & Anderson 2002). Because step-down approach (backward elimination) can lead to biased and spurious statistical results (Whittingham *et al.* 2006), we fit the full models that include all effects (Burnham & Anderson 2002).

First, we used GLMMs to examine the effect of corticosterone implants on circulating corticosterone and prolactin levels. Because kittiwakes were sampled several times, we included bird identity as a random factor to control for inter-bird variation. We tested an effect of 'treatment' (corticosterone-implanted birds vs. controls), 'day' (day of sample: day 0, day 1, day 2, day 3 and day 8) and their interaction on baseline corticosterone and prolactin levels. Similarly, we tested then an effect of 'treatment', 'period' (period of sample: day 0, period A and period B) and their interaction on baseline corticosterone and prolactin levels to test whether hormone levels differed between day 0, period A and period B.

Second, we used GLMMs to examine the influence of corticosterone implants on nest attendance. Because nest attendance of kittiwake parents was monitored during two subsequent periods, we included bird identity as a random factor to control for inter-bird variation. We tested an effect of 'treatment', 'period' (period A vs. period B), the 'initial body condition' variable (body condition at day 0, i.e. the time of implantation) and their interactions on our measurement of nest attendance. Initial body condition was included in the model as an explanatory variable because previous studies have shown that body condition can influence nest attendance in this species (Moe *et al.* 2002; Angelier *et al.* 2007c). Moreover, we also tested an effect of 'treatment' on the 'absence duration' measured at day 3 by using a Kruskal–Wallis test.

Finally, we aimed to determine whether the experimental treatment affected the probability of losing a chick during both periods. However, we were not able to test this potential effect of treatment

Table 1. Model selection and GLMM to test the influence of either (A) day of sampling and treatment ('corticosterone' vs. 'control') or (B) period of sampling ('period A' vs. 'period B') and treatment on corticosterone levels and prolactin levels. Bird identity was included as a random factor in the models

	Dependent variable	Independent variables	d.f.	F	P
A	Corticosterone (ng mL ⁻¹) (n = 125)	Day	4,63	20.17	< 0.001
		Treatment	1,63	31.63	< 0.001
		Day × Treatment	4,63	8.63	< 0.001
	Prolactin (ng mL ⁻¹) (n = 119)	Day	4,57	7.98	< 0.001
		Treatment	1,57	29.38	< 0.001
		Day × Treatment	4,57	10.69	< 0.001
B	Corticosterone (ng mL ⁻¹) (n = 125)	Period	2,67	39.08	< 0.001
		Treatment	1,67	29.76	< 0.001
		Period × Treatment	2,67	17.09	< 0.001
	Prolactin (ng mL ⁻¹) (n = 119)	Period	2,61	11.30	< 0.001
		Treatment	1,61	17.43	< 0.001
		Period × Treatment	2,61	19.75	< 0.001

on this probability during each period because environmental conditions were very good in 2005 and loss of a chick was a very rare event (Moe *et al.* unpublished data). Thus, only 10 chicks died over the whole study period. Because of this small sample size, we used GLMs to examine the influence of corticosterone implants on the probability of loosing a chick during the whole period (distribution: binomial, link function: logit). Thus, we tested an effect of 'treatment' and 'initial body condition' on the probability of loosing a chick over the entire period (from day 0 to day 8).

Results

At the time of implantation (day 0), corticosterone-implanted birds and controls were similar in body condition (Student's *t*-tests, $t = 0.10$, d.f. = 52, $P = 0.919$), baseline corticosterone levels (Student's *t*-tests, $t = -1.40$, d.f. = 45, $P = 0.168$), baseline prolactin levels (Student's *t*-tests, $t = -1.70$, d.f. = 45, $P = 0.096$), date of treatment (Student's *t*-tests, $t = 0.87$, d.f. = 52, $P = 0.388$), sex-ratio ($\chi^2 = 1.19$, d.f. = 1, $P = 0.276$) and brood size ($\chi^2 = 0.28$, d.f. = 1, $P = 0.594$).

EFFECT OF TREATMENT ON CORTICOSTERONE AND PROLACTIN LEVELS

We found a significant effect of treatment, day of sampling and their interaction on baseline corticosterone and prolactin levels (Table 1a). Corticosterone and prolactin levels were not affected by the day of sampling within the control group (corticosterone: $F_{4,28} = 0.81$, $P = 0.532$; prolactin: $F_{4,25} = 1.77$, $P = 0.166$; Fig. 2). In comparison to controls, baseline corticosterone levels of corticosterone-implanted birds were significantly increased at day 1 and day 2 (contrasts; day 1: $F_{1,63} = 25.88$, $P < 0.001$; day 2: $F_{1,63} = 5.78$, $P = 0.019$; Fig. 2), but returned to pre-treatment values from day 3 to day 8 (contrasts; day 3: $F_{1,63} < 0.01$, $P = 0.973$; day 8: $F_{1,63} = 0.35$, $P = 0.554$; Fig. 2). In comparison to controls, baseline prolactin levels of corticosterone-implanted birds were significantly decreased by the treatment at days 1 and 2 and stayed low from day 3 to day 8 – the period when corticosterone levels had returned to pre-treatment values (contrasts: day 1:

$F_{1,57} = 5.64$, $P = 0.021$; day 2: $F_{1,57} = 8.44$, $P = 0.005$; day 3: $F_{1,57} = 32.87$, $P < 0.001$; day 8: $F_{1,57} = 17.47$, $P < 0.001$; Fig. 2).

HORMONAL DIFFERENCES BETWEEN THE TWO PERIODS

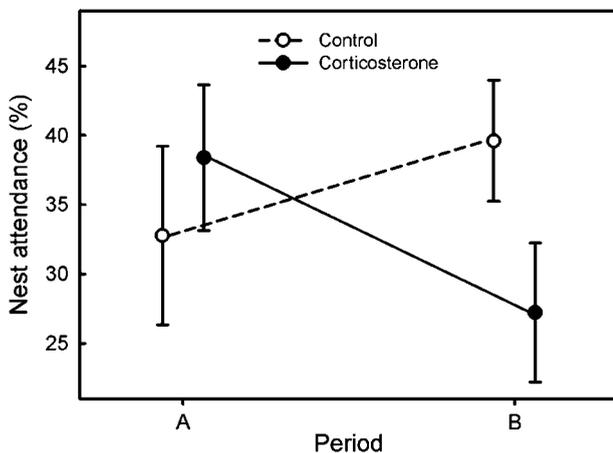
There was a significant effect of treatment, period of sampling and their interaction on baseline corticosterone and prolactin levels (Table 1b). Specifically, estimates of parameters showed that corticosterone levels of corticosterone-implanted birds were higher than those of controls during the period A (d.f. = 67, $t = -7.10$, $P < 0.001$), but were similar to levels of control birds during the period B (d.f. = 67, $t = -1.13$, $P = 0.264$). Prolactin levels of corticosterone-implanted birds were lower than those of controls during both periods (period A: d.f. = 61, $t = 2.67$, $P = 0.016$; period B: d.f. = 61, $t = 6.53$, $P < 0.001$). Within the corticosterone-implanted group, corticosterone and prolactin levels of period A were both higher than those of the period B (corticosterone levels: d.f. = 67, $t = 10.70$, $P < 0.001$; prolactin levels: d.f. = 61, $t = 3.01$, $P = 0.004$). Within the control group, corticosterone and prolactin levels were not affected by the period of sampling (corticosterone levels: d.f. = 67, $t = 1.68$, $P = 0.098$; prolactin levels: d.f. = 61, $t = -0.03$, $P = 0.978$).

NEST ATTENDANCE

We found a significant effect of initial body condition on nest attendance: Birds in poor condition at day 0 spent less time on the nest than birds in good body condition. Although 'period' (period A vs. period B) and 'treatment' *per se* had no influence on nest attendance, we found a significant effect of their interaction 'treatment × period' (Table 2). There was no significant effect of other interactions. Specifically, nest attendance did not significantly vary from period A to period B in control birds, whereas it decreased in corticosterone-implanted kittiwakes (Fig. 3). As a consequence, the nest attendance of corticosterone-implanted birds tended to be

Table 2. Model selection and GLMM to test the influence of period ('period A' vs. 'period B'), treatment ('corticosterone' vs. 'control'), initial body condition (at day 0) on nest attendance. Bird identity was included as a random factor in the models

Dependent variable	Independent variables	d.f.	F	P
Nest attendance (%) ($n = 72$)	Period	1,32	0.50	0.640
	Initial Condition	1,32	12.90	0.007
	Treatment	1,32	0.72	0.463
	Period \times Treatment	1,32	4.23	0.032
	Initial Condition \times Period	1,32	0.03	0.862
	Initial Condition \times Treatment	1,32	0.03	0.861
	Initial Condition \times Period \times Treatment	1,32	< 0.01	0.970

**Fig. 3.** Nest attendance of corticosterone-implanted kittiwakes and controls during periods A and B. Nest attendance did not significantly vary from the period A to the period B in controls, whereas it decreased in corticosterone-implanted kittiwakes. Nest attendance of corticosterone-implanted birds was slightly lower than that of controls during the period B.

lower than that of controls during period B (d.f. = 34, $t = 2.01$, $P = 0.052$; Fig. 3), whereas the nest attendance of corticosterone-implanted birds and controls was similar during period A (d.f. = 34, $t = -0.88$, $P = 0.384$; Fig. 3). When captured at day 3 (beginning of period B), corticosterone-implanted kittiwakes returned to their nest significantly later after release than controls (Kruskal–Wallis test, $\chi^2 = 4.11$, $P = 0.043$; absence duration in minutes, Mean \pm SE: corticosterone-implanted birds, 222.17 ± 59.43 ; controls, 64.80 ± 60.6).

BREEDING SUCCESS

The probability of losing a chick from implantation (day 0) to day 8 (i.e. period A + B) was significantly affected by the treatment (GLM, d.f. = 1, $\chi^2 = 5.77$, $P = 0.016$), but not by initial body condition (GLM, d.f. = 1, $\chi^2 < 0.01$, $P = 0.951$): Corticosterone-implanted birds had a higher probability of losing a chick (33.33%) than controls (5%).

Discussion

In this study of black-legged kittiwakes caring for young, we showed that a 2-days experimental increase in plasma corti-

costerone levels was accompanied by a reduction of plasma prolactin and subsequently by a reduction of nest attendance. Moreover, we showed that prolactin levels did not return to their pre-treatment levels when corticosterone levels have returned to pre-treatment levels, suggesting therefore that corticosterone treatment can affect prolactin secretion in a durable manner. Finally, we showed for the first time that experimentally-induced low prolactin levels were associated with reduced nest attendance (at least during the period B) and ultimately with a lower breeding success.

CORTICOSTERONE TREATMENT AND PROLACTIN SECRETION

In this study, we used corticosterone implants to experimentally increase corticosterone levels of chick-rearing black-legged kittiwakes (i.e. a method already validated in this species, Kitaysky *et al.* 2001; Angelier *et al.* 2007c). Thus, circulating corticosterone levels were increased over the period A (mean: 52.29 ng mL^{-1}) within the physiological range observed in black-legged kittiwakes during a stress response ($50\text{--}100 \text{ ng mL}^{-1}$, Kitaysky *et al.* 1999; Chastel *et al.* 2005; Angelier *et al.* 2007d). By using this experimental corticosterone increase, we found that the maintenance of elevated corticosterone levels over a 2-day period was paralleled by a significant and durable decrease in prolactin levels. This experimental treatment strongly supports the idea that the secretion of these two hormones might be mechanistically linked as previously suggested by correlative observations: an increase in corticosterone levels induced by acute or prolonged stress is also associated with depressed prolactin levels in birds (Maney *et al.* 1999; Criscuolo *et al.* 2002; Chastel *et al.* 2005; Angelier *et al.* 2007a; Groscolas *et al.* 2008; Verreault *et al.* 2008).

Interestingly, we found that the corticosterone-induced decrease of prolactin levels is progressive in kittiwakes: prolactin levels gradually decreased over the period A (from day 0 to day 2). Moreover, this corticosterone-induced decrease in prolactin levels is important (approximately 30% after 2 days). In comparison, a short-term acute stress only slightly reduces prolactin levels in chick-rearing kittiwakes, (approximately 9% after a 30-min stress protocol, Chastel *et al.* 2005). In addition, prolactin levels remained low from day 3 to day 8 (period B) even after corticosterone levels returned to pre-treatment levels, whereas it is known that prolactin levels return quickly to elevated baseline levels after

a short-term acute stress (Chastel *et al.* unpublished results). These results suggest that the maintenance of elevated corticosterone levels during two days affects prolactin physiology in an important, progressive and durable manner.

How did our corticosterone treatment mechanistically affect prolactin levels? Prolactin secretion might have been directly inhibited by corticosterone administration (Horváth *et al.* 2001). On the other hand, a decrease in tactile/visual stimuli from eggs or hatchlings is known to decrease prolactin secretion (Buntin 1996). Prolactin secretion may have thus declined indirectly in response to a lower commitment in brooding activities caused by corticosterone administration (Kitaysky *et al.* 2001). During the period A, the effect of our manipulation on prolactin levels might have resulted from a direct inhibitory effect of corticosterone on prolactin secretion. Indeed, exposure to visual and tactile stimuli from nest or chicks is probably not the main factor explaining the difference in prolactin levels between experimental birds and controls since nest attendance was equivalent between these two groups during the period A. In vertebrates, the mode of inhibitory action of corticosterone on prolactin secretion remains ill defined (reviewed in Freeman *et al.* 2000). Some experimental studies have shown that prolactin levels increase after adrenalectomy in captive rodents (Bánky *et al.* 1994) and that this effect can be reversed by administration of corticosterone (Brann *et al.* 1990). Recent studies have suggested that the inhibitory effect of corticosterone on prolactin secretion could be mediated through the actions of corticosterone on dopamine physiology (Horváth *et al.* 2001), but evidence is still lacking. In that respect, future laboratory experiments should aim to investigate the influence of corticosterone on different regulators of prolactin secretion, such as dopamine (El Halawani *et al.* 1991), vasointestinal peptide (El Halawani *et al.* 1990; Vleck & Patrick 1999; Christensen & Vleck 2008) and lactotroph physiology (Ramesh *et al.* 1996). During the period B, prolactin levels could have remained low as a result of a durable effect of corticosterone on prolactin synthesis, secretion or metabolism, but also as a result of the behavioural consequences of our corticosterone manipulation. Because prolactin levels decrease when stimuli from the nest or the chick are removed (El Halawani *et al.* 1980; Hall 1987; Book 1991), the decrease in nest attendance of corticosterone-implanted birds during the period B may explain that prolactin levels remained low even when corticosterone levels returned to pre-treatment levels. To disentangle these two hypotheses, future studies should experimentally examine the influence of prolactin on nest attendance without manipulating corticosterone levels (through VIP immunization or drug administration, Sharp *et al.* 1998; Angelier *et al.* 2006).

CORTICOSTERONE TREATMENT, PROLACTIN AND PARENTAL BEHAVIOUR

In this study, our experimental manipulation of corticosterone levels resulted in a significantly reduced expression of parental behaviour (nest attendance and motivation to return to the nest after an acute stress, i.e. capture and handling).

Thus, our results confirm previous findings that elevated corticosterone levels can reduce, or even disrupt, parental behaviour in vertebrates (Silverin 1986; Wingfield & Kitaysky 2002; Love *et al.* 2004). However, the expression of parental behaviour seems also to depend on prolactin levels, supporting therefore the hypothesis that corticosterone alone does not reduce the expression of parental behaviour in birds (Criscuolo *et al.* 2005; Angelier *et al.* 2007a). Indeed, during period B (from day 3 to day 8), corticosterone-implanted birds showed a lower nest attendance than that of controls even though corticosterone levels had returned to low pre-treatment levels. In contrast to corticosterone levels, prolactin levels of corticosterone-implanted birds were much lower than those of controls during this period (mean; controls: 53.50 ng mL⁻¹; corticosterone-implanted birds: 41.04 ng mL⁻¹) suggesting, therefore, that prolactin is involved in the regulation of nest attendance in birds. Supporting this effect of prolactin on nest attendance, several studies have reported positive correlations between prolactin levels and the expression of parental cares at the individual level (Sharp *et al.* 1988; Vleck *et al.* 1991; Khan *et al.* 2001; Deviche & Sharp 2001; Van Roo, Ketterson & Sharp 2003; O'Dwyer *et al.* 2006; Angelier *et al.* 2007a). For instance, prolactin levels are positively correlated with the nestling provisioning rate by parents in the house finch, *Carpodacus mexicanus* (Duckworth *et al.* 2003). Also, Wang & Buntin (1999) have demonstrated that prolactin stimulates brooding and protecting behaviour in captive ring doves (*Streptopelia risoria*). In addition, we found that corticosterone-implanted kittiwakes were less likely to quickly resume parental cares after a capture/release protocol than controls (at day 3). At day 3, corticosterone levels of corticosterone-implanted birds were similar to those of controls whereas their prolactin levels were significantly lower than those of controls. Therefore, this result supports the idea that control birds with elevated prolactin levels were more motivated to come back to the nest after a temporary stressful event than experimental birds with low prolactin levels.

Surprisingly, we found no difference in nest attendance between corticosterone-implanted parents and controls during period A even though prolactin levels of experimental birds were decreasing during this period. What could explain this result? Indeed, the effect of corticosterone implantation on prolactin levels was slow and progressive and prolactin levels of corticosterone-implanted birds were significantly but not dramatically lower than those of controls during period A (mean; controls: 53.85 ng mL⁻¹; corticosterone-implanted birds: 47.02 ng mL⁻¹). This slight decrease in prolactin levels may not have been sufficient to induce detectable behavioural effects, and this could explain the absence of differences in nest attendance between corticosterone-implanted parents and controls. Supporting this interpretation, there is growing evidence that the expression of parental behaviours is only reduced when prolactin levels reach a low threshold (reviewed in Sockman *et al.* 2006; Angelier *et al.* 2006, 2007a).

Thus, our results suggest that the well-established negative effects of corticosterone treatment on the expression of parental care (Silverin 1986; Kitaysky *et al.* 2001; Wing-

field & Kitaysky 2002) is probably mediated and reinforced through an effect on prolactin levels (Chastel *et al.* 2005; Criscuolo *et al.* 2005; Angelier *et al.* 2007a; Groscolas *et al.* 2008).

HORMONAL ORCHESTRATION OF PARENTAL DECISIONS

In vertebrates, the maintenance of elevated corticosterone levels over a prolonged period is clearly associated with an allostatic overload (McEwen & Wingfield 2003). Indeed, elevated corticosterone levels result from a negative energy balance: energy demands exceed the energy income and what can be mobilized from stores (McEwen & Wingfield 2003). This corticosterone response promotes the mobilization of protein energy resources and the occurrence of behavioural and physiological adjustments that aim to restore energetic balance (i.e. the activation of an emergency life-history stage, Wingfield *et al.* 1998). Here, we showed that increased corticosterone secretion can have long-term effects on another hormonal mechanism involved in the regulation of parental behaviour – prolactin secretion. Moreover, we also found that corticosterone-implanted birds had lower breeding success than controls. Loss of chicks in corticosterone-implanted kittiwakes probably results from a high predation by glaucous gulls (*Larus hyperboreus*) in poorly attended broods. Because low prolactin levels are associated with poor parental cares and can ultimately stimulate nest desertion (Cherel *et al.* 1994; Buntin 1996; Chastel & Lormée 2002; Groscolas *et al.* 2008), our study may explain how corticosterone can mechanistically compromise the current reproductive episode by redirecting resources away from reproduction (Wingfield & Sapolsky 2003). Therefore, a direct or indirect action of increased corticosterone levels on prolactin secretion is probably an important hormonal mechanism involved in the decision to reduce parental cares when a parent's survival is threatened.

At the ultimate level, this sensitivity of prolactin secretion to corticosterone is probably adaptive. When facing an allostatic overload, life-history theory predicts that individuals should adjust their parental decisions to this poor energetic situation to maximize their fitness (Stearns 1992). Because kittiwakes are long-lived birds with a high residual reproductive value, they should not jeopardize their survival in such a situation, but instead decrease their parental effort and redirect the available energy towards self-maintenance (Drent & Dann 1980; Erikstad *et al.* 1998; Angelier *et al.* 2007c). Because low prolactin levels are associated with reduced parental cares (Buntin 1996), our results support this theory. Therefore, we suggest that both corticosterone and prolactin can play a major role in the orchestration of parental effort in birds (Chastel *et al.* 2005; Sockman *et al.* 2006; Angelier *et al.* 2007a; Groscolas *et al.* 2008). Indeed, short-term increases in corticosterone levels (2 days) may have acted as an emergency signal tuning individuals to a low parental cares strategy, and this was probably achieved by a corticosterone-induced reduction in prolactin secretion. This study highlights the need to

consider the potential synergistic effects of corticosterone and prolactin when focusing on the hormonal basis of parental decisions.

Interestingly, the influence of elevated corticosterone levels on prolactin secretion appears to be persistent since prolactin levels remained low even when corticosterone levels had returned to pre-treatment levels (from day 3 to day 8). Moreover, low prolactin levels were associated with reduced nest attendance and probably with a reduced expression of parental care (Buntin 1996). Do these results indicate that it is adaptive to maintain low parental cares after a temporary allostatic overload in this long-lived species? Indeed, the cost of reproduction can be very important in birds (Cichoń 2000; Hanssen *et al.* 2005). For instance, Golet *et al.* (2004) have demonstrated that raising chicks compromises the future reproductive potential of kittiwake parents by reducing their survival probability and their future fecundity. Moreover, the fitness value of the current reproductive episode can be seriously reduced when the brood has faced a prolonged nutritional deficit (Metcalf & Monaghan 2001); chicks are known to suffer reduced growth, cognitive ability and survival in response to nutritional deficits (Kitaysky 1999; Kitaysky *et al.* 2003, 2006; Pravosudov 2005). In that respect, it is probably adaptive to reduce parental cares after a temporary allostatic overload because intense parental cares would provide low fitness benefits and entail high fitness costs.

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