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Long-term survival effect of corticosterone manipulation in Black-legged kittiwakes

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ABSTRACT

The secretion of corticosterone in response to stress is thought to be an adaptive mechanism, which promotes immediate survival at the expense of current reproduction. However, at the individual level, the hypothesis of a corticosterone-related survival appears to be complex. In this study, we tested this hypothesis by combining for the first time an experimental manipulation of corticosterone levels and capture-mark-recapture (CMR) models. To do so, we increased corticosterone levels of chick-rearing Black-legged kittiwakes (*Rissa tridactyla*) via subcutaneous implants. Then, we monitored the long-term survival of kittiwakes over the 2 consecutive years. Corticosterone-implanted birds showed a significantly lower apparent annual survival than sham-implanted ones (46.9% vs 77.8%). This result is supported by the well-known deleterious effects of elevated corticosterone levels on cognitive and immune functions. Alternately and in the light of recent studies, our experimental manipulation may have down-regulated the endogenous secretion of corticosterone through a prolonged negative feedback. If so, the corticosterone-implanted kittiwakes may have failed to trigger an appropriate stress response during subsequent life-threatening perturbations, hence being unable to adjust their behavior and physiology toward immediate survival. This study highlights the complex long-term consequences of corticosterone manipulation on fitness in free-living vertebrates.

1. Introduction

Reproduction imposes the challenge to allocate an optimal amount of energy and time to the offspring's requirements without jeopardizing long-term survival (life-history theory, Stearns, 1992). This is especially predicted for long-lived species that should behave as "prudent parents" (Drent and Daan, 1980; Stearns, 1992) because their lifetime reproductive success is primarily a function of adult survival (Williams, 1966). At the proximate level, endocrine mechanisms are at the helm of such life history decisions (Wingfield et al., 1998; Ricklefs and Wikelski, 2002). Specifically, glucocorticoid hormones (GC) are released under life-threatening perturbations, such as food shortage, harsh weather or predation risk, and in turn, trigger the adoption of an emergency stage (reviewed in Landys et al., 2006). The behavior and physiology exhibited during this emergency stage enhance the animals' ability to cope with the stressor (Wingfield and Kitaysky, 2002; Wingfield and Sapolsky, 2003) at the expense of non-vital functions such as current reproductive effort (Silverin, 1986; Wingfield and Kitaysky, 2002; Love et al., 2004).

The adrenocortical stress response is thus expected to be an adaptive mechanism, which redirects energy allocation towards immediate survival (Wingfield and Sapolsky, 2003). The stress response may have evolved due to its short-term selective advantages (Wingfield and Sapolsky, 2003; Landys et al., 2006), and occurs despite its adverse long-term effects (reviewed in Sapolsky, 2000). Supporting this, a recent comparative study has shown that long-lived bird species with low fecundity have a stronger acute stress response than short-lived, high fecundity ones (Bókony et al., 2009). However, although stress response has been measured in many species (Romero, 2002; Bókony et al., 2009), few studies have tested this assumption at the individual level and the results are rather variable (reviewed in Breuner et al., 2008). For example, a recent study of American redstarts (*Setophaga ruticilla*) showed that high stress-induced GC were linked to a higher return rate, but this was only observable for birds wintering in low quality habitats (Angelier et al., 2009b). However, other studies showed the opposite pattern. Under stressful events, marine iguanas (*Amblyrhynchus cristatus*) with greater stress-induced GC had a lower survival (Romero and Wikelski, 2001). In white stork (*Ciconia*

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ciconia), nestlings with strong GC reactivity showed a lower return rate (Blas et al., 2007). Song sparrow (*Melospiza melodia*) with greater stress responses were less likely to return to breed the following year (MacDougall-Shackleton et al., 2009). In addition, the hypothesis of GC-related survival appears to be complex because elevated baseline GC levels have also been associated with either low (Kitaysky et al., 2007, in press; Romero and Wikelski, 2002; Brown et al., 2005) or high return rate (Cote et al., 2006; Cabezas et al., 2007). Moreover, it is difficult to draw a general picture on the link between GC and fitness (Breuner et al., 2008; Bonier et al., 2009), since the above studies included measurements of stress responses made in developing chicks (Blas et al., 2007), non-breeding (Cabezas et al., 2007; Angelier et al., 2009b) and breeding adults (Romero and Wikelski, 2001; Brown et al., 2005; MacDougall-Shackleton et al., 2009).

Experimental manipulations of GC levels are thus needed and should be one of the best ways to test the effects of GC on survival. However, the link between manipulated GC levels and subsequent survival has been rarely investigated in free-living vertebrates (Kitaysky et al., 2001; Cote et al., 2006). Furthermore, relating GC levels and survival at the individual levels is rarely achieved because of the need to sample marked individuals from long-term capture-mark-recapture (CMR) studies (Brown et al., 2005). The CMR approach explicitly allows taking into account capture probability and temporary absence from the study area, and thus provides unbiased estimators of adult survival (Lebreton et al., 1992).

In 2005, we manipulated adult Black-legged kittiwakes (*Rissa tridactyla*), a long-lived seabird, with corticosterone implants (Angelier et al., 2007a, 2009a). To do so, we increased plasma corticosterone levels during the chick-rearing phase (Angelier et al., 2007a, 2009a), and within the physiological range observed for this species (Kitaysky et al., 1999b, 2001; Chastel et al., 2005; Angelier et al., 2007a,b, 2009a). Following the hormonal manipulation, corticosterone (hereafter Cort) implanted birds invested poorly into current reproduction (Angelier et al., 2007a, 2009a). The apparent survival rates were estimated using CMR models over the following 2 years (2006 and 2007). Therefore, this experimental manipulation offers the possibility to test for the first time with CMR models whether corticosterone administration affect subsequent survival of a long-lived bird.

2. Materials and methods

2.1. Study area and birds

Our study was conducted during the breeding seasons 2005, 2006 and 2007 with a colony of Black-legged kittiwakes at Kongsfjorden, Svalbard (78°54′N, 12°13′E), 7 km east of Ny-Ålesund, Norway. 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. Birds were individually marked with metal rings. Their sex was already known by molecular sexing (Weimerskirch et al., 2005) carried out previously at the Centre d'Etudes Biologiques de Chizé (CEBC).

2.2. Manipulating corticosterone levels

We manipulated adult kittiwakes between 23 July and 7 August 2005, during the mid chick-rearing period (chick age 15–20 days). In order to reduce variance related to sex differences, we focused our study on males only. At day 0, 43 chick-rearing males were captured at their nest with a noose at the end of a 5 m fishing rod. Blood samples were collected immediately after capture for baseline corticosterone (Romero and Reed, 2005) 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. 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, $n = 22$ Cort-implanted birds) or empty ($n = 21$ sham-implanted birds). Knowing that corticosterone cannot diffuse through silastic, we cut both ends of the silastic tube to allow rapid release of corticosterone over a limited time period. Our aim was to increase plasma corticosterone levels (2 days, see Angelier et al., 2007a, 2009a) within the physiological range (stress-induced levels in male kittiwakes, Kitaysky et al., 1999a; Chastel et al., 2005). However, we did not remove the implants after this experimental increase, and thus, we were not able to monitor the diffusion rate of crystallized corticosterone (see Kitaysky et al., 2003). At day 0, both groups (Cort-implanted birds and sham-implanted ones) did not differ significantly with respect to body condition, baseline corticosterone levels, date of treatment, and brood size (see Angelier et al., 2007a, 2009a for details).

2.3. Subsequent blood samples

Following implantation, kittiwakes were recaptured opportunistically when they were present at the colony (Angelier et al., 2007a, 2009a). Specifically, birds were recaptured 1, 2, 3 and 8 days after the implantation and were sampled for blood as described above. One part of the birds was sampled at day 1 (Cort-implanted birds, $N = 9$, sham-implanted birds, $N = 4$), another part at day 2 (Cort-implanted birds, $N = 5$, sham-implanted birds, $N = 3$) to confirm that our corticosterone manipulation was effective. In addition, 14, then 10 Cort-implanted birds and 18, then 9 sham-implanted birds were sampled at days 3 and 8, respectively. Moreover, baseline corticosterone levels the day after Cort-implantation were compared to average stress-induced corticosterone levels of chick-rearing males sampled in 2000 and 2004 ($N = 75$, see Chastel et al. (2005) and Angelier et al. (2007b) for detailed methodology). This was done to ensure that the experimental increase of corticosterone was within the physiological range observed for Black-legged kittiwakes. In the sham-implanted birds, the corticosterone level was not significantly changed in the days following implantation, whereas in the Cort-implanted birds, the corticosterone value was significantly increased the first and second days after implantation. From days 3 to 8 the corticosterone values were not different from the baseline level in both groups of birds (see Angelier et al. (2009a) for detailed statistical analysis, Fig. 1). The corticosterone level of Cort-implanted birds one day after implantation was not different from the acute stress-induced corticosterone level measured in kittiwakes from the same study site after an acute stress protocol (Chastel et al., 2005; Angelier et al., 2007b; df = 21, $t = -1.72$, $p = 0.099$, Fig. 1).

2.4. Hormone assays

Within 6 h from sampling, blood samples were centrifuged and the plasma was stored at −20 °C for later analysis. All laboratory analyzes were performed at the Centre d'Etudes Biologiques de Chizé (CEBC). Plasma concentrations of corticosterone were determined by radioimmuno-assay as detailed in Lormée et al. (2003). The intra-assay variations was 7.7% and the minimal detectable corticosterone levels was 0.5 ng. No samples fell below this limit.

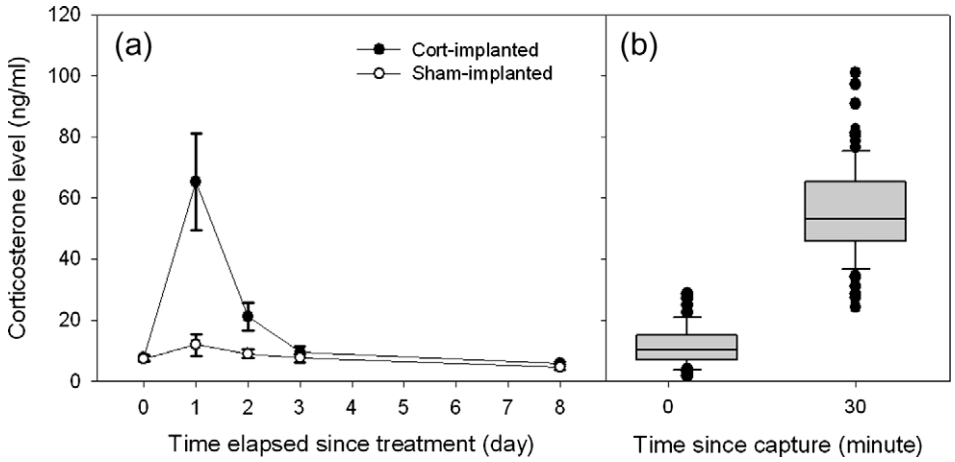

Fig. 1. (a) Effect of corticosterone and empty implants (sham) on plasma levels of corticosterone 1–8 days after implantation in male Black-legged kittiwakes during the chick-rearing period 2005 (mean ± SE). (b) Baseline and stress-induced corticosterone levels reached after an acute stress protocol in Black-legged kittiwakes in the same site (Chastel et al., 2005; Angelier et al., 2007b). Plasma corticosterone measured one day after corticosterone administration (Fig. 1a) where similar to those measured after 30 min of handling protocol (Fig. 1b).

2.5. Survival monitoring

All birds were marked with a plastic ring with a code of three letters allowing identification from a distance without perturbation. Information on survival and local dispersal were gathered during the 2 following years (2006 and 2007), through intensive and extensive field surveys of ringed birds. Field surveys were conducted daily between 26th of June and 9th of August in 2006 and from 26th of April to 4th of August in 2007. Local breeding dispersal was monitored by extending re-sighting effort to four other colonies of kittiwakes (from 120 to 200 pairs), located in the immediate vicinity of the study plot and up to 10 km apart. However, long-scale breeding dispersal was not estimated because other colonies in the region were inaccessible and hence not surveyed. Annual survival rate estimated for non-manipulated males from the same colonies between 2001 and 2007 was 75.4% (range 65.4–83.2%), while mean annual recapture rate was 91.4% (range 76.9–97.2%, unpublished data).

2.6. Data analysis

The re-sighting at time $t + 1$ of a ringed individual manipulated at t depends on three events and their associated probabilities: the probability of surviving from t to $t + 1$, called the survival rate and written Φ; the probability, if alive, of being present in the studied colonies at t, called the presence probability; and a probability, if alive and present, of being resighted: the recapture probability (p). We therefore estimated annual apparent survival and recapture probabilities using the general methods of Lebreton et al. (1992) and Burnham and Anderson (2002). Four models were thus built:

(1) Φ (CORT); p (CORT) considers an effect of corticosterone treatment (CORT) on survival rates from 2005 to 2006 (Φ) and on recapture rates in 2006 (p).
(2) Φ (CORT) considers an effect of CORT on Φ but no effect on p.
(3) p (CORT) considers an effect of CORT on p but no effect on Φ.
(4) Null model considers no effect of CORT on Φ and p.

Program MARK (White and Burnham, 1999) was used to assess the fit of these candidates' models to the data set, using the Akaike Information Criterion, corrected for small sample size (AICc). The model with the lowest AICc is considered as the best one (Burnham and Anderson, 2002). If ΔAICc (difference between the AICc value of one model relative to the AICc value of the best model) is lower than 2.0, the two models are indistinguishable. AICc weight measures a model's relative probability of being the best model for the data among the set of candidate models.

Before comparing the fit of the candidate models, program RELEASE (Burnham et al., 1987) was used to perform a goodness of fit test for the data set (test 1, between groups test: $\chi^2 = 5.529$, $p = 0.063$). Because of only three capture events, tests 2 and 3 of the GOF could not have been performed.

3. Results

The best-fitting model is the second one (called "Φ (CORT)", Table 1). This model postulates that the survival rate of Cort-implanted birds differs significantly from the survival rate of sham-implanted ones, whereas recapture rates in 2006 are similar for the both groups. An effect of corticosterone implantation in 2005 on the apparent survival rate from 2005 to 2006 is highly supported (91.38% of the sum of AICc weights, Table 1) by model selection. Models including effects of the treatment on recapture rate in 2006 were not well supported (25% of the sum of AICc weights, Table 1).

The estimated survival rate from 2005 to 2006 derived from the best-fitting model, i.e. Φ (CORT) was 39.7% lower for Cort-implanted birds than for sham-implanted ones (Fig. 2). The recapture rate of Cort and sham-implanted kittiwakes in 2006 was estimated to 100% by MARK software.

4. Discussion

4.1. Apparent survival, dispersal and skipped breeding

In the present study, we estimated apparent survival rate. In mark-recapture studies, the presence probability is included in

Table 1
Model selection to test the influence of hormonal manipulation (CORT) in 2005 on survival rates (Φ) and recapture rates (p) in 2006. The best model was selected by using the ΔAICc and AICc weights.

#	Model	AICc	ΔAICc	AICc weights (%)	No. parameters
(2)	Φ (CORT)	88.750	0.000	69.07	3
(1)	Φ (CORT); p (CORT)	91.010	2.260	22.31	4
(4)	Null model	93.631	4.881	6.02	2
(3)	p (CORT)	95.307	6.557	2.60	3

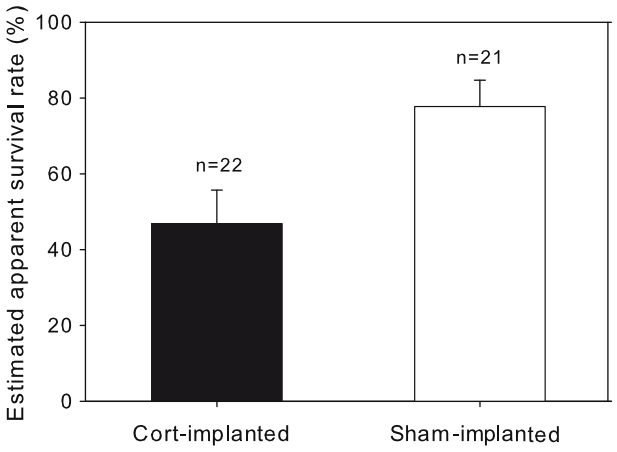

Fig. 2. Effects of the hormonal manipulation in 2005 on estimated apparent survival rate between 2005 and 2006 in males Black-legged kittiwakes (means ± SE). Means and standard errors of survival rates were estimated using MARK software, and are based on the best-fitting model, i.e. model (2) 'Φ (CORT)' (see text for details).

survival rate or in recapture probability, if absence is permanent or temporary, respectively (Lebreton et al., 1992; Kendall and Nichols, 1995). The observed negative effect of corticosterone administration on apparent survival could then be attributed to mortality or to absence. The Cort-implanted kittiwakes had a lower reproductive success in 2005 (Angelier et al., 2009a), which is well-known to reduce breeding propensity and to enhance breeding dispersal the next year in kittiwakes (Cam et al., 1998; Naves et al., 2006; Boulinier et al., 2008). Furthermore, corticosterone is also known to trigger irruptive migration and dispersal (Astheimer et al., 1992; Belthoff and Dufty, 1998; Meylan et al., 2002). One may suggest that the lower apparent survival of Cort-implanted kittiwakes may be the result of multiple skipped breeding events or permanent breeding dispersal. Concerning skipped reproductions, Cort-implanted birds observed in 2006 were all seen incubating or caring for chicks. This suggests that the breeding probability of resighted kittiwakes was not affected by our treatment. Regarding dispersal movement, we only recorded two small scale movements between study plots, involving one Cort-implanted bird and one sham-implanted bird. However, we cannot exclude the possibility that some Cort-implanted birds established permanently in other colonies outside the study area. The apparent low survival rate (47%) of Cort-implanted kittiwakes may therefore be a result of mortality and possible large-scale breeding dispersal.

4.2. Possible effects of experimentally increased corticosterone levels on survival

Our experimental setup resulted in a significant reduction of the apparent annual survival in kittiwakes (46.9% vs 77.8%). It is important to notice that we did not observe any mortality of Cort-implanted birds over a 10-day period (Angelier et al., 2009a). In a similar study, Cort- and sham-implanted kittiwakes showed the same survival during a 3-week period after the implantation (Kitaysky et al., 2001). Hence, mortality would have occurred during the post-breeding period, long after the implantation.

In our study, corticosterone implants were not removed and the crystallized corticosterone could not be metabolized in few days. A similar study conducted in the chicks of the same species (Kitaysky et al., 2003) pointed out that the implants were not totally depleted 4 weeks after the treatment and still contained approximately 2/3 of the initial amount of crystallized corticosterone. Although plasma corticosterone levels only increased over a 2-day period (Angelier et al., 2007a, 2009a), the remaining exogenous corticosterone could have diffused over a long-term period. In that context, our corticosterone manipulation could rather mimic GCs effects within a chronic framework (Breuner et al., 2008). Chronically elevated corticosterone levels are known to compromise cognitive and learning abilities (McEwen and Sapolsky, 1995; Wingfield et al., 1998; McEwen, 1999 but see Pravosudov, 2003), immune functions and recovery capacities (Dhabhar and McEwen, 1997, 1999; McEwen and Sapolsky, 1995; Saino et al., 2003; Berger et al., 2005), and also provoke the waste of muscle tissue (reviewed in Sapolsky, 2000). Ultimately, long-term exposure to high corticosterone levels had detrimental effects on survival in long-lived vertebrates (Romero and Wikelski, 2001; Brown et al., 2005; Kitaysky et al., 2007, in press). If our corticosterone manipulation had consisted in the release of exogenous corticosterone over a long-term period, this would have caused the deleterious effects described above, ultimately reducing survival. The reason why baseline corticosterone levels decreased the 3th and 8th days after the treatment could result from a negative down-regulation of endogenous corticosterone release (see below), or a high clearance rate of exogenous corticosterone from the blood system. Moreover, we only measured total corticosterone levels, i.e. free and bound hormone levels. The binding protein Corticosteroid Binding Globulin (CBG) binds corticosterone with high affinity in circulation (e.g. Breuner and Orchinik, 2001; Shultz and Kitaysky, 2008). Although the primary role of CBG is under debate, the Free Hormone Hypothesis suggests that unbound, or free, concentration of hormone in the plasma is biologically active. It is conceivable that free corticosterone levels may have been higher in Cort-implanted kittiwakes compared to the control ones, while total corticosterone levels were similar between groups.

Other endocrine processes may also be implied in the low apparent survival of the Cort-implanted birds. Administration of exogenous GC is known to inactivate the HPA axis in birds (Vandenborne et al., 2005). Specifically a negative feedback down-regulates corticotropin-releasing factor (CRF) gene expression in the hypothalamus (Kretz et al., 1999; Ma et al., 2001; Feldman and Weidenfeld, 2002). This may reduce CRF receptors production in the pituitary (Pozzoli et al., 1996; Aguilera et al., 2001) resulting in an inhibition of the adrenocorticotropin ACTH secretion (Dallman et al., 1987). Recent lab and field studies in birds highlighted a down-regulation of the HPA system following corticosterone administration, through dermal application (Busch et al., 2008), pellets (Müller et al., 2009) or silastic implants (Romero et al., 2005). Thus, GC administration results in a reduction of the magnitude of the adrenocortical response (Romero et al., 2005; Busch et al., 2008; Müller et al., 2009). This effect was relatively long-lasting since for example, acute stress response was absent 8 days after corticosterone administration in kestrel (*Falco tinninculus*) nestlings (Müller et al., 2009). ACTH challenge confirmed that the sensitivity of the HPA axis was down-regulated at the hypothalamo–pituitary level or higher (Busch et al., 2008). In the present study, exogenous corticosterone may have down-regulated endogenous corticosterone secretion so that HPA axis was deactivated or impaired. Thus, life-threatening perturbations (e.g. poor food condition, predation or inclement weather) might not have induced acute endogenous corticosterone releases in Cort-implanted kittiwakes. This inability to adopt an emergency coping response could have resulted in a high mortality (Wingfield et al., 1998; Wingfield and Kitaysky, 2002; Angelier et al., 2009b). Because Cort-implanted birds were not seen dead over a 10-day period (Angelier et al., 2009a), mortality of kittiwakes might have occurred during fall or winter following Cort-implantation. This would suggest that our Cort-treatment resulted in a longer down-regulation of endogenous stress response (more than 1 week

after implantation). Although this glucocorticoid negative feedback is still poorly studied in free-living individuals (Busch et al., 2008), it has been shown to last less than 20 days by using corticosterone pellets (Müller et al., 2009). The possible effects of GC administration on fitness have thus to be better understood (Breuner et al., 2008).

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