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Original Article

Behavioral and physiological responses to male handicap in chick-rearing black-legged kittiwakes

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Parental investment entails a trade-off between the benefits of effort in current offspring and the costs to future reproduction. Long-lived species are predicted to be reluctant to increase parental effort to avoid affecting their survival. We tested this hypothesis in black-legged kittiwakes *Rissa tridactyla* by clipping flight feathers of experimental males at the beginning of the chick-rearing period. We analyzed the consequences of this handicap on feeding and attendance behavior, body condition, integument coloration, and circulating levels of corticosterone and prolactin in handicapped males and their mates in comparison to unmanipulated controls. Chicks in both groups were compared in terms of aggressive behavior, growth, and mortality. Handicapped males lost more mass, had less bright integuments, and attended the nest less often than controls. Nevertheless, they fed their chicks at the same rate and had similar corticosterone and prolactin levels. Compared with control females, females mated with handicapped males showed a lower provisioning rate and higher nest attendance in the first days after manipulation. Their lower feeding rate probably triggered the increased sibling aggression and mortality observed in experimental broods. Our findings suggest that experimental females adaptively adjusted their effort to their mate's perceived quality or that their provisioning was constrained by their higher nest attendance. Overall, our results suggest that kittiwake males can decrease their condition for the sake of their chicks, which seems to contradict the hypothesis that kittiwakes should be reluctant to increase parental effort to avoid affecting their survival. *Key words:* body condition, cost of reproduction, feather clipping, parental effort, *Rissa tridactyla*. *[Behav Ecol 22:1156–1165 (2011)]*

INTRODUCTION

Life history theory predicts that, in iteroparous species, parental investment (i.e., the trade-off between survival and parental effort) in current reproduction should be balanced by the costs in terms of residual reproductive value (Stearns 1992). Increased parental effort may enhance reproductive success through improved chick growth and survival but may compromise adult survival and lifetime reproductive success. In short-lived passerines, the probability of survival to future reproduction is low, so an increase of parental effort at the cost of reduced survival probability would be expected in response to an increment in chick demand (Linden and Møller 1989). In contrast, in long-lived species, which have many breeding opportunities, lifetime reproductive success is marginally affected by the success of individual breeding attempts. Therefore, adults should be restrictive in increasing effort to moderate the impact on future reproduction (Drent and Daan 1980; Linden and Møller 1989). Several studies support this hypothesis in long-lived seabirds (Ricklefs 1987; Saether et al. 1993; Hamer and Hill 1994; Mauck and Grubb 1995; Navarro and González-Solis 2007), whereas others have reported that provisioning effort was adjusted to offspring's requirement (Tveraa et al. 1998; Granadeiro et al. 2000; but see Table 2 in Velando and Alonso-Alvárez 2003 and Table 1 in Bijleveld and Mullers 2009).

In addition, in species with long-term pair bonds and biparental care, each parent must balance its reproductive investment against that of its partner (Trivers 1972; Chase 1980; Houston and Davies 1985; Houston et al. 2005). Sexual conflict and negotiation over care models predict that only partial compensation for a mate's reduced parental effort must occur to maintain a stable evolutionary strategy of biparental care (Houston and Davies 1985; McNamara et al. 1999; review in Houston et al. 2005). Although most experimental studies that have tested this prediction have shown partial compensation both in short-lived and in long-lived species (Harrison

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et al. 2009), there is considerable variation in the direction and magnitude of responses, ranging from none (Sanz et al. 2000) to complete compensation (Griggio and Pilastro 2007).

One possible approach to explore how birds balance their current reproductive effort against the resulting cost imposed on future reproduction may be by experimental manipulations of reproductive costs to one of the parents (Reznick 1985; Partridge and Harvey 1988). Reproductive costs have often been manipulated by altering chick demands or increasing flight costs (see Table 4 in Sanz et al. 2000; see Table 2 in Velando and Alonso-Alvárez 2003; see Table 1 in Navarro and Gonzalez-Solis 2007). An increase in energetic costs associated with the handicap may negatively affect residual reproductive value through elevated mortality or reduced future reproductive success (Golet et al. 1998; Wernham and Bryant 1998). Handicapped long-lived seabirds are thus expected to reduce their parental effort to limit the impact of the handicap on their condition and future survival, whereas their partners are expected to show restraint in their compensation.

The black-legged kittiwake *Rissa tridactyla* is a socially and genetically monogamous long-lived seabird (Helfenstein et al. 2004) with prolonged biparental care (Coulson and Porter 1985). To test the aforementioned hypotheses, we experimentally decreased the wing and tail area of breeding males and examined changes in body mass and behavior of handicapped parents and their mate. In kittiwakes, siblicide (i.e., fatal sibling aggression) is common and is mainly triggered by a low parental feeding rate (Braun and Hunt 1983; White et al. 2010). Siblicide may thus be considered as an indicator of parental effort, and chick aggression and chick mortality were studied in detail.

In birds, carotenoid-based signals may reflect foraging ability and health (Lozano 1994), and in many bird species, including kittiwakes (Leclaire et al. 2011), integument coloration provides accurate information about current individual physical condition (Faivre et al. 2003; Velando et al. 2006; Martínez-Padilla et al. 2007). Thus, we examined differences in integument coloration between handicapped and control birds. Furthermore, in birds, corticosterone and prolactin hormones seem to mediate the trade-off between parental effort and survival (Wingfield and Sapolsky 2003; Chastel et al. 2005; Angelier et al. 2009; Bókony et al. 2009). Low prolactin levels are associated with reduced nest attendance and chick provisioning (Wang and Buntin 1999; Angelier et al. 2009a), whereas elevated corticosterone levels are associated with physiological stress and may trigger reduced brood provisioning and nest abandonment (Wingfield and Sapolsky 2003; review in Angelier and Chastel 2009). After an acute stress, corticosterone levels increase dramatically (Wingfield et al. 1998), whereas prolactin levels seem to decrease, at least from incubation to midchick rearing (Chastel et al. 2005; Angelier et al. 2007; Angelier and Chastel 2009; Riou et al. 2010), which reduces reproductive activities and promotes survival. The magnitude of the acute corticosterone and prolactin stress response depends on the fitness value of the current reproductive event and has been suggested to be a relevant measure of parental investment (Angelier et al. 2009a). We thus examined differences in corticosterone and prolactin levels between handicapped and control birds, after a standardized stress protocol.

Given the longevity of kittiwakes, handicapped males were expected to limit the impact of the handicap on their survival at the expense of their current reproduction and thus to have lower food provisioning and nest attendance but similar body mass than control males. Given the stress of the handicap, they were expected to have duller integuments than control birds. They were expected to redirect energy toward survival and thus to have higher corticosterone and lower prolactin levels. Females mated with handicapped males were expected to compensate partially for the decrease of their partner parental care and to have higher food provisioning than control females. Chicks with a handicapped father were expected to be overall less fed and to grow less rapidly than control chicks. The first-hatched chicks were then expected to be more aggressive and therefore the second-hatched chicks to die more often of siblicide.

MATERIALS AND METHODS

Study site

The study was conducted from late June to mid-August 2007 and 2008, on a population of black-legged kittiwakes nesting on an abandoned US Air Force radar tower on Middleton Island (59°26′N, 146°20′W), Gulf of Alaska. Artificial nest sites created on the upper walls were observed from inside the tower through sliding one-way windows (Gill and Hatch 2002). This enabled us to easily capture and monitor breeders and chicks. All nest sites were checked twice daily (9:00 and 18:00) to record events such as laying, hatching, or chick mortality. In 2007, a preliminary study was carried out to study the effect of male handicapping on parent and chick body mass and chick mortality. In 2008, we additionally monitored changes in behavior, hormone, and integument coloration of adults.

Experimental procedures

A total of 79 pairs with 2 hatchlings were used for this experiment. Fifteen pairs were used both in 2007 and in 2008. Pairs were randomly assigned to 1 of the 2 treatment groups (Experimental pairs: $n = 20$ and $n = 26$ in 2007 and 2008, respectively, Control pairs: $n = 19$ and $n = 29$ in 2007 and 2008, respectively). Thirty-four adults were sexed using molecular methods and 86 adults were sexed based on copulation and courtship feeding during the prelaying period. Twenty other adults were sexed based on the known sex of their partner. Ten pairs could not be sexed according to these methods and they were sexed based on skull length (head + bill): within a pair female's skull length < male's skull length (Jodice et al. 2000).

Both parents were captured as soon as possible after the second chick hatched (from 0 to 2 days in 2007 and 2008; mean ± standard error [SE]: 0.49 ± 0.04 days). In 2008, blood samples were drawn within 3 min of capture (mean: 2 min 14 s ± 3 s, from 1 min 1 s to 2 min 59 s) to determine baseline corticosterone and prolactin levels. Blood samples were collected from the alar vein with a 1 ml syringe and a 25 gauge needle (maximum amount of blood collected: 700 µl). Birds were then weighed to the nearest 5 g with a Pesola scale and skull length was measured to the nearest millimeter with a caliper. Birds were individually marked with a code of color rings for visual identification. In 2007, one bird of each pair was further colored on the neck and head with picric acid. In 2008, males were painted on the neck with picric acid, whereas females were painted with animal marking sticks (RAIDEX). Finally, we increase male flight costs experimentally by clipping feathers. The wing area of experimental males was reduced by clipping the no. 3, 5, and 7 primary remiges (counted from outside) of each wing and the 2 central rectrices. In 2008, 2 more central rectrices were clipped to adjust the handicap to the high quality environmental conditions of that year. Feathers were cut near their base with scissors. Control males were handled in the same way but no feathers were clipped (Figure 1).

Both parents were recaptured 15 days after the first manipulation (mean: 15.30 ± 0.06 days; from 14 to 20 days in 2007 and from 14 to 17 days in 2008). At recapture, all birds were blood sampled within 3 min of capture (mean: 2 min 34 s ± 3 s, from 1 min 32 s to 3 min 0 s) and weighed. Birds were then kept in an

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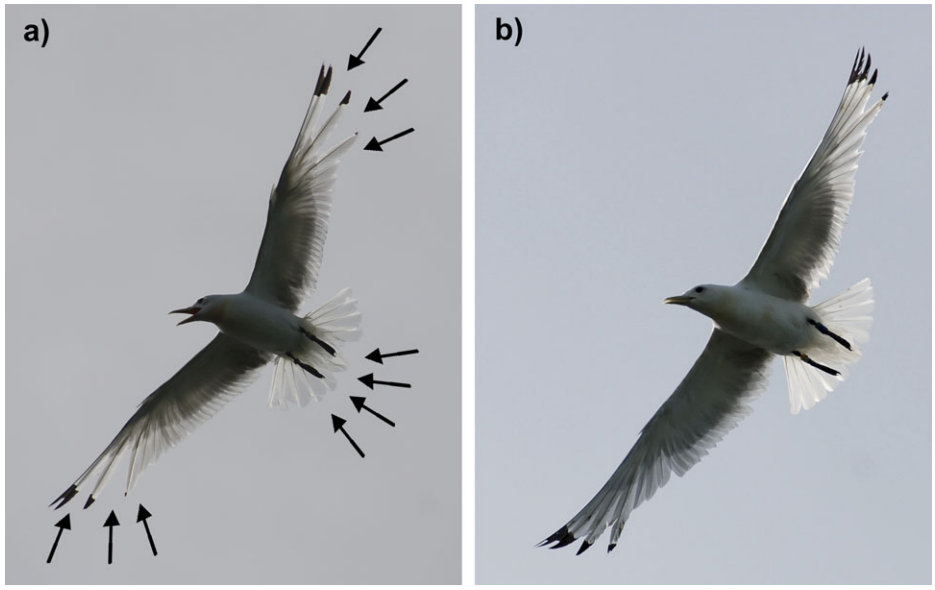

Figure 1
Handicapped (a) and control (b) males. Arrows show the emplacement of clipped feathers. Photos by Emilie Moëc.

individual opaque cloth bag for 30 min. Afterward, a second blood sample was taken to estimate the corticosterone and prolactin responses to capture and handling (Chastel et al. 2005). All blood samples were centrifuged immediately after collection and plasma was stored at −20 °C.

At hatching, A- and B-chicks (the first- and the second-hatched chick, respectively) were marked on the head with a nontoxic marker for identification. In 2007, A-chicks were colored in red, whereas B-chicks were colored in blue. In 2008, color was randomly assigned. Chicks were weighed every 5 days from hatching to 35 days old. Body mass was measured to the nearest gram using an electronic scale.

In the 2009 prelaying period (mean: 11 ± 7 days before laying, from 2 to 26 days), 11 males and 11 females of the 2008 experiment were recaptured, weighed, and measured to assess the effect of the handicap on body condition in the subsequent breeding season.

Behavioral observations

In 2008, we recorded parent and chick behavior during the first 14 days after the manipulation. Observations began the day after males were handicapped and ended 14 days after the manipulation or when the 2 chicks died or disappeared from the nest. Each nest was observed 3 times a day for 15 min, with a period of at least 2 h between 2 observation bouts. Recorded behaviors were chick feeding, chick aggression, and parental attendance. Aggression intensity and feeding quantity were measured using the following predefined scores: 1 for weak aggression (or low food amount given to the chick), 2 for moderate aggression (or medium food amount), and 3 for intense aggression (or large food amount). Observations were done blind to treatment. Feeding and aggression intensity were calculated as the daily mean of the total intensity per observation bout (i.e., per 15 min). We assigned a daily probability of feeding or aggression of 1 when at least 1 feeding or aggression event was recorded, and a probability of 0 when none was recorded on a given day.

Integument color measurements

In 2008, integument coloration was measured from digital photographs. Pictures of eye ring, gape, tongue, and bill of each bird were taken from a standard distance using the camera flash. For each photograph, a color swatch was placed next to the bird to standardize the measurement (Montgomerie 2006). All pictures were then analyzed using Adobe Photoshop 7.0. For each picture, the average components red, green, and blue (RGB system) were recorded within the whole area of the eye ring, tongue, and bill and within a standardized selected area of the gape. This allowed us to determine the hue, saturation, and brightness of each area. Hue corresponds to what we call "color" in everyday speech (i.e., red, orange, and yellow), saturation represents color density (e.g., pink is less saturated than red), and brightness indicates whether a color is dark or light independent of the hue and saturation. The range of our color measurements is smaller than the range of colors perceived by kittiwakes, which possess receptors for UV light (Hastad et al. 2005; Hastad et al. 2009). However, carotenoids are subtractive pigments and some UV components covary with visible components in kittiwakes (our unpublished data) and some other species (Mougeot et al. 2007; Thorogood et al. 2008). The measurement errors from picture-based analysis may thus have relatively little effects on our conclusion. Furthermore, information obtained from digital pictures has revealed patterns and effects of biological meaning in several bird species (e.g., Blas et al. 2006; Martínez-Padilla et al. 2007; Mougeot et al. 2007; Pérez-Rodríguez and Viñuela 2008). A principal component analysis on all integument parameters (i.e., hue, saturation, and brightness of gape, tongue, eye ring, and bill, $n = 12$ parameters) was run to reduce the number of variables. The first 3 principal components (PCs) were used in the statistical analysis.

Hormonal assays

All hormonal analyses were performed at the Centre d'Études Biologiques de Chizé. Plasma concentrations of corticosterone were determined in one assay following methods described in Lormée et al. (2003). Concentrations of prolactin were determined in one assay with the remaining plasma by a heterologous radioimmunoassay as detailed and validated for this species (Chastel et al. 2005). The intraassay variations were 6.9% for corticosterone and 9.4% for prolactin. Because initial blood samples were collected within 3 min of capture, corticosterone and prolactin levels were considered to reflect baseline levels (Chastel et al. 2005; Romero and Reed 2005). When not enough blood was collected for corticosterone and

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prolactin analyses, corticosterone analysis was prioritized over prolactin analysis. A blood amount sufficient for analyses was more difficult to get at the first blood sampling (within 3 min of capture) than at the second blood sampling (after 30 min of capture), leading to a lower sample size for baseline values than for stress-induced values (Table 1).

Data analyses

All analyses were conducted with the SAS system version 9.1. Differences in body mass loss and in coloration at day 15 between experimental and control birds were analyzed with generalized linear mixed models (GLMMs, Proc MIXED). Treatment, Year, and Parental sex were entered as fixed effects and Nest as a random effect. Bird Identity was entered as a random effect in the body mass analysis, as the same bird may have been weighed in 2007 and 2008. Mass loss or color PC at the second capture was the dependent variable. Mass and color PC at the first capture were entered as a covariate in the body mass and coloration analyses.

The difference in body condition in the 2009 prelaying period between 2008 experimental and control birds was analyzed with generalized linear models (GLMs, Proc GLM). Body mass was entered as the dependent variable. Treatment, sex, skull length, and number of days between capture and laying were entered as fixed effects.

Log-transformed corticosterone and prolactin levels and the difference between baseline and stress-induced corticosterone and prolactin levels were analyzed with GLMs, with Sex, Treatment, and Handling time as fixed effects.

Males and females were never seen attending the nest together except during parental shifts (i.e., when parents took turns to brood the chicks). When a parental shift was recorded during the 15 min of observation, we considered only the first parent present on the nest. Attendance took values from 0 (the parent was not seen on the nest during the 3 daily observation bouts) to 3 (the parent was seen on the nest during each of the 3 daily observation bouts). Attendance was thus analyzed with a multinomial distribution (Proc GLIMMIX).

Feeding and aggression probability were analyzed with a binomial distribution (Proc GLIMMIX). Treatment, Rank (except in the aggression analyses), and Age of the chicks were entered as fixed effects and Nest as random effect.

Behavioral observations ended 14 days after manipulation, which corresponded to 15–20 days after first chick hatching. We thus analyzed chick growth between hatching and the age of 20 days. Chicks were last measured at 35 days old to avoid precocious fledging (ca. 40 days old), and we also analyzed chick body mass and tarsus length at the age of 35 days. Chick growth between the age of 0 and 20 days and body mass and tarsus length at the age of 35 days were analyzed with Treatment and Chick Rank as fixed effects and Nest as a random effect. Chick mortality was analyzed with a binomial distribution (Proc GENMOD).

Nonsignificant terms were backward dropped using a stepwise elimination procedure. We used 2-tailed type-3 tests for fixed effects with a significance level set to $\alpha = 0.05$. All GLMMs assumed normal distribution of the error and used the restricted maximum likelihood estimation method (REML-GLMMs) and the Satterthwaite correction for the calculation of fixed effects degrees of freedom (Littell et al. 2006). Values are expressed as mean ± SE throughout.

RESULTS

Year effect

Kittiwake breeding performance was better in 2008 than in 2007 in several ways. In 2008, hatching occurred earlier (median: 28 June vs. 7 July, $U_{256,152} = 45633$, $P < 0.0001$), parents were heavier (parental weight before manipulation: 465 ± 3 vs. 445 ± 4 g, $F_{1,93} = 27.79$, $P < 0.0001$), hatchlings were heavier (37.8 ± 0.4 vs. 34.6 ± 0.4 g, $F_{1,91} = 16.99$, $P < 0.0001$), and B-chicks had a higher growth rate (between hatching and day 20 posthatch: 271 ± 11 vs. 233 ± 11 g, $F_{1,45} = 6.27$, $P = 0.016$, Rank × Year: $F_{1,62.8} = 4.63$, $P = 0.035$) and a lower mortality (30 ± 4% vs. 49 ± 6%, $\chi^2 = 7.62$, $P = 0.0058$). Finally, fledging success was higher (1.41 ± 0.09 chicks vs. 1.05 ± 0.13 chicks, $F_{1,90} = 5.32$, $P = 0.023$).

Breeding desertion and return rate

The treatment had no significant effect on the desertion of breeders. In 2007, one experimental male and one experimental female deserted the nest 5 days after manipulation. In 2008, one experimental male, one control male and one control female deserted the nest 11, 9, and 1 days after the manipulation, respectively.

The treatment had no significant effect on return rate the following year ($F_{1,28} = 0.02$, $P = 0.90$). In total, 19% control and 18% experimental males and 23% control and 24% experimental females were not seen breeding the following year.

Parental body mass

Parental body mass decreased during the first period of chick rearing. This decrease depended on the interaction between Treatment and Sex ($F_{1,77.5} = 5.12$, $P = 0.027$; Figure 2). Experimental males lost more weight than control males ($F_{1,82} = 4.25$, $P = 0.043$), whereas the treatment had no detectable effect on female mass loss ($F_{1,78.9} = 0.77$, $P = 0.38$). The year had no significant effect on body mass loss ($F_{1,71.1} = 0.08$, $P = 0.78$).

Parental body mass in the prelaying season after the treatment was not different between experimental and control birds ($F_{1,17} = 0.02$, $P = 0.89$; least-square means ± SE controlling

Table 1

Baseline and stress-induced corticosterone and prolactin levels 15 days after treatment, in experimental and control males and females, in 2008

		Baseline level (ng/ml)		Stress-induced level (ng/ml)	
		Experimental	Control	Experimental	Control
Corticosterone	Males	14 ± 4 (13)	13 ± 4 (14)	48 ± 5 (23)	51 ± 5 (22)
	Females	24 ± 9 (14)	8 ± 3 (12)	43 ± 5 (21)	45 ± 5 (24)
Prolactin	Males	120 ± 14 (10)	108 ± 6 (9)	123 ± 4 (22)	127 ± 6 (22)
	Females	130 ± 10 (9)	111 ± 7 (9)	132 ± 5 (21)	133 ± 4 (23)

Values are expressed as mean ± SE. Sample sizes are given in parentheses.

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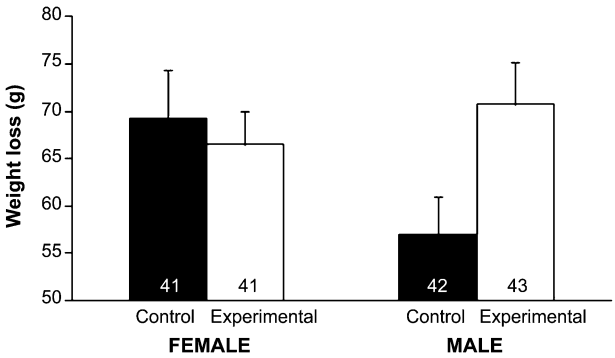

Figure 2
Female and male weight loss during the first 15 days after the manipulation in control and experimental groups in 2007 and 2008. Sample sizes are given in the bars.

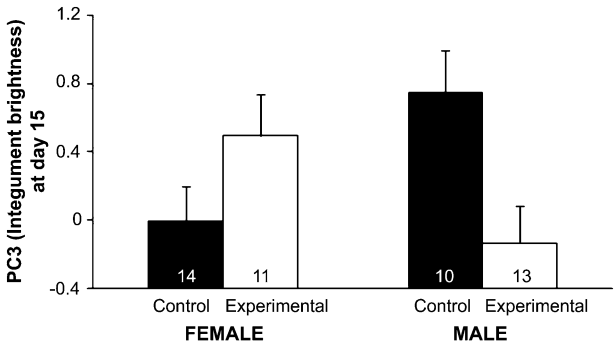

Figure 3
Integument brightness (as described by PC3) of control and experimental males and females at day 15 posttreatment. Shown are least-square means ± SE controlling for PC3 before treatment. Sample sizes are given in the bars.

for skull length and the number of days until laying: males: 428 ± 15 vs. 431 ± 9 g and females: 442 ± 13 vs. 441 ± 10 g).

Integument color

PC1 represented 21% of the total variance and got strong loading from eye ring hue (+0.51) and gape hue (+0.54). PC2 represented 18% of the total variance and got strong loading from eye ring saturation (+0.44), gape saturation (+0.50), and tongue saturation (+0.48). PC3 represented 13% of the total variance and got strong loading from bill brightness (+0.51), gape brightness (+0.48), eye ring brightness (+0.35), and tongue brightness (+0.45). After the treatment, experimental males had lower PC3 than control males ($F_{1,20} = 7.81$, $P = 0.011$, Figure 3), whereas there was no difference in PC3 between experimental and control females ($F_{1,22} = 1.41$, $P = 0.25$, Figure 3; Sex × Treatment: $F_{1,29.4} = 9.70$, $P = 0.0041$, Figure 3). PC1 and PC2 did not depend on Treatment or Sex (all $P > 0.05$).

Parental corticosterone and prolactin levels

Before manipulation, baseline corticosterone and prolactin levels did not depend on Treatment ($F_{1,63} = 1.96$, $P = 0.17$ and $F_{1,63} = 0.49$, $P = 0.48$) or Sex ($F_{1,62} = 0.03$, $P = 0.87$ and $F_{1,64} = 0.57$, $P = 0.45$). After manipulation, baseline corticosterone and prolactin levels did not differ significantly between experimental and control birds ($F_{1,25.2} = 0.68$, $P = 0.42$ and $F_{1,27} = 2.49$, $P = 0.13$, Table 1). Birds responded to handling with an increase in corticosterone level (Wilcoxon signed-rank test: $W = 1392$, $P < 0.0001$, $n = 62$) but no change in prolactin level (paired *t*-tests: $t_{39} = 1.47$, $P = 0.15$, $n = 40$). Stress-induced corticosterone and prolactin levels did not differ significantly between experimental and control birds ($F_{1,50} = 0.73$, $P = 0.40$ and $F_{1,28.4} = 1.34$, $P = 0.26$). Males tended to have a lower stress-induced prolactin levels than females ($F_{1,24.1} = 4.14$, $P = 0.053$; Table 1). The difference between baseline and stress-induced corticosterone and prolactin levels did not depend on treatment or sex (all $P > 0.15$).

Parental attendance

During the first 14 days after B-chick hatching, nests were never seen unattended except for 2 nests where males deserted. Males and females were never seen attending the nest together except during parental shifts. Parental attendance depended on the interaction between Treatment, Sex, and Chick Age ($F_{1,1318} = 7.32$, $P = 0.0069$; Figure 4). In control nests, males attended the nest more often than females through 14 days ($F_{1,43.84} = 13.12$, $P = 0.0008$), whereas in experimental nests, males attended the nest similarly as females during the first half of the period ($F_{1,48.33} = 1.40$, $P = 0.24$) and attended the nest more often than females during the second half ($F_{1,47.07} = 5.32$, $P = 0.026$; Sex × Chick Age: $F_{1,708} = 12.00$, $P = 0.0006$). Parental shifts tended to be less frequent in experimental than control nests (15 ± 2% vs. 22 ± 2%; $F_{1,54} = 3.99$, $P = 0.051$) and decreased over the 14 days of treatment in both groups ($F_{1,646} = 9.03$, $P = 0.0028$).

Feeding and aggressive behavior

During the first 14 days after manipulation, female feeding probability depended on the interaction between Treatment and Chick Age ($F_{1,701} = 5.44$, $P = 0.020$, Figure 5a). During this period, control females decreased their feeding rate ($F_{1,346} = 21.47$, $P < 0.0001$), whereas experimental females fed their chicks at a low rate all through the period ($F_{1,355} = 2.49$, $P = 0.12$). Male feeding probability did not depend on Treatment ($F_{1,55.1} = 0.13$, $P = 0.72$, Figure 5b) but decreased according to the age of the chicks ($F_{1,703} = 9.01$, $P = 0.0028$). Male and female feeding probabilities was not different between A- and B-chicks (in males: 0.16 ± 0.02 vs. 0.15 ± 0.02, $F_{1,1053} = 0.18$, $P = 0.67$ and in females: 0.15 ± 0.02 vs. 0.15 ± 0.02, $F_{1,1053} = 0.01$, $P = 0.93$). Feeding intensity did not

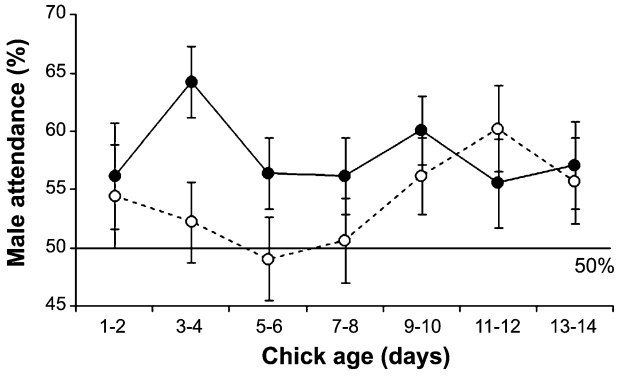

Figure 4
Male attendance in control (black symbols) and experimental (white symbols) nests in 2008. Female attendance (not shown on the figure) is complementary to male attendance (i.e., 100%—male attendance).

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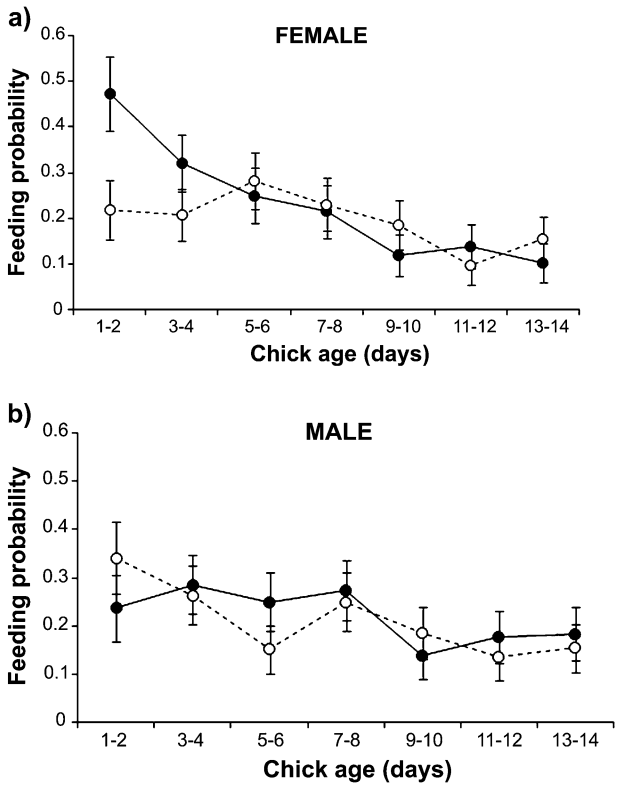

Figure 5
Female (a) and male (b) daily feeding probability according to the age of the chicks in experimental (white symbols) and control nests (black symbols).

depend on Treatment, Chick Age, Chick rank, or their interactions in males (all $P > 0.15$) and females (all $P > 0.15$).

During the first 14 days after manipulation, A-chicks were much more aggressive than B-chicks (76% of A-chicks were aggressive at least once vs. 10% of B-chicks; $F_{1,57} = 38.99$, $P < 0.0001$). A-chick aggression was significantly more frequent in experimental than control nests ($F_{1,54} = 4.43$, $P = 0.040$, Figure 6) and decreased according to Chick Age in both groups ($F_{1,445} = 27.76$, $P < 0.0001$, Figure 6). Experimental A-chicks tended to have higher aggression intensity than control A-chicks (4.83 ± 0.98 vs. 2.80 ± 0.56, respectively; $F_{1,46} = 3.92$, $P = 0.054$). Aggression intensity did not depend on Chick Age ($F_{1,43} = 1.36$, $P = 0.25$).

Chick growth and survival

During the first 20 days after hatching, experimental B-chicks grew less rapidly than control B-chicks ($F_{1,46} = 5.63$, $P = 0.022$; Figure 7), whereas there was no difference in A-chick growth rate between control and experimental broods ($F_{1,70} = 0.04$, $P = 0.84$, Rank × Treatment: $F_{1,61.4} = 6.74$, $P = 0.012$). Tarsus of experimental B-chicks grew also less rapidly than tarsus of control B-chicks (14.9 ± 0.5 vs. 15.9 ± 0.4 mm, $F_{1,46} = 4.68$, $P = 0.036$), whereas there was no difference in tarsus growth rate between A-chick in control and in experimental broods (15.9 ± 0.3 vs. 16.1 ± 0.3 mm, $F_{1,70} = 0.34$, $P = 0.56$, Rank × Treatment: $F_{1,63.3} = 3.21$, $P = 0.078$). At day 35 posthatch, however, A- and B-chicks were not significantly lighter in experimental than control broods (A-chicks: 421 ± 7 vs. 426 ± 8 g and B-chicks: 421 ± 10 vs. 410 ± 14 g; $F_{1,61.2} = 0.00$, $P = 0.97$; Figure 7), and their tarsus length was nonsignificantly

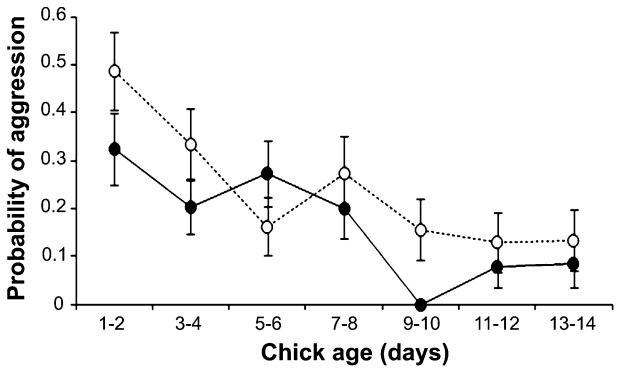

Figure 6
Daily probability of A-chick aggression in control (black) and experimental (white) nests according to chick age (counted from B-chick hatching) in 2008.

different (A-chicks: 36.6 ± 0.3 vs. 36.9 ± 0.3 mm and B-chicks: 35.9 ± 0.3 vs. 36.6 ± 0.4, $F_{1,63.1} = 1.71$, $P = 0.20$).

Ten dead chicks were observed to have severe wounds on the head. These injuries likely resulted from interchick aggression as parents were never observed pecking their offspring. We therefore deduce that these chicks died of siblicide. Such mortality tended to be more frequent in experimental broods than in control broods (17% vs. 4%; $\chi_1^2 = 3.49$, $P = 0.062$).

B-chick mortality tended to be higher in experimental broods than in control broods ($\chi_1^2 = 3.55$, $P = 0.060$; Figure 8). B-chicks died significantly more often than A-chicks ($\chi_1^2 = 15.03$, $P = 0.0001$). A-chick mortality did not differ significantly between experimental and control broods ($\chi_1^2 = 0.20$, $P = 0.66$; Figure 8). Although fledging success was higher in control than experimental broods (1.37 ± 0.10 vs. 1.15 ± 0.11 chicks), the difference was not significant ($F_{1,90} = 2.04$, $P = 0.16$).

DISCUSSION

Long-lived kittiwakes were predicted to prioritize survival and future reproductive output over current reproduction and hence reduce their parental effort when physically challenged. During years of overall good environmental conditions, we found, however, that handicapped kittiwake males maintained feeding probability constant at the expense of their physical condition. In contrast, females paired with

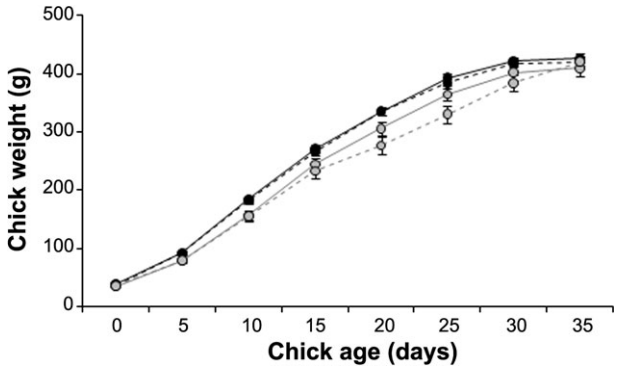

Figure 7
Weight growth of A-chicks (black symbols) and B-chicks (gray symbols) in experimental (dashed lines) and control nests (solid lines). Statistical analyses were done on the difference in mass between day 0 and day 20 and on mass at day 35.

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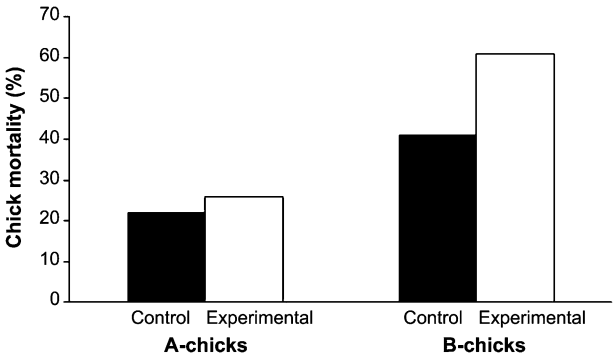

Figure 8
Percentage of A- and B-chicks that died before fledging in control and experimental nests.

handicapped males were found to decrease their feeding rate, apparently triggering chick aggression and siblicide.

Male flexible effort

Handicapped males fed their chicks at the same probability (Figure 5b) and intensity as control males, but they attended the nest less often (Figure 4) and showed a greater decrease in body condition (Figure 2). Decreasing the wing area increases wing loading and thus flight costs (Pennycuick 1989). Consequently, handicapped males may be forced to lengthen their foraging trips in order to find enough food to feed their chicks. This higher foraging effort may have caused their lower body condition. However, differences in attendance between handicapped and control males disappeared in the second half of the experimental period (from 8 to 14 days; Figure 4), which may have 3 alternative explanations. To maintain feeding effort and attendance as in control males, handicapped males may forage just enough to adequately feed their chicks but not enough to sustain their own body condition. Alternatively, during the second period, handicapped males may have been less constrained by the handicap than earlier. This could result from a change in environmental conditions or because the period was less energetically demanding, as chicks needed less brooding and feeding (Moe et al. 2002). The lower body condition of handicapped males would therefore result from the constraint during the first period. Finally, handicapped males may have adaptively reduced their body mass to compensate for the higher flight cost imposed by feather clipping (Norberg 1981; Pennycuick 1989).

Compared with control males, handicapped males had less bright integuments (Figure 3). Colors of many bird species are due to costly carotenoids (review in McGraw 2006) and are secondary sexual traits honestly indicating individual condition (review in Hill 2006). Few studies have focused on soft integument (e.g., gape, tongue, skin, caruncle, cere, tarsi) coloration as a cue of individual quality. In great black-backed gulls *Larus marinus*, however, integument color signals individual quality (Kristiansen et al. 2006), and in chick-rearing kittiwakes, coloration of gape, tongue, and eye ring is correlated with reproductive success, and carotenoid and vitamin A levels in males (Leclaire et al. 2011). The difference in integument color between handicapped and control males supports this signaling function of color in kittiwakes. It also suggests that increasing flight costs in males was constraining and represented energetic stress.

In birds, reduced prolactin levels are associated with reduced nest attendance and chick provisioning, whereas elevated corticosterone levels are associated with physiological stress that can trigger reduced brood provisioning and nest abandonment (Wingfield and Sapolsky 2003; review in Angelier and Chastel 2009; Angelier et al. 2009a). Given the stress of the treatment and the longevity of kittiwake, handicapped males were expected to redirect energy toward survival and thus to have higher corticosterone and lower prolactin levels, as observed in little auks *Alle alle*. (Harding et al. 2009). As in Cory's shearwaters *Calonectris diomedea* (Navarro et al. 2008), king penguins *Aptenodytes patagonicus* (Angelier et al. 2009b), or pied flycatchers *Ficedula hypoleuca* (Kern et al. 2007), however, we found that corticosterone and prolactin baseline levels and stress-induced responses were unaffected by the handicap (Table 1). A possible explanation lies in the timing of our sampling. Differences in male behavior were observed during the week after manipulation, whereas hormone levels were measured at the end of the second week of the experiment. Thus, it may be possible that handicapped males had higher corticosterone and lower prolactin levels in the week after treatment but had recovered normal levels at the time of blood sampling.

Handicapped males did not reduce parental care as shown by feeding rate. In most manipulative studies in long- or short-lived species, the first result is that handicapped individuals decrease their feeding rate (Weimerskirch et al. 2000; Paredes et al. 2005; for review, see Table 4 in Sanz et al. 2000). However, as kittiwake males, rock sparrow *Petronia petronia* males (Griggio et al. 2008) and great tit *Parus major* females (Sanz et al. 2000) have been shown not to reduce parental care when handicapped. Our results suggest that kittiwake males exhibit a strategy of fixed food delivery. This is consistent with other studies in kittiwakes showing that, contrarily to females, males do not adjust their food provisioning to natural variation in brood size during early chick rearing (Leclaire et al. 2010) and do not lose or gain weight when brood size is experimentally increased or decreased (Jacobsen et al. 1995).

Reduction in body condition in males may be interpreted as an increase in reproductive costs (Drent and Daan 1980), which may reduce long-term physiological condition (as for the immune system, e.g., Alonso-Alvarez and Tella 2001) and thereby residual reproductive value, through elevated mortality or reduced future reproductive success (Golet et al. 1998; Wernham and Bryant 1998). We did not detect any effects of the handicap on return rate or body condition the following year. We did not test, however, for survival or fecundity costs across years. In birds, manipulations that have demonstrated effects on adult condition have also often shown effects on adult residual reproductive value (review in Golet et al. 1998). Despite those potential costs, kittiwake males seem to pursue a strategy of high investment in reproduction. Consistent with this result, clutch removal manipulations in kittiwakes (Golet et al. 1998; Golet et al. 2004) and brood size manipulations in glaucous-winged gulls *Larus glaucescens* (Reid 1987) suggested that adult larids may compromise their own body condition or survival for the sake of their chicks. Although this result is in contradiction with many studies in long-lived procellariiformes (e.g., Antarctic petrels *Thalassoica Antarctica*, Saether et al. 1993; Leach's storm petrels *Oceanodroma leucorhoa*, Mauck and Grubb 1995; Antarctic prions *Pachyptila desolata*, Weimerskirch et al. 1999; Cory's shearwaters *Calonectris diomedea*, Navarro and González-Solis 2007) and even in the relatively short-lived tropical house wren *Troglodytes aedon* (Tieleman et al. 2008), it is in accord with experimental studies in Adélie penguins *Pygoscelis adeliae* (Beaulieu et al. 2009), little auks (Harding et al. 2009), or several short-lived passerines (e.g., pied flycatcher males *Ficedula hypoleuca*, Moreno et al. 1995, tree swallows *Tachycineta bicolor*, Winkler and Allen 1995, great tits, Matysiokova and Remes 2011; Sanz et al. 2000).

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Female-mediated brood reduction

As in Cape gannets *Morus capensis* (Bijleveld and Mullers 2009), kittiwake females responded to the low attendance of their handicapped mate by increasing their own nest attendance, without any apparent effect on their body mass. Contrary to males, kittiwake females exhibited flexibility in parental care, as shown by the low feeding probability during the 4 days after manipulation in females mated with handicapped males (Figure 5a). This period is critical for B-chicks as most brood reduction occurs in the first week posthatching. In many siblicidal species, starvation, which is due especially to low female feeding rate in kittiwakes (Leclaire et al. 2010), causes offspring aggression and siblicide (black-legged kittiwake, Braun and Hunt 1983; Irons 1992; White et al. 2010; blue-footed booby *Sula nebouxii*, Drummond and Chavelas 1989; osprey *Pandion haliaetus*, Machmer and Ydenberg 1998; black guillemot *Cepphus grylle*, Cook et al. 2000; but for a review, see Drummond 2001). Thus, the lower feeding rate of experimental females may have triggered higher intersibling aggression (Figure 6), higher weight difference between A- and B-chicks (Figure 7) and consequently higher siblicide rate in experimental than control broods (Figure 8).

Two hypotheses may explain the lower feeding rate of females mated to handicapped males. First, handicapped males are likely to have been perceived as low quality males (Hinde 2006). According to the "differential allocation hypothesis," experimental females may have adjusted their effort to the perceived quality of their mate (Burley 1988; Sheldon 2000; Hinde 2006). Numerous studies have shown that females modify their breeding decisions after pairing according to male attractiveness (e.g., Burley 1986; Gil et al. 1999; Limbourg et al. 2004; Velando et al. 2006; Helfenstein et al. 2008). The cues used by kittiwake females to evaluate their mate's condition remain unknown. Females may have adaptively decreased their feeding frequency in reaction to their mate's phenotypic change (e.g., body condition or color) or sudden lower nest attendance (Figure 4) signaling a corresponding decrease in their foraging ability.

The second hypothesis considers that the low feeding rate of females mated with handicapped males was a consequence of their own higher nest attendance resulting from their mate's longer foraging trips. Because young chicks need to be continuously brooded (Bech et al. 1984), females mated with handicapped males had to compensate for their mate's inability to attend the nest. Females may have needed several days to adjust their feeding effort to their increased nest attendance, and this may have led to lower feeding rates in the days after our manipulation. Although they did not suffer greater weight loss (Figure 2), had similar carotenoid-based integument coloration (Figure 3), and had similar levels of corticosterone and prolactin (Table 1) to control females, females mated with handicapped males may have incurred an extra reproductive cost.

CONCLUSION

Contrary to our prediction, handicapped males showed a loss of body mass and a fixed level of parental care, despite the potential energetic stress of the handicap as substantiated by the duller carotenoid-based coloration. Individual and environmental conditions are known to affect reproductive decisions (McNamara and Houston 1996; Wendeln and Becker 1999; Erikstad et al. 2009). When food is abundant, parents can easily compensate for increased chick requirements, whereas food shortage may render them unable to do so (Erikstad et al. 1997; Erikstad et al. 1998; Velando and Alonso-Alvárez 2003). Our behavioral observations were carried out during an exceptionally favorable breeding season as revealed by the unusually high indices of breeding success. Handicapped males in poorer environmental conditions might have been unable or unwilling to maintain their feeding effort at rates similar to those of controls and the effects of the handicap might have been different. Similar experiments in different breeding conditions are needed to understand how this species optimizes its lifetime reproductive success.

Contrary to males, females showed flexibility in their level of parental care. Our experimental design did not allow us to determine whether their lower feeding rate was adaptively adjusted to the apparent low quality of their mate or whether it was constrained by their higher nest attendance. Manipulation of male sexual secondary traits would be needed to distinguish between those 2 possibilities.

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