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Corticosterone alone does not trigger a short term behavioural shift in incubating female common eiders *Somateria mollissima*, but does modify long term reproductive success

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The trade-off between reproductive effort and adult survival in birds is modulated by several factors. Corticosterone and prolactin have additive effects on reproductive behaviour by stimulating foraging and parental behaviours, respectively. When incubation is associated with fasting, nest desertion is supposed to be activated by an unknown refeeding signal when body condition becomes critically deteriorated. The concomitant rise in corticosterone levels has been suggested to be the triggering factor. We tested the role of corticosterone on reproductive success by observing the effect of corticosterone implants on reproductive success and on plasma prolactin concentration in female common eiders *Somateria mollissima*. Implanted females showed a significant increase in corticosterone and a decrease in prolactin levels. Despite their enhanced daily body mass loss, females did not abandon incubation nor did they start to refeed in the four days following implantation. These data show that the experimentally induced rise in plasma corticosterone concentration alone does not trigger nest desertion. However, after 25 days of incubation, implanted females displayed a higher rate of egg loss, suggesting lower nest attentiveness towards the end of incubation. We suggest that the short-term effects of corticosterone may be dependent on the energy state of the bird. However, the late-induced change in reproductive success is indirectly linked to corticosterone, and we suggest that either a prolactin decrease, or a depletion in protein body reserves, may participate in the long-term adjustment of incubation behaviour in female eiders.

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According to life history theory, long-lived birds should enhance their own survival chances by deserting the nest rather than continuing reproduction when breeding is energetically too expensive (Stearns 1992). In several species, reproduction is associated with fasting because foraging competes with incubation (Cherel et al. 1988). At the end of fasting, birds enter a critical phase, in which body lipid content (the principal form of body reserves) nears exhaustion, causing nest desertion due to increased foraging activities (Robin et al. 1998). This shift in behaviour, concomitant with changes in body reserves mobilisation, leads one to look for a relationship between parental decisions and hormonal changes. Corticosterone, the main avian glucocorticoid (Holms and Phillips 1976), increases the mobilisation of body proteins in fasting birds (Cherel et al. 1988), and

promotes behaviour away from energetically costly activities such as reproduction (Wingfield 1988, Astheimer et al. 1995). These studies highlight the fact that corticosterone's impact on food intake and behaviour is dependent on the physiological state of the bird, e.g. fed birds do not respond to exogenous corticosterone whereas fasted ones showed escape behaviour and higher feeding rates (Gray et al. 1990, Astheimer et al. 1992). However, the degree to which corticosterone is involved in making the nest abandonment decision when incubation is associated with a natural long-term fast remained to be determined.

During incubation, corticosterone is not the only factor that modulates parental decisions. Prolactin has been suggested to be the main hormone stimulating incubating behaviour in birds (Buntin 1996), and an experimental decrease in prolactin levels induced nest desertion in the penguin *Aptenodytes patagonicus* and the eider *Somateria mollissima* (Cherel et al. 1994, Criscuolo et al. unpubl. data). Based on these findings, it is conceivable that corticosterone could cause a decrease in nest attentiveness of eiders via an inhibitory effect on prolactinemia. Indeed, captive penguins fasting too long exhibited a concomitant increase in corticosterone and a decrease in prolactin when reaching the critical phase III of fasting (i.e. when body lipids neared exhaustion, Cherel et al. 1994). Thus, it appears that corticosterone and prolactin could modify life history decisions by redirecting animal activities towards adult survival when incubation is too costly.

The long-lived female common eider fasts during the 24–26 days of incubation (Korschgen 1977), and loses 30–40% of its body weight (Parker and Holm 1990, Gabrielsen et al. 1991). However, female eiders maintain their body mass above a minimal threshold (1.4–1.3 kg) that does not compromise their survival chances. This minimal body mass threshold is achieved by increased foraging behaviour which occurs at the expense of an increased risk of egg predation (Criscuolo et al. 2002a). By following the reproductive success of female eiders implanted with corticosterone, the present study was performed to assess two questions: (i) can egg predation rate be modified when incubating female eiders are exposed to exogenous corticosterone, and (ii) are prolactin levels decreased after corticosterone implantation in incubating eiders.

Methods

The study was conducted in June 2001 in Kongsfjorden, on the west coast of the Svalbard Archipelago (78°55 N, 12°07′ E), in a colony of about 200 common eiders nests on Prins Heinrich Island.

Blood sampling and body measurements

Capture of birds, blood samples (within 3 min), clutch size, body mass (± 2 g) and body size (head-bill length principally), were recorded as previously described (Criscuolo 2001, 2002b). No nest desertions were observed immediately after bird manipulation. Four days after implantation, the females were caught again and blood samples were collected within 3 min. Finally, nest desertion, clutch size and body weight were recorded again.

Corticosterone implantation

Silastic tubes sealed at each end (one per bird, i.d. 1.47 mm, length 25 mm) and filled with 80–90 mg crystalline corticosterone were used for the constitution of the corticosterone implants (see Silverin, 1986). Such a dose was used to induce an increase in plasma corticosterone similar to birds close to the critical phase of fasting (increased proteolysis; Cherel et al. 1988), and to test its effect on prolactinemia. Implantation was performed in the nape of the neck. After the skin in the area had been shaved, and disinfected with alcohol and betadine (iodine solution), a small incision was made and the implant was placed under the skin. The skin was then closed with one or two stitches before it was cleaned again with betadine and sprayed with an aluminium powder.

Experimental groups

A total of 29 females were used in the study. *Captive birds.* Two groups of captive females (A and A2) were used: group A ($n = 5$) consisted of females implanted with 80–90 mg of corticosterone (see above), and held in captivity for five days. This group of captive females was used to determine the effect of the implants on day-to-day plasma concentration of corticosterone, and to verify whether the induced rise was physiologically relevant. These females were caught on the nest one week after the start of incubation. They were placed in cages without food but with water *ad libitum,* and were implanted with corticosterone the same day. Blood samples were collected every day. The second group (A2, $n = 5$) consisted of captive females used to compare the plasma concentrations of corticosterone and metabolites between the implanted groups and females reaching the final phase III of fasting. These birds were caught on the nest during the first week of incubation and kept in captivity, under the same conditions as the group A, until the end of fasting (when body lipids reached near exhaustion). Blood samples were taken every 4–8 days. Birds were fed and released at the end of the experiment. *Free living birds.*

Free living incubating female birds were separated into two groups (C and B): (1) Control incubating females (C) implanted with empty silastic tubes (n = 8), and (2) exogenous corticosterone incubating females (B) implanted with 80–90 mg of corticosterone (n = 11). Corticosterone implantation was done within the first ten days of incubation. Since we were unable to catch and sample all the birds twice (before and after implantation), our final sample size for plasma comparisons consisted of six birds in the C group and eight birds in the B group. Birds were sampled 4 days after implantation (based on previous experiments showing that this was the last day with an elevated level of corticosterone). For these two groups, the rate of nest predation (defined by the number of eggs/ducklings remaining in the nest four days after implantation, and at the hatching or piping day), and the proportion of reproductive success (females having at least one egg hatching after 25 days of incubation) were determined. Because birds were under natural continuous daylight during the experiment, circadian rhythms in the plasma concentrations of these two hormones is likely to be insignificant (Vleck and van Hook 2002).

Plasma corticosterone, prolactin and metabolite assays

The concentrations of corticosterone were determined by a sole radioimmunoassay analysis at the CEPE using a $_{125}$I RIA double antibody kit from ICN Biomedicals. The radioimmunoassays for the measurement of prolactin concentrations were done at the CEBC using a specific avian antibody as previously reported (Mauget et al. 1995, Lormée et al. 1999, Lormée et al. 2000, Criscuolo et al. 2002b). Plasma levels of uric acid (a product of protein catabolism), triacylglycerols (which increase in fed birds), glucose and free fatty acids (a product of lipolysis) were measured at the CEPE by colorimetry using a commercial kit (Sigma).

Statistical analysis

Modifications of body mass loss and plasma concentrations of corticosterone and prolactin among female eiders were compared using an ANOVA for repeated measurements and a Tukey test for posthoc analysis, after testing for normality of the data (Shapiro-Wilk test, $P > 0.01$). The proportion of reproductive success in the two groups of incubating females and the proportion of eggs/ducklings present in the nest at each capture and at the hatching stage was tested with a χ^2 test. Values are means ± standard error (SE). Among others body size measurements, head-bill length was the best body mass predictor and explains 24% of the initial and 35% of the final body mass variations ($P < 0.005$). We used a body condition index obtained by computing residuals of a regression analysis between body mass and head-bill length.

Results

Daily body mass loss

Clutch size, body mass and body condition before implantation did not differ significantly between the C and B groups of incubating females (ANOVA, $P = 0.48$). Initial clutch size was 3.4 ± 0.5 (C) and 3.5 ± 1.1 (B) eggs, while initial body mass ranged from about 1702 ± 165 g to 1816 ± 97 g respectively. The rate of body mass loss differed significantly between the two experimental groups of incubating females. Four days after the corticosterone implantation, females from group B showed a higher daily body mass loss than group C females (ANOVA, $P = 0.002$).

The initial body mass of captive group A females was 1690 ± 83 g and decreased to 1380 ± 45 g after seven days of fasting in captivity. The mean body mass loss of these females was 31.6 ± 3.1 g/day four days after the corticosterone implantation (data not shown). The initial body mass of captive females of group A2 was about 1800 g (Table 1). These birds entered the critical phase of fasting after 24.4 ± 1.2 days of fasting as shown by their

Table 1. Body mass, duration of fasting and plasma levels of corticosterone and metabolites of captive female eiders of the group A2 measured at four periods of captivity: beginning of the experiment (initial), during fasting, just before entering the critical phase of fasting and after one day into the critical phase of fasting, compared with the same parameters measured in B females after corticosterone implantation.

Group A2 of captive females	Initial	Fasting	Before the critical phase	Critical phase of fasting (1)	B females (2)	Difference between phases (1) and (2)
Body mass (g)	1787 ± 67	1460 ± 34	1229 ± 41	1020 ± 47	1519 ± 151	$P < 0.05$
Fasting day	4.4 ± 1.0	12.4 ± 2.2	19.2 ± 1.2	24.4 ± 1.2	15.3 ± 1.8	$P < 0.05$
Corticosterone (ng/ml)	29.5 ± 6.5	30.1 ± 10.8	45.0 ± 28.2	87.1 ± 16.8	72.2 ± 22.5	$P = 0.22$
Uric acid (mmol/l)	0.20 ± 0.01	0.12 ± 0.02	0.25 ± 0.06	0.75 ± 0.10	0.21 ± 0.03	$P < 0.05$

Values are means ± SE. Differences between the critical phase of fasting (1) and B females (2) were tested for with an ANOVA.

accelerated daily body mass loss (see Criscuolo et al. 2002a), their body mass reaching about 1000 g.

Reproductive success

We measured the reproductive success in two separate ways: the percentage of females that had at least one piping egg after 25 days of incubation (reproductive success), and the proportion of eggs (piping or not) left in the nest 4 days after implantation, and at the end of the experiment. Reproductive success measurements were not affected equally by the corticosterone implant. Indeed, the proportion of females having at least one piping egg after 25 days of incubation was 75.0% in the C group and 45.5% in the B group, thus showing no significant difference when plasma levels of corticosterone increased ($n=19$, χ^2 test, $P=0.098$). A similar conclusion was deduced from the rate of nest predation 4 days after implantation, the proportion of eggs in the nest being 74.1% in the C group and 81.6% in the B group ($n=19$, χ^2 test, $P=0.54$). However, after 25 days of incubation, this proportion was 70.4% and 36.8% in groups C and B, respectively. At the end of incubation, implanted females in the B group exhibited a higher rate of egg predation than control females ($\chi^2=7.25$, $n=19$, $P=0.007$).

Plasma corticosterone, prolactin, and uric acid levels

The changes in plasma corticosterone concentrations of captive females (group A) are shown in Fig. 1. Corticosterone increased sharply and reached a maximum the first day after implantation and then returned close to initial values at day 5 (ANOVA, $n=5$, $P=0.032$). In a preliminary study conducted on captive female eiders, we verified that corticosterone levels did not increase without implantation in eiders held in the same captive and stressful conditions (day 1, 20.4 ± 2.2 ng/ml; day 4, 30.0 ± 6.9 ng/ml; day 15, 20.6 ± 2.5 ng/ml; day 20, 23.7 ± 7.3 ng/ml, $n=5$, ANOVA, $P=0.62$). Data for the plasma corticosterone and uric acid concentrations (A2 and B groups) are presented in Table 1. Corticosterone levels in captive females observed at the end of fasting were comparable to those measured in group B females (ANOVA, $P=0.22$). The plasma concentration of uric acid was lower in females from group B compared to captive group A2 females sampled at the end of fasting (0.21 ± 0.03 vs. 0.75 ± 0.10 mmol/l, ANOVA, $P<0.01$). Uric acid level was comparable to the level of captive females in group A2 sampled in the middle phase of fasting (0.21 ± 0.03 vs. 0.25 ± 0.06 mmol/l, ANOVA, $P=0.74$).

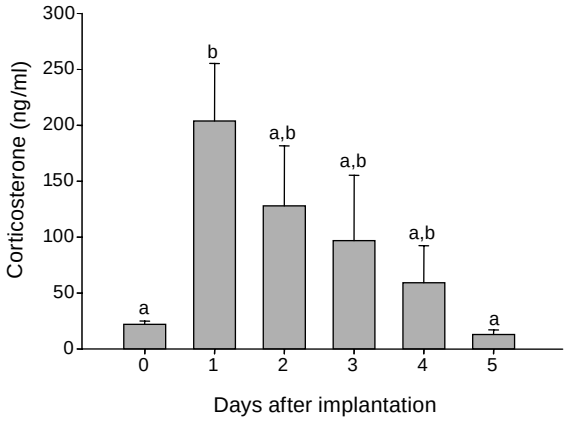

Fig. 1. Plasma concentration of corticosterone in female eiders ($n=5$) caught on the nest (day 0) and after being kept in captivity for five days after high-dose corticosterone implantation. Different letters indicates significant difference (Tukey test, $P<0.045$). Values are means $\pm$ SE.

Corticosterone levels did not differ between group B (19.4 ± 2.7 ng/ml) and group C (14.8 ± 3.1 ng/ml) at the onset of the experiment (ANOVA, $P=0.282$, Fig. 2A). Four days after corticosterone implantation, females from group B presented levels of plasma corticosterone significantly higher than females from group C (72.2 ± 22.5 vs. 15.0 ± 2.9 ng/ml, ANOVA, $P=0.05$).

Initial plasma prolactin concentrations were not different in incubating females (groups B and C) before corticosterone implantation and were around 50 ng/ml (ANOVA, $P=0.412$, Fig. 2B). Four days after corticosterone implantation, group B females exhibited a lower final prolactin level (40.8 ± 0.7 ng/ml), compared with their initial values (47.6 ± 0.7 ng/ml, ANOVA, $P<0.001$). These group B prolactin levels were also significantly lower than that of group C females (ANOVA, $P<0.001$) which were not modified compared to initial levels (48.5 ± 0.8 and 48.6 ± 1.0 ng/ml, respectively, ANOVA, $P=0.621$).

Plasma levels of uric acid in the incubating groups B and C were similar before implantation (range 0.09–0.10 mmol/l, ANOVA, $P=0.282$), but markedly increased in group B females after implantation (0.21 ± 0.03 mmol/l, ANOVA, $P=0.002$, Fig. 2C). However, the uric acid concentration in incubating females was not as high as that found in captive females sampled during the critical phase of fasting (see Table 1). Exogenous corticosterone had no effect on plasma triacylglycerol levels, comparables in all groups (range 0.50–0.55 g/l, ANOVA, $P=0.779$, Fig. 3). Plasma concentrations of free fatty acids (Fig. 3) were similar at the beginning of the experiment (mean range 0.31 ± 0.04–0.38 ± 0.07 mmol/l), but decreased significantly in B females 4 days after corticosterone implantation (0.15 ± 0.06 mmol/l, ANOVA, $P=0.01$).

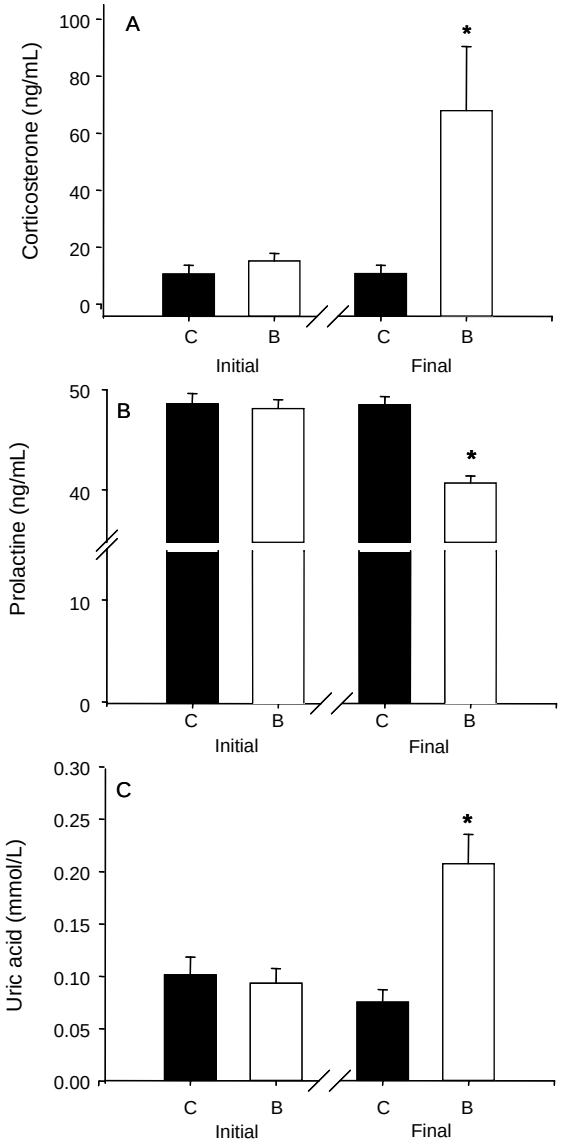

Fig. 2. 2A: Plasma concentrations of corticosterone of the controls (C, n = 6) and the corticosterone-implanted (B, n = 8) groups of incubating females, before and four days after implantation. * indicates significant difference ($P < 0.05$). Values are means ± SE. 2B: Plasma concentrations of prolactin of the controls (C, n = 6) and the corticosterone-implanted (B, n = 8) groups of incubating females, before and four days after implantation. * indicates significant difference ($P < 0.05$). Values are means ± SE. 2C: Plasma concentrations of uric acid of the controls (C, n = 6) and the corticosterone-implanted (B, n = 8) groups of incubating females, before and four days after implantations. * indicates significant difference ($P < 0.05$). Values are means ± SE.

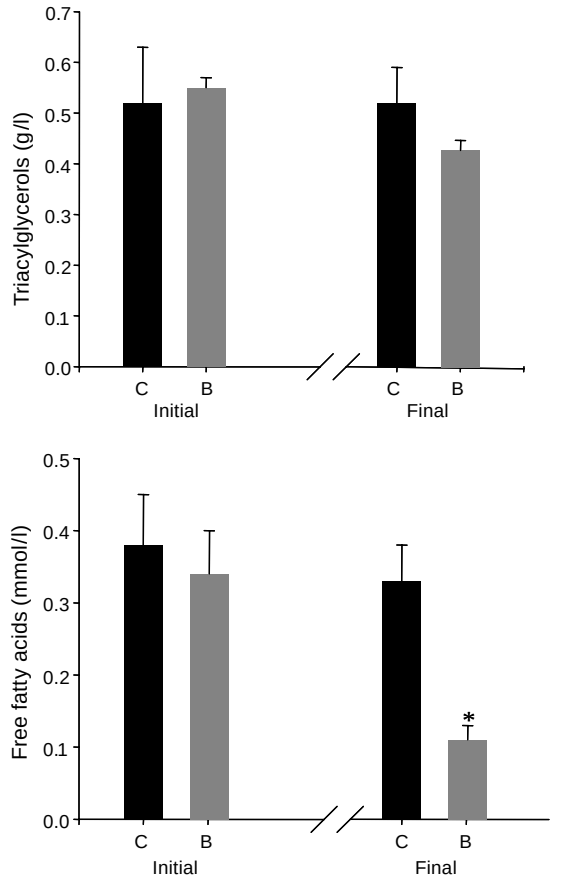

Fig. 3. Plasma concentrations of triacylglycerols of the control (C) and corticosterone (B) groups, before and after implantations. Plasma concentrations of free fatty acids of the control (C) and corticosterone (B) groups, before and 4 days after implantations. * indicates significant differences ($P < 0.05$). Values are means ± SE (n = 28).

Discussion

In an earlier study, we showed that female common eiders modified their incubation behaviour by lowering nest attendance when their body mass reached a critical threshold (Criscuolo et al. 2002a). The common hypothesis is that when body lipids neared exhaustion, the associated rise in corticosterone triggers refeeding (Robin et al. 1998). Our results showed that increased corticosterone levels alone are not associated with a shift in behaviour, despite the fact that high corticosterone levels are associated with a decreased plasma prolactin concentration. However, a lower nest attendance was observed later in incubation, whereas corticosterone levels were returned to basal levels. Therefore, corticosterone short and long-term effects, or behavioural and metabolic effects, have to be distinguished.

Corticosterone, prolactin and egg predation

The present experiment clearly shows that while group B females did not suffer from increased clutch predation during the first four days after implantation, they did so after 25 days, thereby becoming less attentive to their nest later in incubation. This finding raises the question

of which physiological processes can explain: (1) the lack of a short-term effect, and (2) the presence of a long-term effect of corticosterone on female eider behaviour.

With regard to short-term effects, corticosterone is known to trigger different responses depending on its concentration. According to Wingfield et al. (1998), intermediate corticosterone levels increase foraging, while high levels may restrict movement. In white-crowned sparrows *Zonotrichia leucophrys gambelii*, previous research has shown that only intermediate levels of corticosterone caused an increase in perch hopping (Breuner et al. 1998). This may explain why we were unable to detect any effect the first four days after implantation, when corticosterone was at its highest level. Even if food intake in birds seems to be lipid and not carbohydrate dependent (Boswell et al. 1997), the corticosterone-induced twofold increase in protein catabolism (Fig. 2C) and the corticosterone-induced high glycemia (Hazelwood 1965, Wingfield et al. 1998), may promote high nest attentiveness despite a subnormal corticosteronemia. Additionally, several studies emphasised that corticosterone influences locomotor or feeding behaviour only when birds were placed under energetic stress (Gray et al. 1990, Astheimer et al. 1992, Kitaysky et al. 1999). However, the negative energy balance faced during incubation does not seem to be a stressful event for female eiders since these birds are adapted to fasting (Korschgen 1977). Therefore, corticosterone and/or prolactin changes do not act at this moment like an emergency signal, as was illustrated by the absence of change in triacylglycerol levels, an indicator of feeding activity. Prolactin levels were also lowered four days after corticosterone implantation. This emphasizes the hypothesis that corticosterone can regulate prolactin secretion, in order to control parental care. However, in the short term, no increase in egg predation rate or nest desertion can be associated with the lower prolactin levels. Therefore, prolactin may be implicated in a more long-term regulation of incubation behaviour. Indeed, delayed effects of acute high corticosterone levels, such as the unusual protein utilisation early in incubation (higher levels of uric acid, Fig. 2C), might have drained a non-negligible amount of the female body protein reserve. In combination with lower serum prolactin levels, this corticosterone-induced proteolysis at the early stage of incubation could have caused the females to be less attentive to their nest towards the end of incubation.

In conclusion, peripheral action of corticosterone on the protein pool may act differentially or in synergy with its central action on behaviour. Orexigenic effects on the brain receptors of birds induced by glucocorticoids were recently demonstrated in ring doves *Streptopelia risoria* (Koch et al. 2002). The interaction between prolactin and corticosterone gives us the opportunity of exploring the hypothesis of a central modulation of parental behaviour by corticosterone. The existence of a glucocorticoid receptor found in the amphibian brain that, once activated, disrupts reproductive behaviour directly (Orchinik et al. 1991), supports such a theory, as does the finding of a nongenomic action of corticosterone on behaviour (Breuner et al. 1998).

Corticosterone and the unknown refeeding signal

The main difference between B females and non-incubating captive eiders reaching phase III of fasting was the limited rise of uric acid, implanted females having a level similar to captive females at the end of phase II (Table 1). Protein catabolism after corticosterone implantation was not as high as in phase III, leading to the hypothesis that increased proteolysis could be an important factor in triggering refeeding in birds. Exogenous corticosterone also induced a reduction of the plasma concentration of free fatty acids, suggesting a direct role for this hormone in the control of lipolysis. This data is in accord with the effect of high corticosterone levels on fat accumulation in birds (Gray et al. 1990). The data also suggest that the decreased level of free fatty acids observed in phase III of fasting could be corticosterone dependent, and not due to a limitation of the production of free fatty acids by the adipose tissue. In fact, body lipids are not exhausted in phase III (Cherel et al. 1994). Thus in phase III, metabolic mechanisms other than corticosterone rise must play a key role to induce changes in incubation behaviour in fasting birds, and corticosterone alone is not sufficient to cause nest desertion. Other factors linked with lipid metabolism such as leptin may be involved in the stimulation of refeeding.

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