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Modulation of the prolactin and the corticosterone stress responses: Do they tell the same story in a long-lived bird, the Cape petrel?

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ABSTRACT

Over the last decades, the corticosterone stress response has been suggested as a major physiological tool to understand what strategy an individual might adopt in response to environmental perturbations. More recently, another hormone related to parental care – prolactin – has been suggested as a complementary tool to investigate this question. Indeed, both of these hormones are affected by stressors and are involved in parental decisions, such as deserting the nest. Because of these similarities, it remains unclear what the functional distinction between the prolactin and corticosterone stress responses is. Here, we investigated whether natural variations of the corticosterone and prolactin stress responses are functionally linked in free-living Cape petrel (*Daption capense*) parents. If prolactin and corticosterone mediate the same functional response to a stressor and are the proxies of the same response, we predict that corticosterone and prolactin stress responses (1) will be modulated according to the same factors; (2) will affect reproductive performances in the same way; and, (3) of course, will be correlated. Contrary to these predictions, we found that the corticosterone and prolactin stress responses were respectively modulated according to body condition and breeding status. Moreover, prolactin levels, but not corticosterone levels, were related to hatching success in this species. Finally, we did not find any significant correlation between these two stress responses under any circumstances (failed breeders, incubating or chick rearing birds) and this result was overall supported by a review of the existing literature. Therefore, these two stress responses do not seem to be tightly linked and we believe that they may provide complementary pieces of information on parental investment in birds.

1. Introduction

Over their annual cycle, wild vertebrates have to face numerous unpredictable and sudden perturbations that have the potential to jeopardize their immediate survival [68,72,73]. To cope with these perturbations, vertebrates have evolved mechanisms that allow the adjustment of an individual's physiology and behavior to the new situation (the concept of allostasis – stability through change – [44,51]). Specifically, these adjustments redirect the available resources – time and energy – from facultative activities (e.g. reproduction, territoriality) towards physiology and behavior that benefit immediate survival [2,70,72]. Despite this obvious benefit in term of survival, these adjustments can however entail important fitness costs (e.g. suppression of reproduction, loss of territory) and there are situations during the life cycle when these adjustments may therefore have a low adaptive value. In this case, these mechanisms related to allostasis are down-regulated in order to balance the costs and benefits associated with the activation of allostasis ("perturbation resistance potential", [73]).

In this eco-physiological framework, one of the principal mediators of allostasis, the glucocorticosteroid hormones (corticosterone in birds and cortisol in most mammals), has received much attention during the last decades [37,50,52]. In response to stress, corticosterone levels increase and this mediates important behavioral and physiological changes that are thought to benefit to immediate survival [7,16,72]. Importantly, the corticosterone stress response has been shown to be down-regulated when such modulation provides fitness benefits to individuals [1,28,32,34,39–41,47,60]. Therefore, the corticosterone stress response has been suggested as a major physiological tool to understand what strategy an individual might adopt in response to perturbations [12,31,70,71,73].

Recently, it has been suggested that another hormone – prolactin may also be crucial to understand what strategies parents adopt when they have to cope with unpredictable life-threatening events [2,59]. Indeed, elevated prolactin levels appear necessary to maintain parental behaviors and low prolactin levels are often related to breeding failure [2,48,54,56,59] and to the subsequent initiation of molt in bird species [24,26,27]. Importantly, prolactin is also

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known to be affected by stress in many bird species as is corticosterone. In response to a standardized stress protocol, prolactin levels decreased in bird parents and this decrease may explain the disruption of parental care and, thus, breeding desertion [2,4,59]. A few studies have shown that when the prolactin stress response has a low adaptive value it can also be down-regulated [2,4,8,19,32,54].

Despite the role of these two hormones in mediating the stress response of parental birds, it remains unclear what the functional distinction between the prolactin and corticosterone stress responses is. The regulation of these two hormones seem to be connected under some but not all circumstances [5,8,19,29,35,48,53]. For instance, Angelier et al. [9] recently demonstrated that an experimental increase of corticosterone levels resulted in a decrease in prolactin levels in incubating black-legged kittiwakes (*Rissa tridactyla*). On the other hand, some studies suggested that prolactin can promote corticosterone secretion in birds [35,45]. Thus, Miller et al. [45] showed that circulating corticosterone levels are affected by plasma prolactin concentrations in mourning doves (*Zenaida macroura*). In addition, other studies have reported that these two stress responses can be disconnected [8,19,32,53]. Because of this inconsistent link between corticosterone and prolactin, it remains difficult to understand whether the corticosterone and prolactin stress responses provide the same information in parent birds or not. Indeed, the prolactin stress response is rarely examined in breeding birds and, therefore, we currently lack data to understand the link between these two stress responses in free-living birds [2]. Understanding this link is, however, of importance for eco-physiologists because it may be crucial to examine the prolactin stress response in parental vertebrates if it provides additional information to the corticosterone stress response.

In this study, we investigated whether natural variations of the corticosterone and prolactin stress responses are functionally linked in wild birds during the parental phase. To do so, we examined both of these stress responses in free-living Cape petrel parents (*Daption capense*). If prolactin and corticosterone mediate the same functional response to a stressor and are the proxies of the same response (hypothesis 1), we predict that corticosterone and prolactin levels will be correlated. In this case, we also predict that the corticosterone and prolactin stress responses will be modulated according to the same factors (e.g. body condition, breeding status). If the prolactin and corticosterone responses to stress are at least partially independent and do not mediate exactly the same functional response to a stressor (hypothesis 2), we predict that corticosterone and prolactin levels will not be correlated. In this case, we also predict that these stress responses will not necessarily be modulated according to the same factors (e.g. body condition, breeding status). Importantly, previous studies have reported that corticosterone and prolactin levels can be correlated under some but not all circumstances [2,4,5,8,19,32,34,45,53], suggesting that the ecological or physiological context may affect the relationship between these two hormones. To better understand this discrepancy between studies, we tested our hypotheses in Cape petrels, which were engaged in different parental activities (incubating birds, brooding birds, and failed breeders). Therefore, we were able to test the link between corticosterone and prolactin levels for individuals that were not in the same parental state. Finally, we also tested whether corticosterone and prolactin levels were related to reproductive performance in Cape petrels (i.e. hatching success). If one hormonal stress response but not the other predicts hatching success, this will support the idea that these two stress responses are at least partially independent and should be investigated together to examine the physiological link between environmental conditions and performance hypothesis 2).

2. Materials and methods

2.1. Study site and species

Cape petrels were studied on Pointe Géologie archipelago, Adélie Land (66°40′S, 140°01′E), Antarctica during the austral summer (January 2010). Cape petrels are long-lived birds with high survival probability and low fecundity. Sexual maturity is acquired at 7 year of age, on average, and birds breed annually. During the incubation and brooding periods, both parents alternate incubating and brooding the single egg/chick and males and females equally share the parental duties. Once the chick becomes thermally emancipated and large enough to defend itself against predators, both parents simultaneously forage at sea to feed their chick and restore their own body reserves [63,64]. As previously recorded at other breeding sites [65], most of breeding failures are the result of egg or chick predation by South polar skuas (*Catharacta maccormicki*). Importantly, Cape petrels still visit their nests after having lost their egg [62] making this species suitable to investigate the physiology of failed breeders.

2.2. Capture, blood sampling and measures

This study was carried out during the austral summer (from January 5 to February 7 2011). During this period, 55 Cape petrels were captured while attending their nest (15 incubating birds, 27 brooding birds and 13 failed breeders). Failed breeders were captured while visiting their nest after egg predation occurred. Accurate time of egg loss was not known for these birds. All these Cape petrels were bled according to the standardized technique described by Wingfield [67]. Immediately after capture, an initial blood sample was collected from the alar vein with a 1-mL heparinized syringe and a 25-gauge needle. All these initial blood samples were collected within 3 min of capture and we did not find any effect of bleeding time on corticosterone levels or prolactin levels. Therefore, these initial blood samples were considered to reflect baseline levels of corticosterone and prolactin ([19,51], thereafter called 'baseline' sample). A second sample was then taken 30 min after the first (thereafter called 'stress induced' sample). Cape petrels were kept in individual opaque cloth bags between the initial and second blood samples. All petrels were weighed to the nearest 2 g with a Pesola scale. Their skull length (head + bill), tarsus length, culmen length and wing length were measured to the nearest 0.5 mm. During handling of a parent, its egg or chick was kept warm in a small box. At the end of the sampling process, petrels were immediately returned to the nest with their egg or chick. In addition, hatching success was monitored for incubating petrels by following their nests until predation or hatching occurred. In other words, we sampled (1) failed breeders that had already lost their egg at the time of capture, (2) incubating petrels that were then successful in hatching their egg, (3) incubating petrels that were not successful in hatching their egg and (4) brooding birds. Fledging success was not considered in this study because only a very low percentage of chicks fail to fledge: most breeding failures occur during the incubation stage [65].

2.3. Molecular sexing and hormone assays

Blood samples were centrifuged (3500 rpm for 10 min), and plasma and red cells were stored at −20 °C until laboratory analyses (hormones assays and molecular sexing). The sex was determined by polymerase chain reaction amplification of part of two CHD genes present on the sex chromosomes at the Centre d'Etudes Biologiques de Chizé (CEBC), as detailed in Angelier et al. [5]. Plasma concentrations of corticosterone were determined

by radioimmunoassay at the CEBC (see [42] for details). Plasma concentrations of prolactin were determined by a heterologous radioimmunoassay at the CEBC (see [3]). Pooled plasma samples of Cape petrels produced a dose–response curve that paralleled chicken prolactin standard curves (Fig. 1). All samples were run in one assay for both hormones (intra-assay variation; corticosterone: 7.1%, prolactin: 4.8%). Minimal detectable prolactin level was 0.5 ng/ml. All blood samples were collected between 8:00 and 18:00 and we did not find any diurnal rhythm of prolactin and corticosterone levels in this species (GLM, $p > 0.500$ for all groups).

2.4. Statistical analyses

All analyses were performed with SAS statistical software (SAS Institute, v. 9.2.). For all analyses, only one partner of each nest was studied, so that data obtained from male and female Cape petrels were considered independent samples. For all variables tested, normality assumptions we checked by plotting the residuals against the predicted value and by running the Shapiro–Wilk test for normality. We performed all our model selection starting from the most general model that included all the variables/factors and their interactions and we suppressed step by step the non significant interactions, variables or factors. First, we built two sets of models (one per hormone) to explain corticosterone and prolactin levels. Specifically, we used mixed models ('proc mixed', SAS) with 'breeding status' (3-classes factor: 'failed breeder', 'incubating' and 'brooding'), 'sex' (2-classes factor: 'male' and 'female') and body condition (continuous variable) as explanatory factors/variables and 'sample' as the repeated explanatory factor (2-classes factor: 'baseline' and 'stress induced'). Then, we used contrasts to test specific hypothesis regarding the selected model ('contrast statement', SAS). Second, we investigated the link that may exist between prolactin and corticosterone levels. We used mixed models to explain prolactin levels ('proc mixed', SAS) with 'breeding status' (3-classes factor: 'failed breeder', 'incubating' and 'brooding'), 'sex' (2-classes factor: 'male' and 'female') and 'corticosterone levels' (continuous variable) as explanatory factors/variables and 'sample' as the repeated explanatory factor (2-classes factor: 'baseline' and 'stress induced'). Although we did not find any significant effect of corticosterone levels or any interaction including corticosterone levels, there is an increased risk of committing type 1 errors when running such complex models with several interactions and, therefore, it is possible that we failed to detect a significant biological relationship between corticosterone levels and prolactin levels for a specific group. To minimize this risk, we also ran correlations ('proc corr', SAS) between corticosterone levels and prolactin levels for each sex ('male' and 'female'), breeding status ('failed breeder', 'incubating' and 'brooding') and sample ('baseline' and 'stress induced'). This approach usually increases the risk of committing type 2 errors but, in our case, it allowed us confirming that we did not fail to detect a significant biological relationship between corticosterone and prolactin levels for a specific group. Finally, we tested for incubating birds ($n = 15$) whether prolactin and corticosterone levels of successful birds differed from those of the birds that lost their egg. We built two sets of models (one per hormone) to explain corticosterone and prolactin levels. Specifically, we used mixed models ('proc mixed', SAS) with 'hatching success' (2-classes factor: 'success' and 'failure'), 'sex' (2-classes factor: 'male' and 'female') as explanatory factors/variables and 'sample' as the repeated explanatory factor (2-classes factor: 'baseline' and 'stress induced').

3. Results

3.1. The corticosterone stress response

Cape petrels responded to the standardized stress protocol with a large and rapid increase in plasma corticosterone levels ($F_{1,53} = 982.69$, $p < 0.001$; Fig. 2). This increase in corticosterone levels was not affected by sex ($F_{1,50} = 0.18$, $p = 0.677$; Fig. 2), breeding status ($F_{2,51} = 0.76$, $p = 0.474$; Fig. 2) or any interaction ($p > 0.750$ for all interactions; Fig. 2). However, the increase in corticosterone levels was correlated with body condition ($F_{1,53} = 11.15$, $p = 0.001$; Fig. 3): the increase in corticosterone levels of individuals in poor body condition was greater than that of individuals in good body condition.

In addition, corticosterone levels were negatively correlated with body condition ($F_{1,52} = 15.67$, $p < 0.001$), but were not affected by sex ($F_{1,50} = 0.01$, $p = 0.935$), breeding status ($F_{2,51} = 0.36$, $p = 0.700$) or any interaction ($p > 0.900$ for all interactions). Specifically, birds in poor condition had higher baseline corticosterone levels than birds in good condition (baseline: $F_{1,53} = 3.90$,

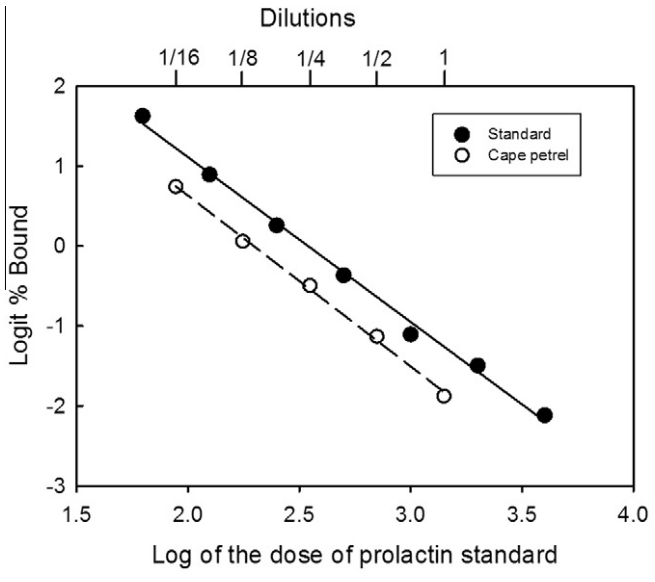

Fig. 1. Dose response curve for standard and Cape petrel prolactin.

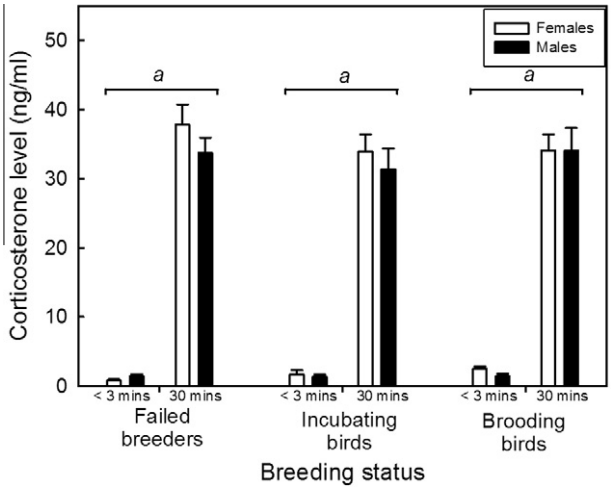

Fig. 2. Corticosterone stress response of Cape petrels engaged in different parental activities (failed breeders, incubating birds, brooding birds). Letters above bars denote statistical results of stress response comparisons between the different groups. Different letters denote statistical significance between two groups. The corticosterone stress response and absolute levels of corticosterone did not differ between any groups. Data are expressed as means + standard error.

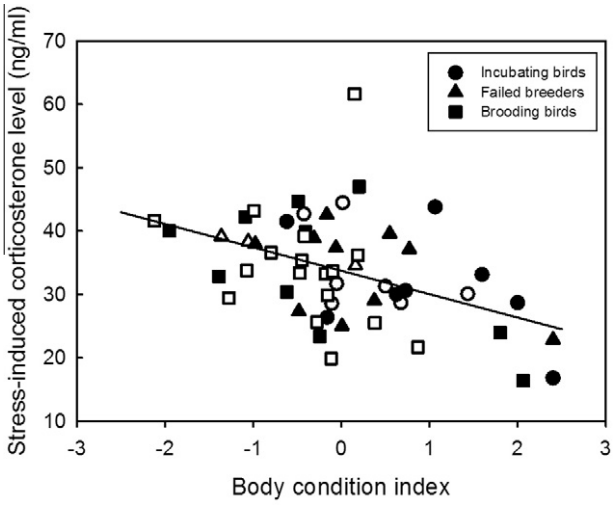

Fig. 3. Negative relationship between stress-induced corticosterone levels and body condition in Cape petrels that were engaged in different parental activities (failed breeders, incubating birds, brooding birds). Open and filled symbols respectively denote females and males.

$p = 0.054$, Fig. 3). In addition, birds in poor condition had higher stress-induced corticosterone levels than birds in good condition (stress-induced: $F_{1,53} = 13.65$, $p < 0.001$; Fig. 3).

3.2. The prolactin stress response

Cape petrels responded to the standardized stress protocol with a change in plasma prolactin levels ($F_{1,52} = 4.34$, $p = 0.042$; Fig. 4) that was significantly affected by the status of individuals (GLM, $F_{2,52} = 12.14$, $p < 0.001$). Specifically, prolactin levels of failed breeders increased in response to the standardized stress protocol whereas those of incubating and breeding birds decreased (contrasts: failed breeders, $F_{1,52} = 6.80$, $p = 0.012$; incubating birds, $F_{1,52} = 18.68$, $p < 0.001$; brooding birds, $F_{1,52} = 6.55$, $p = 0.013$; Fig. 4). Moreover, the decrease in prolactin levels of incubating birds was significantly more pronounced than that of brooding birds (contrast, $F_{1,52} = 12.97$, $p < 0.001$; Fig. 4). The change in prolactin levels in response to the stress protocol was not affected by sex ($F_{1,51} = 0.76$, $p = 0.389$; Fig. 4), body condition ($F_{1,48} = 0.27$, $p = 0.609$) or any interaction ($p > 0.850$ for all interactions).

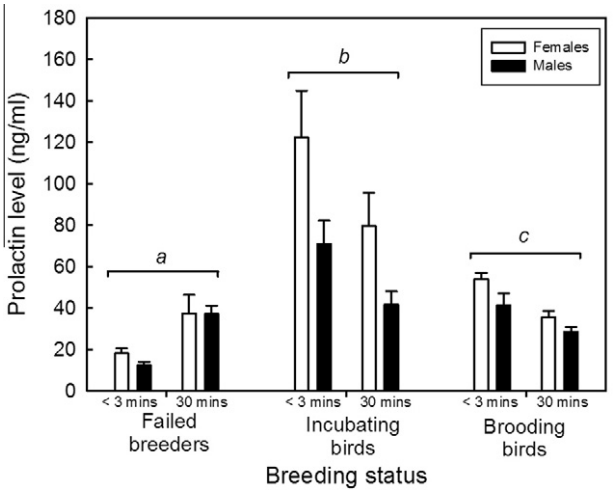

Fig. 4. Prolactin stress response of Cape petrels engaged in different parental activities (failed breeders, incubating birds, brooding birds). Letters above bars denote statistical results of stress response comparisons between the different groups. Different letters denote statistical significance between two groups. Prolactin levels of failed breeders significantly increased in response to the standardized stress protocol whereas those of incubating and breeding birds significantly decreased. Baseline and stress-induced prolactin levels of females were significantly higher than those of males for incubating birds, but not for brooding birds and failed breeders. Data are expressed as means + standard error.

In addition, prolactin levels were affected by breeding status ($F_{2,49} = 38.62$, $p < 0.001$; Fig. 3), sex ($F_{1,49} = 14.74$, $p < 0.001$; Fig. 4) and the interaction between sex and breeding status ($F_{2,49} = 6.92$, $p = 0.002$; Fig. 4). However, prolactin levels were not affected by body condition ($F_{1,48} = 0.13$, $p = 0.724$) or any other interaction ($p > 0.500$ for all other interactions). Specifically, baseline prolactin levels of failed breeders were lower than those of brooding birds (contrast: $F_{1,52} = 16.00$, $p < 0.001$; Fig. 4) and those of brooding birds were lower than those of incubating birds (contrast: $F_{1,52} = 43.93$, $p < 0.001$; Fig. 4). Baseline prolactin levels of females were higher than those of males for incubating birds (contrast, $F_{1,49} = 14.22$, $p < 0.001$; Fig. 4), but not for brooding birds and failed breeders (contrasts, brooding: $F_{1,49} = 1.36$, $p = 0.250$, failed breeders: $F_{1,49} = 0.11$, $p = 0.737$; Fig. 4). Stress-induced prolactin levels of failed breeders were similar to those of brooding birds (contrast: $F_{1,52} = 0.56$, $p = 0.456$; Fig. 4) but those of brooding birds were lower than those of incubating birds (contrast: $F_{1,52} = 15.29$, $p < 0.001$; Fig. 4). Stress-induced prolactin levels of females were respectively higher than those of males for incubating birds (contrast, $F_{1,49} = 14.71$, $p < 0.001$; Fig. 4), but not for brooding birds and failed breeders (contrasts, brooding: $F_{1,49} = 0.85$, $p = 0.361$, failed breeders: $F_{1,49} < 0.01$, $p = 0.982$; Fig. 4).

3.3. Link between the corticosterone and the prolactin stress responses

Prolactin levels were not explained by corticosterone levels and all the terms including the 'corticosterone' variable were excluded ($p > 0.100$ for all terms; Fig. 5). As in our previous analysis on the prolactin stress response, the same model was therefore selected and included the 'sex' and 'breeding status' factors, their interaction, the 'sample' factor and the 'breeding-status x sample' interaction (see previous paragraph for specific statistics). Confirming this result, we did not find any significant correlation between prolactin and corticosterone levels whatever the sex and the breeding status of individuals (correlations: baseline: $p > 0.100$ for all correlations; stress-induced: $p > 0.125$ for all correlations; Fig. 5).

3.4. Reproductive performance

Incubating petrels responded to the standardized stress protocol with an increase in plasma corticosterone levels ($F_{1,14} = 9.08$, $p = 0.009$; Fig. 6). This increase was not affected by the sex of individuals ($F_{1,12} = 1.19$, $p = 0.297$; Fig. 6) and did not differ between successful birds and the birds that lost their egg ($F_{1,13} = 1.84$, $p = 0.198$; Fig. 6). Baseline corticosterone levels were not affected by the sex of individuals ($F_{1,12} = 0.22$, $p = 0.650$; Fig. 6) and did not differ between successful birds and the birds that lost their egg ($F_{1,13} = 0.09$, $p = 0.772$; Fig. 6). Stress-induced corticosterone levels were not affected by the sex of individuals ($F_{1,12} = 1.41$, $p = 0.258$; Fig. 6) and did not differ between successful birds and the birds that lost their egg ($F_{1,13} = 1.76$, $p = 0.208$; Fig. 6).

Incubating petrels responded to the standardized stress protocol with a decrease in plasma prolactin levels ($F_{1,14} = 9.08$, $p = 0.009$; Fig. 5). This decrease in prolactin levels was not affected by the sex of individuals ($F_{1,13} = 0.33$, $p = 0.573$; Fig. 5) and did not differ between successful birds and the birds that lost their egg ($F_{1,12} = 0.41$, $p = 0.534$; Fig. 5). However, baseline prolactin levels of successful birds were significantly higher than those of birds that lost their egg ($F_{1,12} = 6.03$, $p = 0.030$; Fig. 5). Moreover, baseline prolactin levels of females were also higher than those of

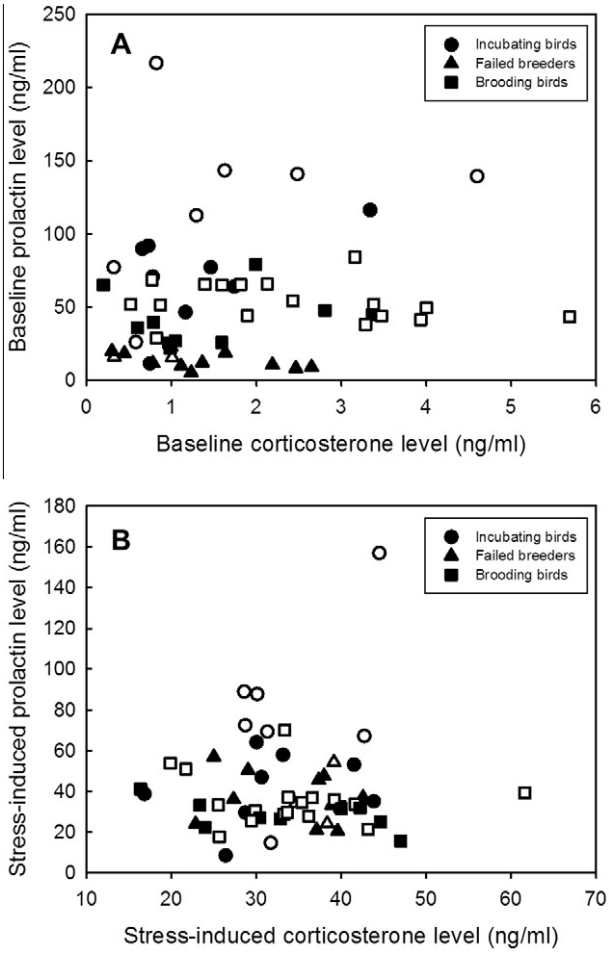

Fig. 5. (A) Relationships between baseline prolactin and baseline corticosterone levels; (B) Relationships between stress-induced prolactin and stress-induced corticosterone levels. Sampled Cape petrels were engaged in different parental activities (failed breeders, incubating birds, brooding birds) and prolactin and corticosterone levels were not significantly correlated in any group. Open and filled symbols respectively denote females and males.

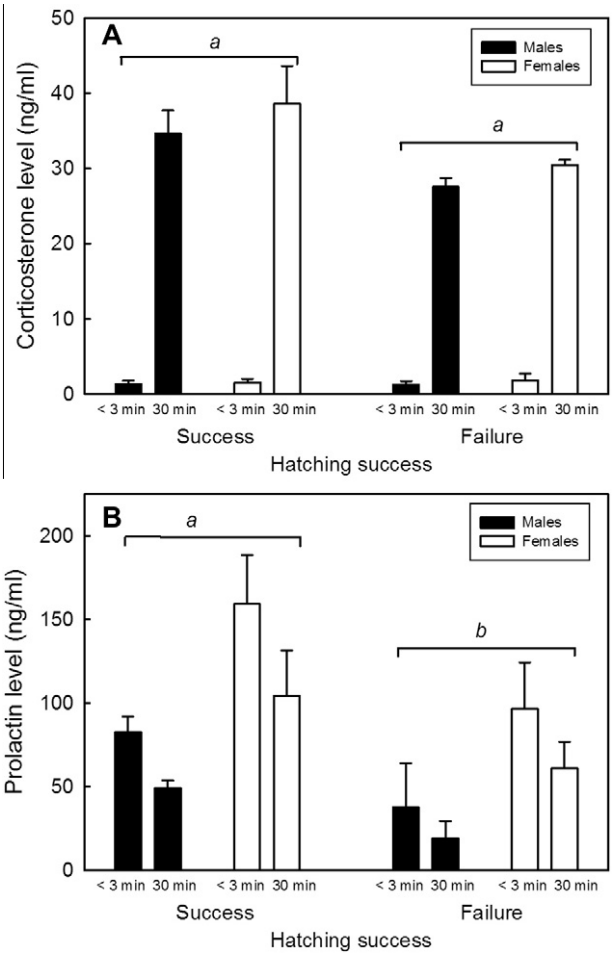

Fig. 6. (A) Corticosterone stress response in relation with hatching success in incubating Cape petrels. Letters above bars denote statistical results of hormonal levels comparisons between the different groups. Different letters denote statistical significance between two groups. Baseline and stress-induced corticosterone levels did not significantly differ between successful birds and the birds that lost their egg. (B) Prolactin stress response in relation with hatching success in incubating Cape petrels. Independently of sex, baseline and stress-induced prolactin levels of successful birds were significantly higher than those of birds that lost their egg. Data are expressed as means + standard error.

males ($F_{1,12}$ = 10.23, p = 0.008; Fig. 5). Stress-induced prolactin levels of successful birds were significantly higher than those of birds that lost their egg ($F_{1,12}$ = 6.35, p = 0.027; Fig. 5). Moreover, stress-induced prolactin levels of females were also higher than those of males ($F_{1,12}$ = 11.80, p = 0.005; Fig. 5).

4. Discussion

We investigated the relationship between the prolactin and the corticosterone stress responses in free-living parent seabirds. First, we reported that these two stress responses were not modulated in the same ways (corticosterone: body condition, prolactin: breeding status). Second, we found that prolactin levels, but not corticosterone levels, were related to hatching success in this species. Finally, we did not find any significant correlation between these two stress responses despite investigating this question under different levels of parental effort (incubating birds, brooding birds and failed breeders). All these results suggest that these two stress responses are not tightly linked (at least in Cape petrels) and that they may provide complementary pieces of information on parental investment. Therefore, we believe that both of these stress responses should be investigated if possible when focusing on the endocrine proxies of parental investment in birds.

4.1. Modulation of the prolactin and corticosterone stress response

Numerous studies have reported for many years now that the corticosterone stress response is modulated according to multiple energetic and environmental factors and that such modulation can be related to individual life-history strategies [1,28,34,39–41,60,66,70]. Importantly, recent studies found that the prolactin stress response can also be modulated according to these factors (reviewed in [2]; see also [4,32,53]). In that context, our prediction was that these two hormonal stress responses should be modulated according to the same factors if they are part of the same overall general mechanism. Contrary to this prediction, we found this was not at all the case in our study species. The prolactin stress response differed with breeding status in Cape petrels but, surprisingly, this was not the case for the corticosterone stress response. Moreover, the corticosterone stress response was attenuated in petrels that were in good condition, but we did not find any relationship between the prolactin stress response and body condition. Finally, a review of the existing literature shows that there is often a discrepancy in the factors that modulate the corticosterone and the prolactin stress responses in breeding birds (Table 1). All these

Table 1
Review of the existing studies that focused on the modulation of the prolactin and corticosterone stress response in parent birds.

Common name	Modulation of the stress response			Reference
	Factor considered	Prolactin	Corticosterone	
Black-legged kittiwake	Breeding status	Yes	No	[19]
	Body condition	No	No	[19]
	Foraging handicap[a]	No	No	[38]
Snow petrel	Age	Yes	No	[4]
	Breeding status	Yes	No	[8]
	Body condition	No	Yes[b]	[8]
Common tern	Age	Yes	Yes	[32]
	Body condition	No	Yes	[32]
Mourning dove	Body condition	No	No	[45]
	Parental effort[c]	No	Yes	[45]
Manx shearwater	Breeding status	Yes	Yes	[49]
	Body condition	No	No	[49]
Eurasian hoopoe	Breeding stage[b]	Yes	NA	[53]
	Body condition	No	NA	[53]
Cape petrel	Breeding stage	Yes	No	This study
	Body condition	No	Yes	This study

[a] Experimental handicap by clipping feathers.
[b] Only for females.
[c] Parental effort is measured by the change of weight of the brood; NA: not available.

results strongly suggest that these two stress responses are, at least partly, disconnected and may be involved in different part of the physiological and behavioral responses to a stressor in birds.

Interestingly, the prolactin stress response was more pronounced for incubating birds than brooding birds. As previously reported in other studies of prolactin [2,4,32,53], this result supports the brood value hypothesis [39], which suggest that the stress response should be attenuated when the fitness value of the current reproduction is high. In Cape petrels, the fitness value of a chick is much larger than that of an egg since most breeding failures occur during the incubation period [65] and most chicks will successfully fledge (this study). In addition, it is also likely that baseline prolactin levels of incubating are higher than those of brooding birds because birds are in more intimate contact with their egg than their chicks [54,55]. More astonishingly, prolactin levels increased in response to the stress protocol in failed breeders. Although this pattern is totally opposite to what has been reported in most breeding birds (reviewed in [2]) and even failed breeders from other species [8,19], such a result has been found in another seabird species, the Manx shearwater, *puffinus puffinus* [49] and also mammal species [46,48]. In failed breeders, inter-individual variations in hormones levels (prolactin, corticosterone) could result from different timing of egg loss. Indeed, it is possible that some failed breeders lost their egg a long time before to be sampled whereas others did loose it only recently. At the ultimate level, this difference between failed breeders and breeders is difficult to explain because failed breeders do not have obvious benefits to maintain behaviors related to parental cares after breeding failure and, thus, to increase prolactin secretion in response to a stressor. As suggested by [49], prolactin regulation and actions may simply differ to a large extent between parents and failed breeders and, thus, explain this surprising pattern. However, it remains to be clarified why this pattern is found in failed breeders from some species ([49]; this study) but not others [8,19].

Alternatively, it is possible that this prolactin stress response does not result from the stress of our standardized protocol, but rather from a side effect of this protocol. Actually, prolactin secretion is governed by two well-documented factors in birds [55,56]. First, photoperiod is known to act on prolactin regulation with long photoperiods stimulating prolactin secretion [25] and obviously, this factor is not affected by the standardized stress protocol. Second, it is very well-known that the presence of eggs or young during post-laying period stimulates prolactin secretion over and above that induced by the photoperiod [30,54]. This mechanism is essential to consider because the removal of an adult bird from its nest and, thus, from the egg(s)/young(s) stimuli during the stress protocol could lead to a decrease in prolactin levels and, therefore, explain the "prolactin stress response". This mechanism is however unlikely to explain the whole phenomenon (i.e. the important decrease in prolactin levels that we documented) for two reasons. First, prolactin levels also decrease in response to this standardized stress protocol in birds that are not engaged into parental activities (non-breeders or failed breeders that do not have eggs or chicks [8,19]) suggesting that removal from the egg/chick is not the only factor being responsible for such a decrease in prolactin levels. Second, prolactin levels decrease dramatically during a standardized stress protocol whereas the birds are removed from their nest for 30 min only. In comparison, prolactin levels only slightly decrease during a foraging trip in several seabird species whereas parents are away from their egg/chick for days during such a trip [2]. Although this factor may be crucial to consider in precocial species and domesticated species (poultry), our study suggests that stress is probably the main cause of this decrease in prolactin levels in altricial species.

4.2. Corticosterone, prolactin and fitness

In our study, prolactin, but not corticosterone, levels were related to hatching success. This result shows that the corticosterone and the prolactin stress responses were not correlated in the same ways to reproductive performances in Cape petrels and, therefore, supports the hypothesis that these stress responses do not mediate the same components of parental investment in birds. Previous studies have reported that the corticosterone stress response can be related to fitness in vertebrates but they also emphasized that the relationship between corticosterone and reproductive success is complex and context-dependent (reviewed in [10,13,16]. Elevated corticosterone levels have classically a negative effect on parental behaviors and are therefore correlated with a low breeding success (reviewed in [37,50,52,70]. On the other hand, moderately elevated corticosterone levels can also be necessary to sustain metabolic processes, foraging and parental effort [6,14,37,43]. In some situations, low corticosterone levels may therefore be the sign of a reduced parental investment and, thus, be correlated with breeding failure (reviewed in [13]). This complexity could explain the inconsistencies in the relationship that link corticosterone

and reproductive performances (reviewed in [13,16] and this may also explain why corticosterone levels were not related to hatching success in our study.

Contrary to corticosterone, the influence of prolactin levels on reproductive success has been investigated in a very limited number of studies and we currently lack data on this topic (reviewed in [2]). In our study, we found a significant positive influence of prolactin on breeding success since low baseline and stress-induced prolactin levels were associated with incubation failure in Cape petrels. This is not really surprising since this hormone plays a major role in parental behaviors [2,54,56,59]. For example, experimental increases of prolactin levels resulted in improved parental behaviors (i.e. brood provisioning) in house finches [11]. Similarly, Angelier et al. [9] demonstrated in another seabird species that birds with reduced prolactin levels were taking a long time to come back to their nest after a disturbance, supporting the idea that low prolactin levels are associated with temporary and definitive nest desertion ([4,18,29,57,58], but see [36]).

4.3. Are corticosterone and prolactin levels correlated in parent birds?

In this study, our aim was to provide additional useful information on the relationship that links corticosterone and prolactin levels in breeding birds. An accurate review of the literature shows that the relationship between corticosterone and prolactin is not consistent across bird species (Table 2). For instance, baseline corticosterone and prolactin levels were correlated in incubating Snow petrels [8] but not in incubating Common terns, *Sterna hirundo* [32] or, in Giant petrels, *Macronectes spp.* [23]. Even more surprisingly, the relationship between these two hormones is not always consistent in the same species, suggesting therefore that the functional relationship between prolactin and corticosterone is complex and probably depends on the ecological or energetic context. For example, Chastel et al. [19] reported in black-legged kittiwakes (*R. tridactyla*) that stress-induced corticosterone and prolactin levels were negatively correlated in failed breeders, but not in incubating birds. Such difference is astonishing and there is not yet any strong evidence to explain how the ecological or energetic context may drive these discrepancies. It is possible that corticosterone and prolactin levels are negatively correlated under challenging conditions only. Supporting this idea, it is known that perturbations induce an elevation of corticosterone levels [50,71,72] but also that the maintenance of elevated corticosterone levels for an extended period can induce a decrease in prolactin concentrations [2,9,21,58]. Consequently, we examined the relationship between the corticosterone and prolactin stress responses in Cape petrels engaged in different activities (i.e. different ecological contexts). We did not report any significant correlation between the corticosterone and the prolactin stress response in Cape petrels whatever their breeding status was (failed breeders, incubating birds or brooding birds). Our results show that considering the ecological context (e.g. breeding status) is probably not sufficient to explain the inconsistencies that are observed in the prolactin–corticosterone relationship. Moreover, this shows that prolactin and corticosterone levels are often disconnected and, therefore, supports our idea that the prolactin and corticosterone stress responses mediate different parts of the physiological and behavioral responses to a labile perturbation factor. What would then be the specific effects of the corticosterone and prolactin stress responses?

Corticosterone is well-known for its action on metabolic processes (reviewed in [37,52]). For instance, elevated corticosterone levels are known to mobilize energy from body reserves to allow the organism to escape or fight the stressor and, thus, to promote

Table 2
Review of the existing studies that examined the correlations between prolactin and corticosterone levels in parent birds. Studies have not been included in this summary when experimental manipulations of corticosterone or prolactin occurred or when prolactin and corticosterone levels were reported during the non-breeding period. In addition, several studies did not report the correlations between prolactin and corticosterone levels avoiding us to use their data.

Common name	Situation	Hormone levels	Correlation	Reference
Black-legged kittiwake	Chick-rearing	Baseline	No	[19]
	Chick-rearing	Stress-induced	No	[19]
	Failed breeders	Baseline	No	[19]
	Failed breeders	Stress-induced	Negative	[19]
Common eider	Incubation	Baseline	No	[22]
Wandering albatross	Incubation	Baseline	No	[3]
Black-browed albatross	Chick-rearing	Baseline	Negative	[5]
Snow petrels	Incubation	Baseline	No	[4]
	Incubation	Stress-induced	No	[4]
	Incubation	Baseline	Negative	[8]
	Incubation	Stress-induced	No	[8]
	Failed breeders	Baseline	No	[8]
	Failed breeders	Stress-induced	No	[8]
King penguin	Incubation	Baseline	No	[29]
Mourning dove	Early chick-rearing	Baseline	Positive	[45]
	Late chick-rearing	Baseline	No	[45]
Common tern	Incubation	Baseline	No	[32]
	Incubation	Stress-induced	No	[32]
Eurasian hoopoe	Chick-rearing	Baseline	No	[53]
	Chick-rearing	Stress-induced	Negative[a]	[53]
Northern and Southern	Incubation	Baseline	No	[23]
Giant petrel	Chick-rearing	Baseline	No	[23]
	Failed breeders	Baseline	No	[23]
Kentish plover	Chick-rearing	Baseline	No	Kosztolányi et al. (unpublished)
	Chick-rearing	Stress-induced	No	Kosztolányi et al. (unpublished)
Cape petrels	Incubation	Baseline	No	This study
	Incubation	Stress-induced	No	This study
	Chick-rearing	Baseline	No	This study
	Chick-rearing	Stress-induced	No	This study
	Failed breeders	Baseline	No	This study
	Failed breeders	Stress-induced	No	This study

[a] Marginally significant correlation.

immediate survival. On the other hand, prolactin is more directly connected to parental behavior in parent birds (reviewed in [2,56,59]) and the prolactin stress response may be more related to specific parental decisions in response to a labile perturbation factor (e.g. provisioning and protecting the brood). Although the prolactin and corticosterone stress responses can mediate different physiological and behavioral components, energy management and parental strategies can obviously be tightly connected under some circumstances and this may explain the correlations that were found in some studies (Table 2). Indeed, elevated corticosterone levels are also well-known to activate an emergency life-history stage that redirects the energy available from parental effort towards immediate survival (reviewed in [37,50,70,72]). Moreover, experimental elevations of corticosterone levels induce a decrease in prolactin levels [9,21,58] and this may explain that corticosterone and prolactin levels are negatively correlated in some situations (Table 2). On the other hand, at lower level, prolactin may also promote corticosterone secretion [35,45]. Such moderately increased corticosterone levels may allow sustaining the energetic requirements of parental activities [6,14,43]. This may in turn explain the positive correlations that are sometimes reported between prolactin and corticosterone levels. These results suggest that the relationship between corticosterone and prolactin may depend on threshold value of both of these hormones [2,57,74]. At low corticosterone levels, this relationship might be driven by a positive action of each hormone on the secretion of the other [35,45,74] whereas, at elevated corticosterone levels, this relationship might be driven by the negative effect of corticosterone on prolactin levels [9,21,58,74]. In addition, the relationship between corticosterone and prolactin is probably even more complex because, for instance, the corticosterone levels need to be elevated for a prolonged period to exert their negative action on prolactin levels [9].

All this complexity may explain why the relationship between corticosterone and prolactin appears so inconsistent (Table 2) but we believe that further investigating this link will allow better understanding the strategies individuals use to cope with labile perturbation factors. To do so, future studies should focus on other physiological mechanisms that can link the regulation and the actions of these two hormones (reviewed in [69]; e.g. corticosterone binding globulin [15,43]; corticosterone receptors [17,50,61]; prolactin receptors [20]; steroidogenic enzymes, such as 11β-hydroxysteroid dehydrogenase, expressed in target cells that can modulate how much corticosterone encounters genomic receptors, [33]). In addition, it also appears essential to examine how ecological and energetic factors can affect the link between the prolactin and the corticosterone stress responses in wild vertebrates.

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