Family-transmitted stress in a wild bird

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Recent data suggest that, in animals living in social groups, stress-induced changes in behavior have the potential to act as a source of information, so that stressed individuals could themselves act as stressful stimuli for other individuals with whom they interact repeatedly. Such form of cross-over of stress may be beneficial if it enhances adaptive responses to ecological stressors in the shared environment. However, whether stress can be transferred among individuals during early life in natural populations remains unknown. Here we tested the effect of living with stressed siblings in a gull species where, as in many vertebrates, family represents the basic social unit during development. By experimentally modifying the level of stress hormones (corticosterone) in brood mates, we demonstrate that the social transfer of stress level triggers similar stress responses (corticosterone secretion) in brood bystanders. Corticosterone-implanted chicks and their siblings were faster in responding to a potential predator attack than control chicks. In gulls, fast and coordinated reactions to predators may increase the chances of survival of the whole brood, suggesting a beneficial fitness value of cross-over of stress. However, our data also indicate that living with stressed brood mates early in life entails some long-term costs. Near independence, fledglings that grew up with stressed siblings showed reduced body size, high levels of oxidative damage in lipids and proteins, and a fragile juvenile plumage. Overall, our results indicate that stress cross-over occurs in animal populations and may have important fitness consequences.

glucocorticoids | group living | phenotypic programming | social environment | stress cross-over

Interacting with others in the social environment has important fitness consequences in group-living animals (1), and the behavior of other individuals in the group may generate important information about the prevailing environmental conditions (socially acquired information) (2, 3). Environmental stressors can activate the endocrine and neuroendocrine stress response, a highly conserved reaction in vertebrates that promotes several physiological and behavioral changes (4–6). Such adaptive response helps to cope with stressors and may potentially act as a source of information for other group members about the current conditions in the shared environment. Indeed, some evidence suggests that the behavior of stressed individuals may have consequences for their social partners (7, 8). However, whether stress responses may be transferred among group members is unclear, and their possible consequences have not hitherto been investigated.

Stressors trigger the hypothalamic–pituitary–adrenal (HPA) axis, a neuroendocrine pathway by which stress hormones (glucocorticoids) are released from the adrenal cortex to the general circulation (reviewed in ref. 9). The HPA axis integrates internal and external (environmental) stimuli, and the downstream release of glucocorticoids translates and transmits such information to different organs, potentiating an adaptive response (10, 11). Thus, the secretion of stress hormones leads to a rapid redistribution of energy resources away from nonvital functions and organs and to changes in behavior that help animals cope with the stressor (reviewed in refs. 9 and 12). For instance, exposure to predators causes elevation of stress hormones, which in turn enhances antipredator responses such as increased vigilance and fast reaction against predators (13, 14), thereby promoting short-term survival (4).

Empirical studies suggest that the use of socially acquired information is an important process involved in antipredator behavior or resource acquisition, and has an inherent adaptive value (2). In this context, stress-induced changes in behavior or state may act as a source of information for other group members not directly exposed to the stressful stimuli and may potentially lead to a horizontal transfer or cross-over of stress. The cross-over of stress is a well-known phenomenon in the field of human psychology, and some recent evidence suggests that the cross-over of stress could be a biological phenomenon more important than previously thought in animal social groups (e.g., 7, 10).

The social environment has important consequences also for health and fitness (15). Despite the possible benefits of stress cross-over, a protracted stress exposure can be damaging (16, 17), particularly when occurring during early stages of development (18–20). Early exposure to stressful conditions can, for instance, negatively affect growth rates (11, 16), the development of key body structures [i.e., feathers in birds (21)], and promote the accumulation of oxidative damage (22). To fully understand the role of stress cross-over in animal populations, it is fundamental to assess the costs and benefits, if any, of such phenomena.

Here we first tested whether stress responses are transferred among family members during early development of a long-lived social seabird, the yellow-legged gull (*Larus michahellis*). As in many animals with prolonged parental care, interactions among siblings are the most common social relationship exhibited by gull chicks during early life (23) (Fig. S1). We created experimental broods in which two of the three siblings were exposed to

Significance

Different environmental stimuli can lead animals to go into an emergency state and experience stress; but can an individual notice the stress experienced by other members of its social group and develop a similar physiological reaction? We demonstrate that such a form of cross-over of stress can actually occur in wild animal populations. Gull chicks that grew up with experimentally stressed siblings showed increased secretion of stress hormones. In the short term the cross-over of stress seemed to be favorable, improving chicks' antipredator behavior, but in the long term the chicks grew slowly and attained a reduced adult size, showed increased accumulation of cell damage, and developed a poor-quality juvenile plumage. The cross-over of stress can be an important but complex selective force.

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increased levels of stress hormones by corticosterone implants, and control broods in which two chicks received a sham (empty) implant. Corticosterone is the main stress hormone present in birds (6). This experimental design allowed us to test the effects of growing up with stressed siblings in the nonimplanted counterparts. We examined the effect of stress cross-over on chick phenotype (corticosterone level, growth, and behavior) during development. We first evaluated whether stress cross-over early in life has similar programming effects on phenotype to those directly exposed to stress (corticosterone-implanted). We expected that the cross-over of stress may bring short-term benefits to brood mates of the corticosterone-implanted chicks, such as enhanced antipredator behavior, but at the cost of long-term negative consequences in body size, oxidative damage, and plumage quality.

Results

Early Postnatal Effects. The rate of growth during the first 8 d after hatching in tarsus length and body mass was significantly lower in the chicks implanted with corticosterone and their nonimplanted brood mates (i.e., chicks in experimental broods; hereafter "stress group"; Table 1 and Fig. S2). As a result, at 8 d of age, chicks in the stress group were lighter and structurally (bone size) smaller than chicks in the control group ($P \leq 0.02$ in all Tukey's post hoc tests; Fig. 1 *A* and *B*). Interestingly, corticosterone-implanted and nonimplanted chicks in the stress broods showed a similar reduction in growth (Tukey's post hoc tests: tarsus, $P = 0.480$; body mass, $P = 0.480$; Fig. 1 *A* and *B* and Fig. S2). Moreover, the stress group showed an increase in basal corticosterone levels (Table 1 and Fig. S2). Thus, at 8 d of age, chicks in the stress group had higher corticosterone levels than chicks in the control group ($P \leq 0.011$ in all Tukey's post hoc tests), and corticosterone-implanted or nonimplanted chicks in the stress group showed a similar corticosterone level (Tukey's post hoc test: $P = 0.608$; Fig. 1*C*). Sex was never significant in the models (Table 1), and there were no significant two-way interactions between fixed factors (all $P > 0.106$). To evaluate whether these effects were mediated by nutritional differences, we measured plasma triglycerides and proteins, both very sensitive markers of nutritional status in gulls (24). However, these markers did not differ between the experimental groups at ages 1 and 8 d ($P > 0.25$ in all pairwise comparisons; Table S1).

The stress treatment affected the chicks' antipredator behavior [brood treatment (corticosterone vs. sham): $F_{42.06} = 8.60$, $P = 0.005$; chick manipulation (implanted vs. nonimplanted): $F_{1,32.90} = 0.150$, $P = 0.701$; brood treatment × chick manipulation: $F_{1,33.09} = 0.272$, $P = 0.605$; Fig. 1*D*). Chicks from the stress group, irrespective of whether they were corticosterone-implanted or not, were faster in crouching and hiding after listening to adult alarm calls in comparison with chicks from the control group (time to crouch; mean ± SE; control, 7.16 ± 0.95 s; stress, 4.30 ± 0.38 s; Fig. 1*D*). There was no effect of sex ($F_{1,52.88} = 0.397$, $P = 0.531$) or chick order ($F_{1,29.26} = 0.509$, $P = 0.481$) on the antipredator behavior. None of the three- and two-way interactions between fixed factors was significant in the models (all $P > 0.089$).

Late Postnatal Effects. At the end of the growth period, 30 d after hatching, we recaptured nonimplanted fledglings. Chicks that grew up with corticosterone-implanted siblings were still lighter ($F_{1,23} = 5.361$, $P = 0.030$; Table S2) and structurally smaller (tarsus: $F_{1,23} = 7.963$, $P = 0.010$; Table S2) than chicks with nonstressed siblings (Fig. 2 *A* and *B*). By that age, however, the difference in basal corticosterone level between the experimental groups had disappeared (brood treatment: $F_{1,23} = 0.456$, $P = 0.506$). The sex of the chicks and its interaction with treatment were not significant (sex: $F_{1,23} = 1.543$, $P = 0.227$; brood treatment × sex: $F_{1,22} = 1.069$, $P = 0.312$). Similarly, the levels of plasma triglycerides and proteins did not differ between experimental groups ($P > 0.152$ in both cases), and no other variable included in the models was significant (Table S3).

Growing up with stressed siblings affected the fledglings' oxidative status (Table 2); chicks that lived with corticosterone-implanted siblings had a higher level of lipid peroxidation (Fig. 2*C*) and advanced oxidation protein products (Fig. 2*D*) in comparison with nonimplanted chicks from the control group. The experimental treatment did not affect the antioxidant capacity of plasma or glutathione peroxidase level (Table 2). The sex of the chick and the rest of the variables included in the models of oxidative stress markers were not significant (Table 2).

Finally, our data also revealed that birds that grew up with stressed siblings developed weaker wing feathers, as suggested by the lower density of barbs and barbules in the primary covers (barb density: $F_{1,19} = 6.307$, $P = 0.021$; barbule density: $F_{1,19} = 4.708$, $P = 0.043$; Fig. 2 *E* and *F*). The social interaction with stressed siblings also tended to affect body feathers, but the differences between the two treatments were not significant (Table S4).

Discussion

Our findings reveal that experimental elevation of stress level in gull chicks can trigger stress responses in brood bystanders. This phenomenon was clearly evidenced by the increased secretion of glucocorticoid hormones in nestlings that were reared with experimentally stressed siblings. Increased basal corticosterone levels enhanced antipredator behavior but negatively affected growth in the implanted chicks. These effects of corticosterone implants were exactly mimicked in their social brood mate, suggesting that physiological and behavioral responses to stress may be acquired following exposure to stressed individuals in the social environment. We also provide strong evidence indicating that such form of horizontal transfer of stress has negative consequences for the affected individuals. Near independence, fledglings that grew up with stressed siblings were lighter, had a reduced skeletal size, showed increased levels of oxidative damage in several macromolecules, and developed a more fragile juvenile plumage than the control fledglings. In summary, our findings strongly suggest that the cross-over of stress may be a biological phenomenon more common than previously thought in vertebrates, and also highlight the critical role that the social environment may have on individual fitness.

Nonimplanted chicks had nearly identical basal corticosterone level to that seen in the corticosterone-implanted sibling at 8 d of age, suggesting that living with stressed siblings induces an up-regulation of the HPA axis. The transmission of stress from one chick to another may be caused by different mechanisms. In the first week of life, gull chicks in a brood closely interact with each other in many social contexts; together they explore the territory and respond to parental behavior and environmental threats, such as predators (23, 25) (Fig. S1*B*). Because corticosterone implants are likely to reduce begging displays (26), promote neophobic and fearful behaviors (16, 27), or even alter chicks' odor (28), corticosterone-implanted chicks may have acted as a source of information for their (nonimplanted) sibling, affecting their physiology and development. Previous studies suggest that the use of socially acquired information (e.g., behavior or states of conspecifics) may be a widespread phenomenon in animal social groups and a major driving force in social evolution (2). The cross-over of stress may also be the result of empathic-like reactions. The existence of this type of mechanism involving the recognition of others' emotional state has been described in humans, apes, and some rodents (8, 29), but the evidence is less clear in birds (but see refs. 30 and 31). Alternatively, stressed chicks could have generated stress on their parents (32), resulting in reduced

Table 1. Summary of linear mixed models for the effects of treatments and covariates on tarsus length, body mass, and basal corticosterone levels of yellow-legged gull chicks between 1 and 8 d of age

	Tarsus length				Body mass				Plasma corticosterone			
Variable	Estimate	$df_{n,d}$	*F*	*P*	Estimate	$df_{n,d}$	*F*	*P*	Estimate	$df_{n,d}$	*F*	*P*
Intercept	34.609				134.073				6.354			
Age, day 8	7.129	1,133.33	1120.661	**<0.001**	68.882	1,132.91	974.144	**<0.001**	2.852	1,96.48	89.684	**<0.001**
Brood treatment, control	1.936	1,51.86	5.538	**0.023**	19.522	1,49.98	7.766	**0.008**	−0.860	1,49.609	0.850	0.361
Chick manipulation, nonmanipulated	0.128	1,138.58	0.267	0.605	3.409	1,139.26	1.790	0.183	0.067	1,82.28	0.095	0.758
Sex, female	−0.0209	1,178.58	0.507	0.481	−1.012	1,177.71	0.120	0.729	−0.196	1,114.90	0.635	0.427
Chick order, first	−0.533	1,139.80	4.610	**0.034**	−2.969	1,140.49	1.35	0.246	0.127	1,82.39	0.343	0.560
Age × brood treatment	−1.949	1,133.33	16.253	**<0.001**	−18.744	1,132.92	13.948	**<0.001**	1.148	1,96.22	5.705	**0.019**

Nonsignificant interactions were removed from full models, and significant terms are highlighted in bold.

parental care and indirectly increasing corticosterone level in their nonimplanted siblings (32). However, this possibility seems unlikely because chicks from the control and stress groups showed similar nutritional status. Regardless of the mechanism, our results demonstrate stress transmission (either direct or indirect) among family members.

Corticosterone implants shortened the time taken by gull chicks to react against a potential threat (i.e., predator). This

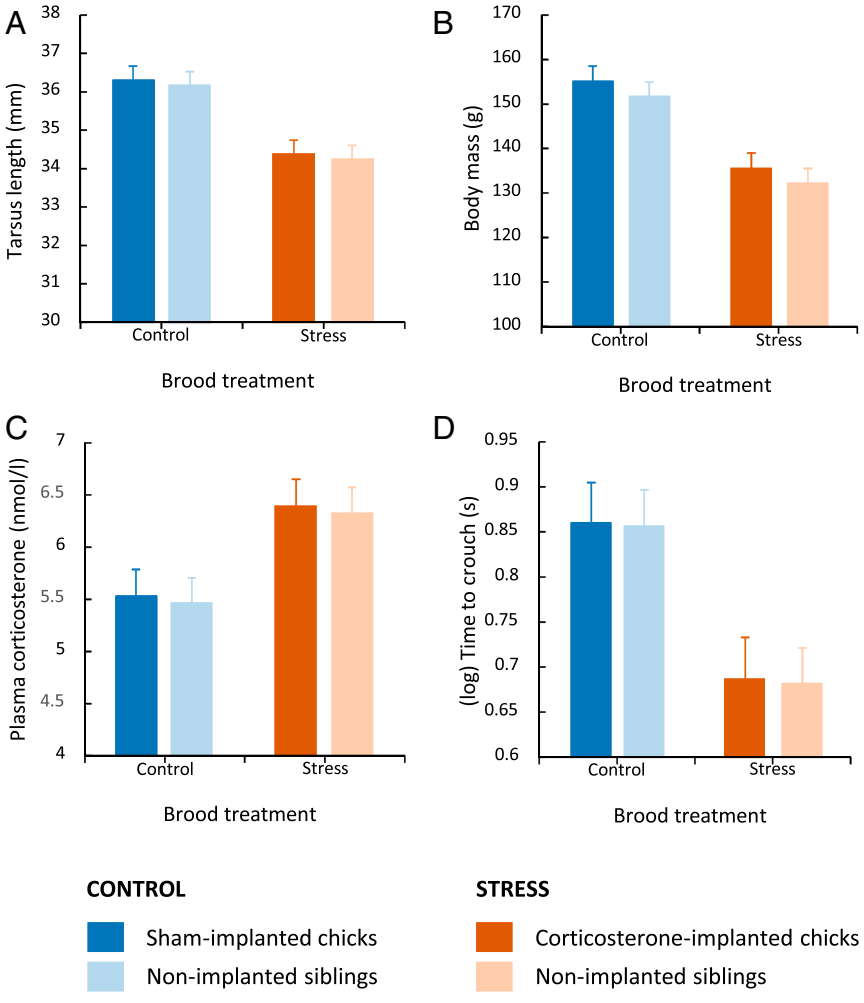

Fig. 1. Brood mates of corticosterone-implanted chicks showed reduced growth, increased basal glucocorticoid secretion, and a faster reaction time to predation risk. Tarsus length (*A*), body mass (*B*), and basal corticosterone level (root-squared) (*C*) at 8 d of age, and antipredator behavior (log-transformed time to crouch) (*D*) at 9 d of age in implanted (dark bars) and nonimplanted (light bars) yellow-legged gull chicks from control (blue) and stress (orange) broods. In each brood, two of three chicks were s.c. implanted between the shoulders with a surgical silastic implant filled with corticosterone (stress broods) or left empty (control broods), and the remaining sibling was left without being manipulated (nonimplanted). Data show estimated marginal mean ± SE (see Fig. S2 for the comparison between initial and final values of each variable).

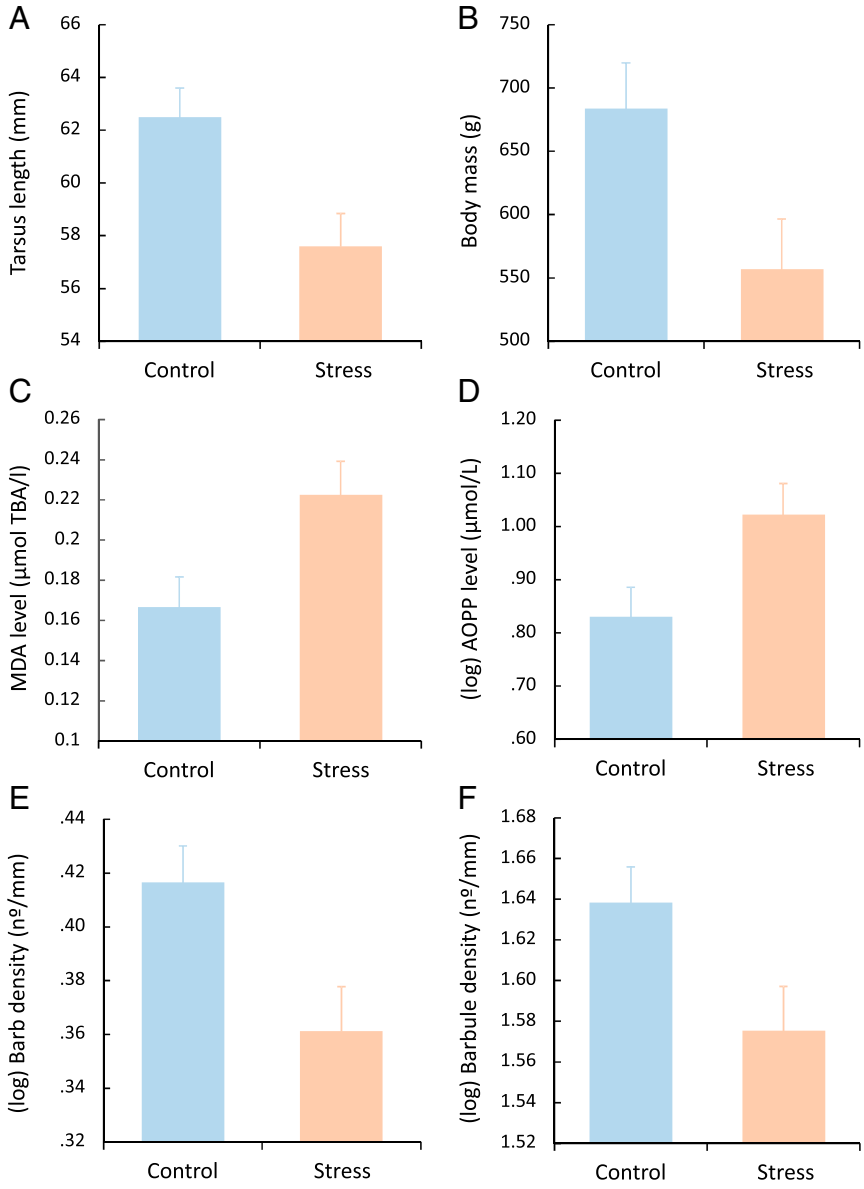

Fig. 2. Brood mates of corticosterone-implanted chicks were smaller, had increased levels of oxidative damage, and developed a more fragile juvenile plumage. Tarsus length (*A*), body mass (*B*), lipid peroxidation level (MDA) (*C*), advanced oxidation protein product (AOPP) level (*D*), barb density (*E*), and barbule density (*F*) in nonimplanted yellow-legged gull fledglings (30 d of age) from control (blue) and stress (orange) broods; note that none of these birds received an implant. Data show the estimated marginal mean ± SE. TBA, thiobarbituric acid reactive substances.

supports previous studies in other vertebrate species (e.g., 13, 14) and emphasizes the adaptive value of stress responses for promoting short-term survival (9). Importantly, our results suggest that stress transfer from brood mates experiencing an external stressor (predator) in the shared environment may enhance the chance of coping with this stressor (antipredator behavior) in brood mates that did not directly receive the stimuli. The coordinated crouching behavior of a brood probably reduces the predation risk of the whole brood (33), suggesting an important adaptive value of cross-over of stress. In some social species, the cross-over of stress may also be important for the persistence of chronic stress in populations under high predation risk, although the majority of living individuals do not directly interact with predators (e.g., 34). Information transmission about environmental stressors, such as predators, is a key factor underpinning group living and affecting collective behavior (35, 36). The transfer of stress among members of the same social group has important implications for understanding the evolution of social living, providing a mechanism through which social factors may influence the collective behavior of social groups.

The cross-over of stress had, however, some negative consequences for gull chicks. The elevated corticosterone levels seen in stressed chicks (corticosterone-implanted chicks and their nonimplanted siblings) impaired growth, as occurs in many taxa (reviewed in ref. 16). Corticosterone reduces and inhibits the production of important growth-related hormones such as growth hormone, somatomedin, and insulin-like growth factors, increases muscle protein degradation, and induces a general metabolic shift (see ref. 37 for a complete review of mechanisms). These effects were long-lasting, as evidenced by reduced skeletal size and body mass at age 30 d in siblings of the corticosterone-implanted chicks, although the effect on corticosterone levels disappeared at this age. Most yellow-legged gull chicks reach the adult skeletal size by this age and,

Table 2. Summary of the linear models for the effects of stress treatment and covariates on antioxidant defenses and oxidative damage in nonimplanted yellow-legged gull fledglings

	TAC				GPx				AOPP				MDA			
Variable	Estimate	$df_{n,d}$	*F*	*P*	Estimate	$df_{n,d}$	*F*	*P*	Estimate	$df_{n,d}$	*F*	*P*	Estimate	$df_{n,d}$	*F*	*P*
Intercept	2.697				2,337.923				0.942				0.178			
Brood treatment, control	0.990	1,20	1.707	0.206	368.200	1,23	2.721	0.113	−0.193	1,22	5.605	**0.027**	−0.056	1,20	5.858	**0.025**
Sex, female	−0.521	1,20	0.482	0.495	−816.394	1,23	13.910	**0.001**	0.110	1,22	1.830	0.190	0.006	1,20	0.072	7.792
Chick order, first	−0.235	1,20	0.094	0.762	−224.794	1,23	1.005	0.327	0.050	1,22	0.368	0.550	0.037	1,20	2.400	0.137
UA	0.114	1,20	0.426	0.521												
TRIG													0.001	1,20	3.234	0.087

Nonsignificant interactions were removed from full models, and significant terms are highlighted in bold.

therefore, the reduced size shown in socially stressed individuals is likely to have long-term fitness consequences (38).

The negative consequences of living with stressed siblings were also evidenced by reduced feather quality and increased oxidative damage of the fledglings. Feather microstructure is impaired by high corticosterone levels at the time of feather development (15). Although corticosterone level at 30 d of age did not differ between experimental groups, it might be possible that the effect of stress cross-over on basal corticosterone was still evident during part of the time period when feather development took place [i.e., between 12 and 40 d of age in this species (39)]. Living with stressed siblings also resulted in increased level of oxidative damage to lipids and proteins when chicks were near fledging, indicating that stress cross-over was oxidatively costly for gull fledglings. These oxidative costs support the general view of glucocorticoid hormones as modulators of oxidative status in vertebrates (22). In birds, prenatal elevation of corticosterone levels is associated with postnatal oxidative costs (40), agreeing with our result of the long-term effect of early exposure to corticosterone on oxidative damage. Importantly, high levels of oxidative damage at fledging are related to a reduction in adult survival in seabirds (41, 42).

In conclusion, our experimental manipulation clearly demonstrated that stressed animals can act by themselves as a source of stress for their siblings, and that such a form of horizontal transfer of stress may entail some possible short-term benefits by enhancing antipredator behavior, but at the cost of growth reduction, oxidative damage accumulation, and a more fragile plumage development. These costs may have important long-term fitness consequences. Future studies should, therefore, explore whether the short-term benefits could outweigh the long-term costs. We only investigated stress cross-over in the family context and in particular among siblings, but the function of social cues in the transfer of stress responses may be a widespread biological phenomenon that should be explored in other social contexts.

Materials and Methods

Study Area and General Procedures. We performed the study between April and July 2016 in a large breeding colony of yellow-legged gulls on Sálvora Island, northwest Spain. In this species, modal clutch size is three eggs, chicks hatch asynchronously, and broods remain together until fledging (35 to 40 d of age), allowing the young birds to interact for a prolonged period. We surveyed the study area once daily during egg laying and marked 64 three-egg nests with numbered sticks. We visited each nest every day until clutch completion to mark eggs and register egg-laying order. After clutch completion, we assigned the nests randomly to two different experimental groups: "control" or "stress" broods. To disrupt any potential stress covariation between parental and offspring phenotype, we cross-fostered the whole clutch between pairs of nests that had similar laying dates (±1 d) within each experimental group. We checked each nest daily, beginning 2 d before the estimated hatching date. Because yellow-legged gull chicks are semiprecocial and can move far away from their nest, before hatching we installed a fenced enclosure around each nest to keep chicks in their territory (see ref. 43 for further details about fence dimension). At hatching, we marked all chicks with numbered leg flags made with Velcro. In some nests, two or more eggs failed to hatch (control, $n = 6$; stress, $n = 5$), so the final sample size was 53 nests (control, $n = 27$ nests; stress, $n = 26$ nests).

The study was carried out with permission granted by the authorities of Parque Nacional de las Islas Atlánticas (364/RX598377). All experimental procedures complied with the standards of animal experimentation and animal welfare established under current Spanish law (RD53/2013) and were approved by the Xunta de Galicia review board.

Experimental Manipulation of the Level of Social Stress Within a Brood. In all broods, two chicks were s.c. implanted between the shoulders with a 10-mm surgical silastic tube (Dow Corning; BB518-58) when they were 1 d old (hereafter "implanted" chicks). In the stress broods, implants were filled with crystallized corticosterone (Sigma-Aldrich; 27840), whereas in the control broods, implants were empty (sham). Therefore, one chick per brood was kept without being manipulated (hereafter "nonimplanted" chick). The chick hatched from either the first- or the second-laid egg was randomly assigned as the nonimplanted chick in each nest. Chicks hatched from third-laid eggs were always implanted (corticosterone or sham) and kept with their siblings but not taken into account in the study. We decided to focus on only the first two hatched chicks (one implanted and one nonimplanted) for several reasons. The first two chicks have similar competitive abilities, but the third suffers a competitive disadvantage (44). Thus, by focusing on the first two chicks, we controlled the effect of sibling competition on stress hormone level (45), maximized sample size, and reduced researcher disturbance (in our study population the first two chicks showed a synchronous hatching, i.e., minimizing the number of visits per nest).

Implants were inserted under the skin through a small incision (2 to 3 mm) that was then sealed with surgical glue (Vetbond; 3M). All incisions healed well, and no bird showed any permanent detrimental effects from the surgical procedures. A pilot study confirmed that our corticosterone implants successfully elevated basal corticosterone levels for at least the following 7 d after implantation and within the normal range of variation in this colony [square-root transformed range, 3.8 to 18.5 nmol/L (43); Fig. S3].

Sampling. We blood-sampled and measured the first two hatched chicks in each brood at days 1 (just before the implantation) and 8 of age. We collected blood samples from the brachial vein with heparinized capillary tubes, measured their tarsus length (±0.1 mm), and weighed them (±1 g) using a Pesola spring balance. Blood samples were always collected within 3 min of capture to avoid any increase of baseline corticosterone levels as a consequence of handling (46). Blood samples were kept cold until plasma was separated from red blood cells (within a few hours after collection) and stored in liquid nitrogen. Red blood cells from day 1 were used for molecular sexing of the chicks as described (47). We assessed the antipredator response of the chicks at 9 d of age by assessing the latency to respond to adult alarm calls (*SI Materials and Methods*).

To avoid the stress of reduced territory size, we removed the enclosures around the nests after blood sampling the chicks at 8 d of age. We marked all of the nonimplanted chicks with a numbered plastic ring with an individual three-digit combination to facilitate their long-term identification. At 30 d of age, when nonimplanted chicks were fully grown and near fledging, we searched for them around their territories. For all nonimplanted chicks we found alive ($n = 27$; 14 control and 13 stress), we took a third blood sample (within 3 min of capture), measured their tarsus length, and weighed them (±5 g). We also plucked the third primary cover feather (counted from the outermost) of the left wing and one of the central scapular feathers to assess the effect of experimental manipulation on feather quality. Feathers were stored in small

paper envelopes until analysis (see below). Although the majority of birds had already developed their primary covers and scapulars by 30 d of age, in four birds feathers were still growing the day of sampling and therefore the birds were not feather-sampled (one control and three stress). For each feather, we determined barb and barbule density (*SI Materials and Methods*).

Biochemical Analyses. In plasma sampled at age 1, 8, and 30 d, we measured basal corticosterone, uric acid (UA), and triglyceride (TRIG) levels using commercially available kits, and protein levels as described (48) (details can be found in *SI Materials and Methods*). We also measured different biomarkers of antioxidant defense [nonenzymatic total antioxidant capacity (TAC) and glutathione peroxidase (GPx)] and lipid and protein oxidative damage [malondialdehyde (MDA) and advanced oxidation protein product (AOPP) level] in the blood samples taken from nonimplanted chicks at 30 d of age (*SI Materials and Methods*).

Statistical Analyses. We used linear mixed-effect models (LMMs) to test the effect of experimental manipulations [brood treatment (control vs. stress) and chick manipulation (implanted vs. nonimplanted)] on chicks sampled at 1 and 8 d of age. Brood identity and chick identity were included as random terms. We also ran an LMM to examine the effect of experimental manipulations on chick antipredator behavior (time to crouch), including brood identity as a random term. In nonimplanted birds recaptured at 30 d of age, we analyzed the effect of stress treatment using linear models (LMs). Residuals obtained from the models were always normally distributed. We report results for full models (see Table S5 for sample sizes) after removing nonsignificant interactions (49). Additional details are provided in *SI Materials and Methods*.

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