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Effects of Cortisol on Aggression and Locomotor Activity in Rainbow Trout

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Noninvasive administration of cortisol through the diet resulted in relatively rapid (<1.5 h) and highly reproducible increases in plasma cortisol in rainbow trout, comparable to changes seen in fish subjected to substantial stress. Juvenile rainbow trout were reared in isolation for 1 week, before their daily food ration was replaced by a meal of cortisol-treated food corresponding to 6 mg cortisol kg^{-1}. All fish were observed for 30 min, beginning at 1 or 48 h following the introduction of cortisol-treated food. Additional cortisol (75% of the original dose on Day 2, and 50% on Day 3) was administered to the long-term cortisol-treated group. The resulting blood plasma concentrations of cortisol were similar in short- and long-term treated fish, and corresponded to those previously seen in stressed rainbow trout. Controls were fed similar food without cortisol. Half of the fish from each treatment group (controls and short- and long-term cortisol) were subjected to an intruder test (a smaller conspecific introduced into the aquarium), while half of the fish were observed in isolation. In fish challenged by a conspecific intruder, short-term cortisol treatment stimulated locomotor activity, while long-term treatment inhibited locomotion. Aggressive behavior was also inhibited by long-term cortisol treatment, but not by short-term exposure to cortisol. Cortisol treatment had no effect on locomotor activity in undisturbed fish, indicating that the behavioral effects of cortisol were mediated through interaction with other signal systems activated during the simulated territorial intrusion test. This study demonstrates for the first time that cortisol has time- and context-dependent effects on behavior in teleost fish.

Key Words: **aggression; behavior; cortisol; glucocorticoid hormones; locomotion; salmonid; teleost.**

Increased secretion of the glucocorticoid hormones cortisol and corticosterone is a principal component of the physiological stress response in vertebrates (Wingfield, 1994; Pickering and Pottinger, 1995; Mommsen, Vijayan, and Moon, 1999; Sapolsky, Romero, and Munck, 2000). These steroid hormones play a major role in maintaining a balanced energy homeostasis by stimulating some processes (e.g., gluconeogenesis) and inhibiting others (e.g., digestion), and increased glucocorticoid levels during stress may also serve to restrain some of the body's own defense mechanisms (e.g., the immune system).

In addition, glucocorticoids have behavioral effects that are mediated through genomic and nongenomic effects in the brain (see, e.g., Sandi, Venero, and Guaza, 1996; Oitzl, Van Haarst, and De Kloet, 1997; Haller, Halasz, Makara, and Kruk, 1998; Marinelli, Aouizerate, Barrot, Le Moal, and Piazza, 1998; Orchinic, 1998; Moore and Evans, 1999; Rose, 2000), as well as through effects on energy homeostasis (Haller, 1995). On the other hand, behavior affects glucocorticoid levels (e.g., Sapolsky, 1990; Sander, 1992; Øverli, Harris, and Winberg, 1999), and in many cases where changes in behavior and glucocorticoid hormones co-occur, causes and effects are not easily separated. For instance, in salmonid fish, as in many mammalian species (Sapolsky, 1990; Blanchard, Spencer, Weiss, Blanchard, McEwen, and Sakai, 1995; Shively, Laber-Laird, and Anton, 1997; Kramer, Hiemke, and Fuchs, 1999), socially subordinate individuals are often characterized by both chronically increased plasma cortisol levels and distinct behavioral changes, like suppressed aggressive behavior, reduced feeding, and low spontaneous locomotor activity (Ejike and Schreck, 1980; Abbott, Dunbrack, and Orr, 1985; Winberg, Nilsson, Spruijt, and Höglund, 1993; Winberg and Lepage, 1998; Øverli, Winberg, Damsgård, and

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Jobling, 1998, Øverli *et al.*, 1999). As pointed out by Gregory and Wood (1998), sustained exposure to elevated cortisol levels may well contribute to this behavioral inhibition.

It has also recently been shown that rainbow trout genetically selected for high (HR) or low (LR) post-stress cortisol levels display differences in behavior (Pottinger and Carrick, 2001; Øverli, Pottinger, Carrick, Øverli, and Winberg, 2002). In one experiment, HR trout displayed a higher level of locomotor activity than LR fish in response to a conspecific territorial intruder (Øverli *et al.*, 2002). Furthermore, LR fish started to feed earlier than HR fish after transfer to a new rearing environment (Øverli *et al.*, 2002), and LR fish became socially dominant over size-matched HR fish in staged fights for social dominance (Pottinger and Carrick, 2001). Thus, differences in stress responsiveness and behavior are interrelated in rainbow trout, and HR and LR rainbow trout may in fact represent selection for different behavioral–physiological stress coping styles, as have been identified in mammals (Koolhaas, Korte, De Boer, Van Der Vegt, Van Reenen, Hopster, De Jong, Ruis, and Blokhuis, 1999).

The causal mechanisms underlying concomitant differences in stress responsiveness and behavior in fish are, however, largely unknown. The behavioral and physiological characteristics of HR and LR rainbow trout may be functionally linked through a number of factors that influence both endocrine and behavioral responses, or through direct behavioral effects of cortisol. The behavioral profile of HR rainbow trout is consistent with some reported effects of cortisol in poikilotherms (decreased appetite and competitiveness: Gregory and Wood, 1998; increased locomotor activity: Cash and Holberton, 1999). Behavioral effects of cortisol are, however, poorly studied in teleosts, and the fact that behavioral effects of cortisol are likely to be dose-, time-, and context-dependent has not been given attention. We therefore decided to study the effect of short-term (1 h) and long-term (48 h) treatment with exogenous cortisol in juvenile rainbow when reared alone and when subjected to a conspecific intruder after adaptation to rearing in isolation. Two behavioral patterns that previously have been shown to be affected by social interactions as well as selection for stress responsiveness in salmonid fish were studied: aggression (in intruder-tested fish) and locomotor activity (in intruder-tested as well as undisturbed fish). In other vertebrate groups, long-term glucocorticoid exposure repeatedly has been found to inhibit both aggression and the activity level, while short-term treatment acts in the opposite direction (Leshner, Korn, Mixon, Rosenthal, and Besser, 1980; Wingfield and Silverin, 1986; Tokarz, 1987; Hayden-Hixon and Ferris, 1991; DeNardo and Licht, 1993; Sandi *et al.* 1996; Haller, Albert, and Makara, 1997; Breuner, Greenberg, and Wingfield, 1998). The consistency of the above observations over a range of species suggests that cortisol might have similar time-dependent effects on behavior in teleost fish. Thus, it could be hypothesized that short-term treatment with this hormone leads to increased locomotor activity and aggression in rainbow trout, while sustained exposure should be inhibitory.

METHODS

Subjects and Housing

The experimental fish were juvenile rainbow trout weighing 64.4 ± 1.0 g (mean ± SEM) that had been obtained from a commercial fish farm. Prior to the experiment, the fish had been kept indoors in a 1-m^3 holding tank at a rearing density of approximately 0.04 kg/L for >2 weeks. The holding tank was continuously supplied with aerated Uppsala tap water at 8–11°C and the light–dark regimen was continuously adjusted to conditions at 51° north latitude. In the holding tank and throughout the experiment, fish were fed daily with commercial trout pellets (EWOS ST40) at 1% of their body weight.

The experiment was conducted in glass aquaria (100 × 50 × 50 cm) continuously supplied with aerated tap water (0.9 L/min, 8–10°C). Light (12 h:12 h/light:dark) was provided by 2 × 30-W Lumilux daylight fluorescent tubes placed 100 mm above the water surface. Each aquarium was divided into four 50-L compartments by removable PVC walls. Rainbow trout were transferred from the holding tank, weighed, and reared in isolation (one fish per 50-L compartment) for 6 days. During acclimation, fish were hand-fed pelleted food once daily, between 1000 and 1400 h. The amount of food was gradually increased from just a few pellets on Day 1 up to an amount corresponding to 1% body weight at Day 6. Fish that did not eat after 6 days in the experimental aquaria (approximately 10% of the individuals originally transferred from holding tanks) were discarded from the experiment.

Cortisol Treatment

At Day 7 of rearing in isolation, the daily meal of 48 fish was replaced with pellets that had been treated with cortisol. Cortisol food was prepared by immersing the food in 96% ethanol containing dissolved cortisol (hydrocortisone, 11β,17α,21-trihydroxypregn-4-ene-3,20-dione, Sigma Chemical) corresponding to 600 mg cortisol kg^{-1} food. After evaporation of the ethanol cortisol remained incorporated into the diet (Gamperl, Vijayan, and Boutilier, 1994), and a 1% body weight meal constituted a dose of cortisol corresponding to 6 mg kg^{-1} fish. Controls (n = 20) were given the same amount of untreated food. A possible problem with using this method of cortisol administration in long-term studies may arise from the potentially negative effects of chronic cortisol elevation on food intake (Gregory and Wood, 1998). However, in the current experiment at the end of the acclimation period the fish readily consumed the daily ration within 1–2 min of delivery, even following repeated cortisol treatment.

Among the cortisol-treated rainbow trout, 24 fish were observed and sampled on the same day as the first meal was given (see below), and 24 fish were given cortisol food once daily for another 2 days, corresponding to approximately 48 h of exposure to elevated cortisol levels. In the 48-h group, the amount of cortisol in the food was reduced to 75% of the original dose on Day 2 of cortisol feeding, and then to 50% on Day 3. Reduced doses were given since a pilot experiment revealed that the full dose given on 3 consecutive days would elevate plasma cortisol to levels exceeding those normally observed in rainbow trout even after severe stressors (>200 ng mL^{-1}, data not shown). Controls and cortisol-treated fish were kept in separate, but similar aquaria.

Behavioral Observations and Cortisol Assay

Video recording of controls and short-term cortisol-treated fish were started 1 h after feeding on Day 7 after transfer to rearing in isolation, and lasted 30 min. Long-term cortisol treated-fish were filmed from 1 h after the last cortisol meal, on Day 9 of rearing in isolation. When video recording started, intruders (rainbow trout weighing between 40 and 60% of the resident fish) were introduced into the aquaria of half of the fish from each treatment group, while half of the fish were filmed when undisturbed. All fish in a partitioned aquarium were filmed simultaneously, and were treated similarly (i.e., cortisol–no cortisol, intruder–no intruder). After 30 min the observed fishes was netted and anesthetized in 0.5 mL L^{-1} 2-phenoxy ethanol, and a blood sample was obtained from the caudal vessels using a heparinized syringe. Following centrifugation at 1500g for 3 min plasma aliquots were frozen and kept at −80°C, until plasma cortisol was analyzed by a previously validated radioimmunoassay (Winberg and Lepage, 1997; Winberg, Nilsson, Hylland, Södersröm, and Nilsson, 1997; modified from Olsen, Falk, and Reite, 1992) (minimum detectable level 0.5 ng/mL, intraassay coefficient of variation 2.1%, interassay coefficient of variation 7.1%). All behavioral observations and sampling were carried out between 900 and 1400 h during 4 weeks in October–November 2000.

In total, six experimental groups were formed (controls, short-term cortisol, and long-term cortisol, with or without intruder), with group n either n = 10 (controls) or n = 12 (cortisol-treated fish). From the videotapes, the following parameters were recorded: For all experimental fish, swimming activity was calculated as percentage time spent swimming (Øverli *et al.*, 2002) during the first 15 min from the start of filming (i.e., 60–75 min after feeding). For intruder-tested fish, latency until the resident fish attacked the intruder and the number of aggressive acts performed against the intruder for 15 min after the first attack were recorded in addition to swimming activity. An aggressive act was scored when resident fish either (a) approached the intruder rapidly, resulting in the intruder being displaced and fleeing; (b) approached and bit the intruder, whether or not the bite resulted in the intruder fleeing; or (c) approached and chased the intruder at least twice the intruder's body length, with or without biting it. The quality of the video recordings did not allow for registration of more subtle behavioral patterns, like lateral and frontal displays. If the resident fish did not attack the intruder within 30 min of its introduction, 0 aggressive acts were scored, and latency was set to 30 min.

Data Analysis

Data on locomotor activity were square root transformed to obtain homogeneity of variance, before being analyzed by two-way ANOVA with cortisol treatment and and intruder as independent variables and activity as the dependent variable. This ANOVA procedure was followed by the least significant difference (LSD) post hoc test, to determine P values for differences between specific experimental groups. Cortisol concentrations were analyzed as above, without prior

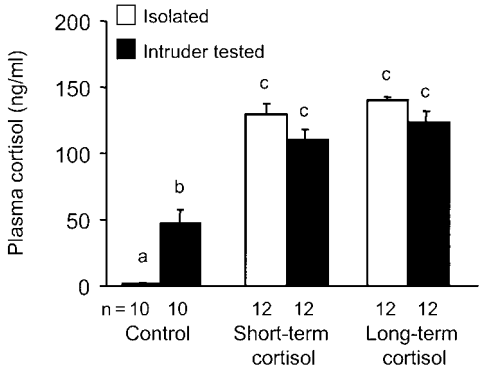

FIG. 1. Total plasma cortisol concentrations (mean + SEM) in controls and short-term (1.5 h), and long-term (48.5 h) cortisol-treated rainbow trout that were either sampled from rearing in isolation or subjected to an intruder test. Experimental groups assigned different letters displayed statistically significant differences in plasma cortisol (two-way ANOVA followed by LSD post hoc test).

transformation. Aggression data (number of aggressive acts registered during 15 min) from the intruder-tested fish were log transformed according to the formula log (aggressive acts +1), before being analyzed by one-way ANOWA followed by the LSD post hoc test. Latency to attack in intruder-tested fish was analyzed as above, but without prior transformation.

RESULTS

Plasma Cortisol

Similar to what has been shown previously (Pickering and Duston, 1983; Pickering, 1984; Barton, Schreck, and Barton, 1987), the method of incorporating cortisol into the fish diet resulted in relatively rapid (< 1.5 h) and highly reproducible increases in plasma cortisol levels in rainbow trout (Fig. 1). The effect of cortisol treatment on plasma cortisol was highly significant ($F(2,62) = 142.98$, $P < 0.001$). There was no overall effect of the intruder on plasma cortisol, but a significant interaction effect of cortisol treatment and the presence of the intruder was indicated ($F(2,62) = 13.91$, $P < 0.001$). This result is probably explained by the fact that post hoc comparisons revealed that intruder-tested controls had significantly higher cortisol levels than controls sampled directly from rearing in isolation, while no effect of the intruder was seen in cortisol-treated fish (Fig. 1).

At 1.5 and 48.5 h after introduction of cortisol-treated food, plasma cortisol levels in the cortisol-fed fish resembled those observed in highly stressed rainbow trout (>100 ng/mL). Some, but not all, socially subordinate fish reached such levels after sustained (24 h) social stress during pair rearing (Øverli *et al.*, 1999). In comparison, a brief stressor (90 s handling and confinement) elevated plasma cortisol to about 40 ng/mL in rainbow trout (Barton, Peter, and Paulencu, 1980). In accordance with what has been shown previously (Øverli *et al.*, 1999), rainbow trout fed normal food had very low cortisol levels (<5 ng/mL) when sampled from rearing in isolation.

Locomotor Activity

Figure 2 shows the locomotor activity of the various experimental groups calculated as percentage time spent moving during 15 min. The effect of cortisol treatment alone on locomotor activity did not reach statistical significance, but there was a highly significant effect of the intruder ($F(2,62) = 15.83$, $P < 0.001$), and there was also a significant interaction effect between cortisol treatment and the presence of an intruder ($F(2,62) = 6.42$. $P = 0.003$). In post hoc tests, both controls and short-term cortisol-treated fish displayed significantly higher locomotor activity during the intruder test than what was seen in corresponding experimental groups reared in isolation (post hoc: $P = 0.04$ and $P < 0.001$, respectively), but there was no such difference between isolated and intruder-tested individuals among long-term cortisol-treated fish. In fact, 48 h cortisol treatment apparently led to an inhibition of locomotor activity in intruder-tested fish, as

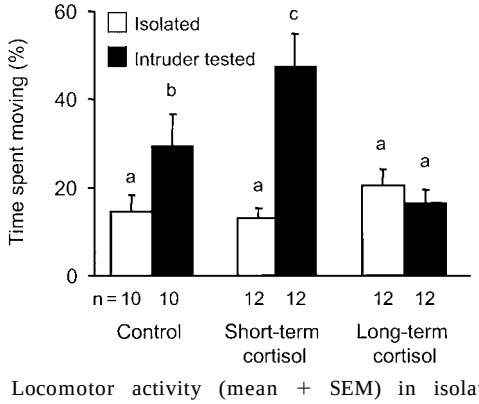

FIG. 2. Locomotor activity (mean + SEM) in isolated and intruder-tested controls and fish subjected to short- or long-term cortisol exposure, calculated as percentage time spent swimming between 60 and 75 min after food had been distributed. Experimental groups assigned different letters displayed statistically significant differences in locomotor activity (two-way ANOVA followed by LSD post hoc test).

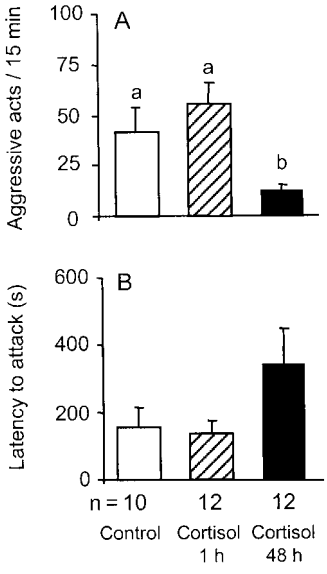

FIG. 3. Number of aggressive acts (mean + SEM) registered during 15 min after the first attack (A) and latency to first attack (B) (mean + SEM) in intruder-tested controls and fish subjected to short- or long-term cortisol exposure. Experimental groups assigned different letters displayed statistically significant differences in aggression (ANOVA followed by LSD post hoc test).

compared with intruder-tested controls (post hoc: $P = 0.04$) as well as short-term cortisol-treated fish (post hoc: $P < 0.001$). Short-term cortisol treatment had the opposite effect on locomotor activity: In the observation period between 60 and 75 h min after cortisol administration, intruder-tested cortisol fish moved significantly more than intruder-tested controls (post hoc: $P = 0.03$). Effects of cortisol treatment on locomotor activity were not evident in undisturbed fish.

Aggressive Behavior

Aggression appeared to follow the same general pattern as locomotor activity (Fig. 3). The intruder-tested groups varied within a single variable (cortisol treatment), for which the one-way ANOVA indicated a highly significant effect ($F(2,31) = 6.01$, $P = 0.007$). Post hoc tests revealed a significant reduction in the number of aggressive acts performed against the intruder in the 48-h cortisol group, as compared with both controls (post hoc: $P = 0.02$) and short-term cortisol-treated fish (post hoc: $P = 0.002$) (Fig. 3A). There was, however, no significant difference between controls and short-term cortisol-treated fish. There was also a nonsignificant trend toward increased latency to first attack in long-term cortisol-treated fish, but it should be noted that the apparently large elevation in mean latency for the long-term group (Fig. 3B) was dependent on two individuals failing to attack the intruder within 30 min and, therefore, scoring an attack latency of 900 s.

DISCUSSION

Administration of exogenous cortisol through food is noninvasive and stress free. This method is therefore especially well suited to examine short-term effects of cortisol (or other steroid hormones) on behavior (see Breuner *et al.*, 1998), avoiding the complications induced by capture and injection of the animals. In the current study, effects of exogenous cortisol on locomotor activity were evident only in intruder-tested fish, and these effects were opposite after short- and long-term cortisol treatment. The intruder elicited increased locomotion in control fish, and this response was significantly enhanced by short-term cortisol treatment. In contrast, long-term cortisol treatment abolished the response, so that locomotor activity was similar in intruder-tested and undisturbed fish (cf. Fig. 2). These data suggest a phylogenetically conserved response, as Sandi *et al.* (1996) demonstrated a stimulatory effect of corticosterone on locomotor activity in rats at 7.5 and 15 min, but not 60 min, following injection. (In the present study, behavioral observations were started 1 h after cortisol administration, since poikilotherm animals were studied.) Sandi *et al.* (1996) also demonstrated a context-dependent effect, since increased locomotor activity was seen in rats transferred to a novel environment, but not in animals that had previously been exposed to the test environment. Additional data presented by Sandi *et al.* (1996) support the idea that the rapid effect of corticosterone was mediated by a nongenomic mechanism, since the effect was not abolished either by the protein synthesis inhibitor cycloheximide or by specific glucocorticoid and mineralocorticoid receptor antagonists.

Similarly, Breuner *et al.* (1998) showed that administration of corticosterone-injected mealworms to white-crowned sparrows (*Zontrichia leucophrys gambelii*) leads to a rapid (within 7 min) elevation of plasma corticosterone and activity (perch hopping) in the birds. This effect was later shown to be both dose dependent and modulated by photoperiod (Breuner and Wingfield, 2000). In the currently reported experiment, a stimulatory effect of cortisol on locomotor activity was seen in intruder-tested fish when ob-

served between 60 and 75 min after cortisol administration, but not after 48 h of cortisol exposure. Aggressive behavior was also inhibited by long-term cortisol treatment, but a stimulatory effect of short-term treatment, as has been observed in mammals (e.g. Hayden-Hixon and Ferris, 1991; Haller *et al.*, 1997), could not be confirmed statistically (cf. Fig. 3). Previous studies have reported a reduction of aggressive behavior as a result of long-term cortisol treatment in lizards (Tokarz, 1987; DeNardo and Licht, 1993), birds (Wingfield and Silverin, 1986), and mammals (Leshner *et al.*, 1980). To our knowledge, there has been only one previous study on the effects of cortisol on agonistic behavior in teleost fish. Using an immersion technique, Munro and Pitcher (1985) found that cortisol decreased aggression and increased submissive behavior in the cichlid *Aequidens pulcher*, while testosterone had the opposite effect.

Several molecular mechanisms may be involved in the switch from a stimulatory to an inhibitory effect of cortisol on behavior. In mammals, a shift in the balance between glucocorticoid (GR) and mineralocorticoid (MR) receptor activation is an important factor in mediating the time- and dose-dependent effect of corticosteroids on behavior and neural activity (Oitzl *et al.*, 1997; Joëls and Vreugendhil, 1998; de Kloet, 2000; Sapolsky *et al.*, 2000). Teleost fish do not appear to produce aldosterone (Sandor and Mehdi, 1980), so cortisol is involved in the regulation of both metabolism and salt/water balance in fish (Laurent and Perry, 1989; Madsen, 1990; Bern and Madsen, 1992; Pickering and Pottinger, 1995; Wendelaar Bonga, 1997; Mommsen *et al.*, 1999). Both GR- and MR-like receptors have however been cloned in rainbow trout (Ducouret, Tujague, Ashraf, Mouchel, Valotaire, and Thompson, 1995; Takeo, Hata, Segawa, Toyohara, and Yamashita, 1996; Colombe, Fostier, Bury, Pakdel, and Guiguen, 2000), and the distribution of GR receptors in the forebrain of rainbow trout was described by Teitsma, Bailhache, Tujague, Balment, Ducouret, and Kah (1997). The precise role of these steroid receptors in behavior has not been explored in fish, but GR receptor autoregulation has been demonstrated in rainbow trout (Pottinger 1990; Lee, Goodrich, Struve, Yoon, and Weber, 1992), and this mechanism may have contributed to the differential effect of long- and short-term cortisol treatment. A role for membrane-bound steroid receptors in behavior has been described in amphibians (Orchinik, Murray, and Moore, 1991; Moore and Orchinik, 1994), but has to our knowledge not been demonstrated in teleost fish. Other rapid effects of cortisol have, however, been reported in fish (Shih, Chou, Chi, Tchen, and Lo, 1990; Borski, Helms, Richman, and Grau, 1991). For instance, prolactin release from tilapia (*Oreochromis mossambicus*) pituitaries in an *in vitro* preparation was blocked by cortisol, and this effect became significant within 20 min (Borski *et al.*, 1991). Thus, the timing of the current experiment was such that neither genomic nor nongenomic effects of cortisol could be excluded in the short-term cortisol group.

Since cortisol treatment was without effect on locomotor activity in undisturbed fish, it could be assumed that the behavioral effects of cortisol were mediated through interaction with other signal systems that were activated when the experimental fish was challenged by the presence of an intruder. Excitatory and inhibitory signals mediated by amino acids are modulated by glucocorticoids (reviewed by Joëls, 1997). Glucocorticoids have also been shown to facilitate dopamine-mediated behaviors, like locomotor activity (Marinelli *et al.*, 1998). Glucocorticoids may influence catecholaminergic systems through effects on neurotransmitter release, monoamine oxidase, uptake sites, and pre- and postsynaptic receptor densities (Veals, Korduba, and Symchowicz, 1977; Gilad, Rabey, and Gilad, 1987; Piazza, Rougé-Pont, Deroche, Maccari, Simon, and Le Moal, 1996; Marinelli *et al.*, 1998; Day, Campeau, Watson, and Akil, 1999; Lammers, D'Souza, Qin, Lee, Yajima, and Mouradian, 1999; Rougé-Pont, Abrous, Le Moal, and Piazza, 1999). Brain serotonergic activity, which has been shown to be increased by corticosterone in lizards (Summers, Larson, Ronan, Hofmann, Emerson, and Renner, 2000), may be altered directly or through glucocorticoid effects on precursor availability or synthetic enzymes (Chaouloff, 2000). Glucocorticoids also have regionally dependent effects on brain corticotropin-releasing hormone (CRH, or corticotropin-releasing factor, CRF) gene expression (Schulkin, Gold, and McEwen, 1998), and CRH in turn has been shown to stimulate locomotor activity by direct actions on brain neurons (Moore, Roberts, and Bevers, 1984; Lowry and Moore 1991; Lowry, Rose, and Moore, 1996). The extensive cross-talk between glucocorticoids and other signal systems undoubtedly contributes to the time, dose, and context dependency of the behavioral significance of these hormones.

The time-dependent behavioral response to stress hormones appears to have adaptive significance. The following argument was made by Haller *et al.* (1998):

(i.e., a fight or flight type of response) should be attempted. If, on the other hand, the organism is not able to escape a chronic, or repeated, stressor for a long time, it would be better off entering a passive, energy-saving wait-and-see mode (i.e., a conservation-withdrawal response). A similar argument can be made about the behavioral inhibition seen in socially stressed animals: A passive strategy suitable to minimizing confrontation and competition with established dominant individuals is usually observed in chronically subordinate animals (Sapolsky, 1990; Blanchard *et al.*, 1995; Winberg and Nilsson 1993; Shively *et al.*, 1997; Øverli *et al.*, 1998). During the formation of new hierarchies, on the other hand, the competitive abilities of opponents are not known, and individuals might gain from actively participating in the competition.

In conclusion, the current results confirm that glucocorticoids have time- and context-dependent effects on aggression and locomotor activity in rainbow trout, in much a similar manner as has been reported in mammals. Thus, glucocorticoids are probably involved in the mediation of behavioral effects of stress in salmonids, and may well contribute to the different behavioral profile of rainbow trout selected for high and low poststress cortisol concentrations (Pottinger and Carrick, 2001; Øverli *et al.*, 2002). These observations add to a growing body of literature indicating that central signaling systems involved in the control of physiological and behavioral stress responses are phylogenetically conserved among vertebrates (Winberg and Nilsson, 1993; Winberg *et al.*, 1997).

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