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Review

Corticosteroids: Friends or foes of teleost fish reproduction?

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ABSTRACT

Reproduction in vertebrates is controlled by the Hypothalamus–Pituitary–Gonad axis and the main hormone actions have been extensively described. Still, despite the scattered information in fish, accumulating evidence strongly indicates that corticosteroids play essential roles in reproductive mechanisms. An integrative approach is important for understanding these implications. Animal husbandry and physiological studies at molecular to organismal levels have revealed that these corticosteroids are regulators of fish reproductive processes. But their involvements appear strongly contrasted. Indeed, for both sexes, corticosteroids present either deleterious or positive effects on fish reproduction. In this review, the authors will attempt to gather and clarify the available information about these physiological involvements. The authors will also suggest future ways to prospect corticosteroid roles in fish reproduction.

Contents

1. Introduction

In teleost fish, corticosteroids are involved in a wide range of physiological regulations in the fields of stress, immune and inflammatory responses, energetic metabolism and osmoregulation. Although these roles are well documented (Wendelaar Bonga, 1997; Mommsen et al., 1999), corticosteroid functions in reproduction have received limited attention. Most of the studies related to reproductive endocrinology focused on the classical endocrine actors of the brain–pituitary–gonad axis such as GnRH, gonadotrophins or gonadal sex-steroids. Until now, data about corticosteroids are quite scattered. Still, increasing clues suggest the relevance to focus on them in order to refine our knowledge about the endocrine control of fish reproduction. Reports about the inhibitory and stimulatory effects of corticosteroids and stress on reproduction are sometimes controversial. The aim of this review is to clarify these equivocal effects. Based on recent studies in the area, we examined their possible physiological roles in regulating reproduction. Initially we provide a brief overview of the major involvements of corticosteroids in mammals, before outlining the available information in fish and finally discussing how to prospect new research strategies for the exploration of corticosteroid functions in the future.

2. The main corticosteroid roles in mammal reproduction

Corticosteroids are steroid hormones produced from cholesterol by the adrenal cortex mainly.

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They are classically divided into two groups: the glucocorticoids and the mineralocorticoids. Many cell types contain receptors that bind glucocorticoids allowing these hormones (mainly cortisol in non-rodents and corticosterone in rodents) to control numerous biological processes such as carbohydrate, lipid and protein metabolism. They also play major anti-inflammatory roles and regulate the immune response. Mineralocorticoids, mainly represented by aldosterone, regulate hydromineral balance, mainly by promoting sodium retention in the kidney and acting *via* mineralocorticoid receptor. But there are evidences that mineralocorticoids are involved in other biological processes such as the regulations of cardio-vascular system (Struthers, 1995; Duprez, 2007; Fejes-Toth and Naray-Fejes-Toth, 2007) or structural proteins (Gekle et al., 2007). Furthermore, the presence of corticosteroid receptors in reproductive tissues including ovary and testis leads to the consideration that corticosteroids can exert positive or inhibitory effects on reproductive functions.

Regarding females, there is a general consensus to say that glucocorticoids can exert inhibitory effect on the whole Hypothalamus–Pituitary–Gonad axis including ovarian steroidogenesis (Michael et al., 1993). Therefore, it is hypothesized that they are detrimental to reproduction. However, in a recent review, Tilbrook et al. (2000) underscored that *in vivo* disruptive effects of glucocorticoids in non-rodent mammals are not systematic. Actually, according to Brann and Mahesh (1991), corticosteroids may also play a positive role in the regulation of follicle development, as acutely elevated levels may increase both FSH and LH releases, contrary to chronic corticosteroid exposure.

There is also no general agreement about the roles of corticosteroids and especially glucocorticoids in the regulation of oocyte maturation. In human and other mammals, there is a preovulatory increase of follicular cortisol content during the luteinizing hormone surge leading to ovulation, while levels are reduced during the rest of the menstrual cycle (Harlow et al., 1997; Andersen, 2002; Acosta et al., 2005). It has been suggested that cortisol exerts a positive action during ovulation in human (Fateh et al., 1989; Jimena et al., 1992; Andersen, 2002) but it had no effect on oocyte maturation in mouse (Andersen, 2003) and was suggested to be inhibitory in pig, albeit at pharmacological doses (Yang et al., 1999). In human, the increase of intrafollicular cortisol would result from an increase of cortisone uptake from the circulating fluid. There, this latter would be converted into cortisol by 11β-HSD enzyme that would switch from type-2 into type-1 near the ovulation period (Lewicka et al., 2003). Nevertheless, it is not clear yet whether the peri-ovulatory increase of follicular cortisol is a cause or a consequence of human oocyte maturation (Michael, 2003). According to Hillier (2001), it might be involved in

Table 1
Plasma concentrations of corticosteroids in the plasma of some immature and mature, male and female teleosts (ng/mL).

Steroids	Species	Reproductive status	Female	Male	Reference	Method
11-dehydrocorticosterone	*Chalcalburnus tarichi*	Mature	**22–38**		Unal et al. (2006)	HPLC
11-deoxycortisol	*Pleuronectes americanus*	Mature		**0.6–1.9**	Campbell et al. (1976)	Double isotope derivative assay
11-deoxycortisol	*Pleuronectes platessa*	Mature	**32**		Scott and Canario (1990)	HPLC + RIA
11-deoxycortisol	*Hoplostethus atlanticus*	Mature	**2–5**	**2–4**	Pankhurst and Conroy (1988)	RIA
		Immature	***1–3***			
11-deoxycortisol	*Perca fluviatilis*	Mature	**1–9**		Noaksson et al. (2005)	HRGC/HRMS
		Immature	***1–5***			
11-deoxycortisol	*Oncorhynchus mykiss*	Mature	**18.1**	**4.8**	Campbell et al. (1980)	Double isotope derivative assay
11-deoxycortisol	*Oncorhynchus mykiss*	*Immature*	***0–3***		Doyon et al. (2006)	RIA
11-deoxycorticosterone	*Cyprinus carpio*	Mature	**<0.1**		Kime and Dolben (1985)	RIA
11-deoxycorticosterone	*Oncorhynchus mykiss*	Mature		**1**	Milla et al. (2008)	RIA
		Immature		***0.02–0.1***		
11-deoxycorticosterone	*Oncorhynchus mykiss*	Mature	**2**	**3.1**	Campbell et al. (1980)	Double isotope derivative assay
11-deoxycorticosterone	*Tilapia aurea*	Mature	**22.2**		Katz and Eckstein (1974)	Chromatography + isotope derivative assay
		Immature	***0.6***			
11-deoxycorticosterone	*Pleuronectes americanus*	Mature	**0.5**	**1**	Campbell et al. (1976)	Double isotope derivative assay
Cortisol	*Pleuronectes platessa*	Mature	**12–15**	**11–14**	Wingfield and Grimm (1977)	RIA
		Immature	***1–3***	***1–4***		
Cortisol	*Dicentrarchus labrax*	Mature	**60**		Rocha and Reis-Henriques (1999)	RIA
		Immature	***8***			
Cortisol	*Platichthys flesus*	Mature	**40**		Lu et al. (2007)	RIA
		Immature	***5–15***			
Cortisol	*Fundulus heteroclitus*	Mature	**30–80**		Bradford and Taylor (1987)	RIA
		Immature	***10–15***			
Cortisol	*Oncorhynchus mykiss*	Mature		**20–25**	Hou et al. (2001)	RIA
		Immature		***2–4***		
Cortisol	*Oncorhynchus mykiss*	Mature	**70**		Koldkjær et al. (2004)	RIA
		Immature	***10–20***			
Cortisol	*Oncorhynchus tshawytscha*	Mature		**397**	Barry et al. (2001)	ELISA
		Immature		***76***		
Cortisol	*Oncorhynchus masou*	Mature	**300–400**	**300–350**	Westring et al. (2008)	TR-FIA
		Immature	***30–90***	***40–70***		
Cortisol	*Oncorhynchus nerka*	Mature		**52**	Woodhead (1975)	Chromatography
Cortisol	*Oncorhynchus nerka*	Mature	**457**		Carruth et al. (2000)	RIA
		Immature	***259***			
Cortisol	*Oncorhynchus nerka*	Mature	**140–200**	**50–85**	Kubokawa et al. (1999)	RIA
Cortisol	*Salmo trutta*	Mature	**15–50**	**5–20**	Pickering and Christie (1981)	RIA
		Immature	***1–7***	***1–7***		
Cortisol	*Catostomus commersoni*	Mature	**71–120**	**57–89**	Scott et al. (1984)	RIA
Cortisol	*Piaractus mesopotamicus*	Mature	**125–140**		Gazola et al. (1996)	RIA
		Immature	***10–30***			
Corticosterone	*Oncorhynchus nerka*	Mature		**73**	Woodhead (1975)	Chromatography
Cortisone	*Oncorhynchus nerka*	Mature		**61**	Woodhead (1975)	Chromatography
Cortisone	*Salmo salar*	Mature	**70–160**	**50**	Idler et al. (1964)	Chromatography
Cortisone	*Pleuronectes americanus*	Mature	**7.9**	**12.7**	Campbell et al. (1976)	Double isotope derivative assay
		Immature	***0.8–1.8***	***1.2–1.4***		

the anti-inflammatory response to tissue injuries caused by ovulation. To date, the role of cortisol during peri-ovulatory period has not been entirely elucidated.

Prenatal maternal stress has been found to have long-lasting effects on the behavioural and physiological development of the offspring. In the case of human, this maternal stress is often associated to an increase of cortisol in the women plasma and in the foetus (De Weerth and Buitelaar, 2005). During gestation, cortisol levels normally gradually increase in the serum and amniotic fluid of pregnant woman (Challis et al., 1983; Sarkar et al., 2007). High expression of 11β-HSD type-2, which oxidizes cortisol into cortisone, has been measured in both foetus and placenta from mid-gestation to parturition (Seckl and Chapman, 1997). Both low expression of this enzyme or high cortisol/dexamethasone levels in the pregnant female/woman were associated to decrease placental weight and lower bodyweight at birth (Seckl and Chapman, 1997; reviewed by Michael and Papageorghiou, 2008). It was hypothesized that 11β-HSD2 may protect the foetus from deleterious actions of active cortisol/corticosterone (Sun et al., 1999). These deleterious effects may happen during early, mid- or late pregnancy and impact foetus but also further child/young animal development (e.g. hypertension, post-natal learning capacities, cognitive function and response to stress) (Michael and Papageorghiou, 2008). Regarding the role of mineralocorticoids, aldosterone production increases during pregnancy in order to allow the water retention needed for volume expansion (Escher and Mohaupt, 2007).

In males, glucocorticoids and especially cortisol or corticosterone seem to have an inhibitory effect all along the spermatogenesis process (Weber et al., 2000). These glucocorticoids inhibit testosterone production within the Leydig cells *via* a pathway mediated by the glucocorticoid receptor (Ge et al., 2005a). In addition, glucocorticoids may induce spermatogonia and spermatocyte apoptosis and decrease sperm yield (Gao et al., 2003; Wagner and Claus, 2004). However, according to Wagner and Claus (2004), it is not clear whether these effects are part of a normal regulative step or detrimental to the animal. The activity of 11β-hydroxysteroid dehydrogenase (11βHSD2) within Leydig/Sertoli cells would protect the testis from the adverse effects of cortisol or corticosterone by converting these hormones into inactive cortisone or 11-dihydrocorticosterone, respectively (Nacharaju et al., 1997; Ge et al., 2005a).

Contrary to glucocorticoids, mineralocorticoids seem to exert some positive actions in male reproduction. Mineralocorticoid receptor was found to be expressed in Leydig and Sertoli cells, as well as in spermatozoa (Ge et al., 2005b; Fiore et al., 2006). The mineralocorticoid hormone aldosterone might be involved in the regulation of spermatic fluid osmolarity and in spermatozoa motility (Fiore et al., 2006). In addition it appears that it would also stimulate testosterone production within the Leydig cells (Ge et al., 2005b).

3. The corticosteroids in fish

Corticosteroids are mainly synthesized in the interrenal tissue in teleost fish. This tissue which is embedded inside the anterior part of the kidney is homologous to mammalian adrenal cortex. Contrary to mammals, the interrenal tissue does not form a compact gland and the anatomical distinction between cortical and medullary zonations is lacking. Indeed, the postcardinal veins and their branch are surrounded by close clusters of chromaffin cells (medullary homologue) and steroidogenic cells (cortical homologue) (Wendelaar Bonga, 1997). Whereas the corticosteroid biosynthesis pathways differ between fish and mammals (Prunet et al., 2006), there are not great differences between the corticosteroid pattern. The main divergence is the absence of the mineralocorticoid aldosterone in fish (Gilmour, 2005). Otherwise, to our knowledge, 18-hydroxycorticosterone and 1α-dehydroxycorticosterone have not been detected in the teleost plasma yet. In conformity with the findings in other vertebrates, the main corticosteroids isolated from fish blood are cortisol, cortisone, 11-deoxycortisol and corticosterone. But, their concentrations depend on the species, sex and reproductive status (Table 1).

Female and male gonads may have also the capacity to produce the main corticosteroids (cortisol, 11-deoxycortisol, corticosterone and 11-deoxycorticosterone). First, in the fish species investigated, they own all the enzymes responsible for their synthesis even if 11-β hydroxylase gene expression was not abundant in the ovary (Figs. 1 and 2: Kobayashi et al., 1998; Kusakabe et al., 2002; Kazeto et al., 2003; Li et al., 2003; Socorro et al., 2007; Zhou et al., 2007). Second, 11-deoxycorticosteroids (11-deoxycortisol and 11-deoxycorticosterone) were shown to be important products of ovarian and/or testicular steroidogenesis (Colombo et al., 1973, 1978; Tesone and Charreau, 1980; Kime et al., 1992). Third, the presence of cortisol and 11-deoxycortisol in ovary, sperm and seminal fluid supports that gonadal corticosteroidogenesis (Canario and Scott, 1990; Scott et al., 1991a,b).

To manage their physiological actions, corticosteroids bind to nuclear receptors which act as ligand-dependent transcription factors. The relative ability of corticosteroids to evoke the transcriptional activities of corticosteroid receptors is of paramount importance to estimate their capacity to exert physiological actions. In rainbow trout *Oncorhynchus mykiss*, cortisol and, to a lesser extent, 11-deoxycortisol

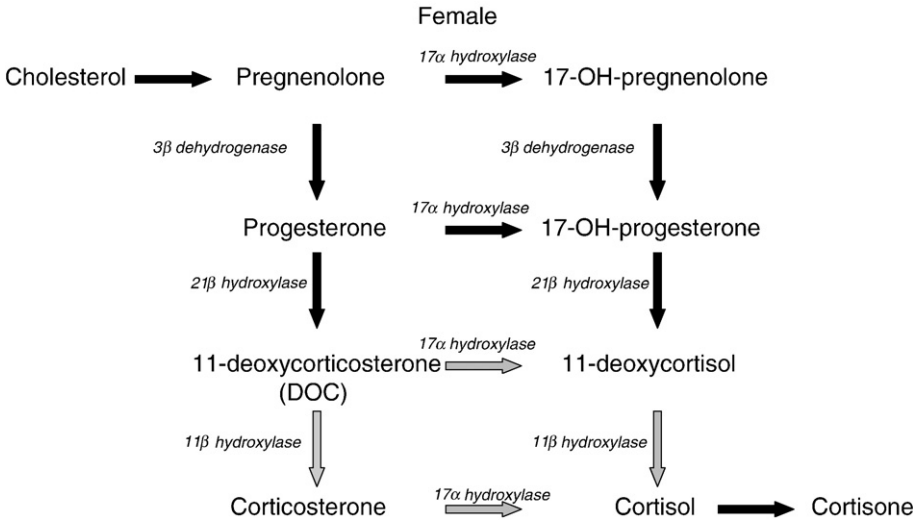

Fig. 1. Schema of the probable pathways of corticosteroid biosynthesis in female teleost. Solid black arrows show that the enzyme catalysing the steroid metabolism is active in the gonad; the grey arrows indicate that the enzyme required for that step is expressed in the gonad but its activity needs to be confirmed.

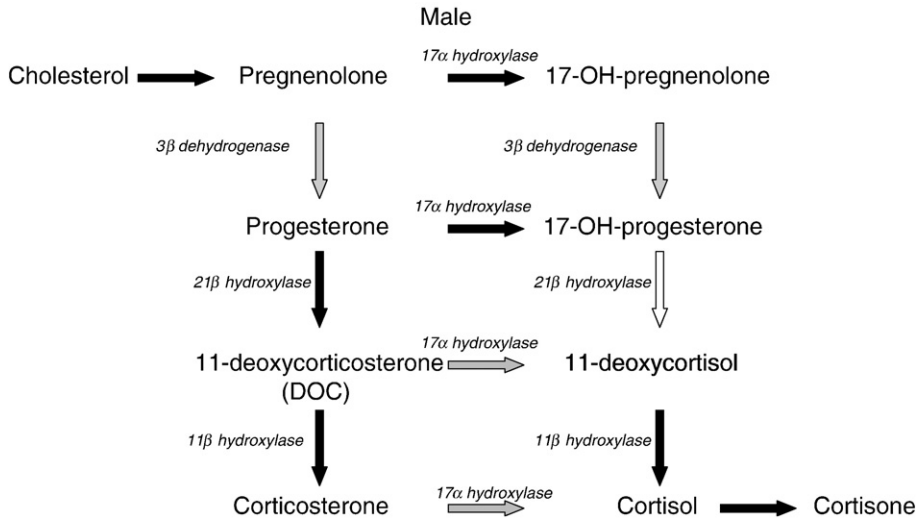

Fig. 2. Schema of the probable pathways of corticosteroid biosynthesis in male teleost. Solid black arrows show that the enzyme catalysing the steroid metabolism is active in the gonad; the grey arrows indicate that the enzyme required for that step is expressed in the gonad but its activity needs to be confirmed. Unfilled arrow indicates lack of clear information.

and corticosterone appeared to transactivate *in vitro* the two glucocorticoid receptors (Bury et al., 2003). Interestingly, the two glucocorticoid receptors but also both GR2 isoforms in the cichlid *Haplochromis burtoni* are differentially sensitive to cortisol (Greenwood et al., 2003; Bury et al., 2003). The situation is far from clear for the mineralocorticoid receptor for which both cortisol and 11-deoxycorticosterone (DOC) are likely to act as physiological ligands (Sturm et al., 2005). In *H. burtoni*, the mineralocorticoid receptor sensitivity was similar to the mammalian one, in being more sensitive to both cortisol and aldosterone than the glucocorticoid receptors (Greenwood et al., 2003). Corticosteroid receptors gene expression (gluco- and mineralocorticoid receptors) has been found in the male/female gonads. In rainbow trout, the transcripts were detected in the ovary (Sturm et al., 2005; Milla et al., 2006). Both corticosteroid receptors were also identified in the testis of trout and other species (Colombe et al., 2000; Park et al., 2007; Filby and Tyler, 2007; Milla et al., 2008). In the testis, mineralocorticoid receptor is strategically located along the reproductive axis and thus is in a position to regulate reproductive function (Milla et al., 2008). Finally, the presence of factors known to interfere with the corticosteroid actions in mammals supports the implication of corticosteroids in fish reproduction. In this regard, 11β-hydroxysteroid dehydrogenase which prevents illicit activation of the mineralocorticoid receptor in mammals by catabolizing cortisol (Farman and Rafestin-Oblin, 2001), is expressed in the ovary and testis, particularly from mid-gametogenesis to the final stages of maturation, and displays some activities at least in the testis (Kusakabe et al., 2003; Ozaki et al., 2006; Milla et al., 2006). Overall, production, receptivity and metabolic capacity of corticosteroids in the gonads are thus a first argument to suppose some potential implications in teleost reproduction.

4. Changes in plasma corticosteroids during fish reproduction

Whereas the seasonal changes of the classical sex-steroid concentrations (androgens, estrogens…) during a reproductive cycle have been extensively investigated so far, data about the variations of plasma corticosteroid concentrations are limited. Still, as a determinant indicator of corticosteroid roles in fish reproduction, their plasma levels vary greatly throughout the reproductive cycle. In both sexes, some fish species exhibit a broad increase in plasma cortisol levels during the pre-spawning or spawning period (Wingfield and Grimm, 1977; Pickering and Christie, 1981; Kusakabe et al., 2003; Noaksson et al., 2005; Westring et al., 2008) even if this result was not observed in some species like black bream *Acanthopagrus butcheri* (Haddy and Pankhurst, 1999). As plasma cortisol levels in mature fish have been measured at high concentrations in several fish species, often more elevated than in immature fish (Table 1), one can hypothesize that a plasma cortisol up-regulation at the breeding season may concern numerous species. More precisely, in rainbow trout, brown trout *Salmo trutta*, goldfish *Carassius auratus* and common carp *Cyprinus carpio* a surge in plasma cortisol was measured at the time of ovulation but such a transient increase needs to be investigated in other fish species (Cook et al., 1980; Pickering and Christie, 1981; Bry, 1985; Kime and Dolben, 1985). Few studies have compared the plasma cortisol profiles during the reproductive cycle for both sexes but it seems, at least in salmonids, that the elevation starts earlier in males than in females but that the amplitude is higher in females (Pickering and Christie, 1981; Table 1).

This difference in the plasma cortisol profile might also been species-specific as illustrated by the broad levels measured plasma of the salmon species (Table 1). This salmon example also illustrates the specificity of diadromic fish for which the preparation to mating coincides with numerous physiological changes including in the osmoregulatory and metabolic processes. In the wild, salmons experience a large and sustained rise in plasma cortisol levels during the pre-spawning and/or spawning period (Carruth et al., 2000; Westring et al., 2008). Progressive hyperplasia of the interrenal tissues coincidently with the increase of corticosteroid concentrations was described in salmons during their anadromous spawning migrations (Robertson and Wexler, 1959; Hane and Robertson, 1959). In fish, cortisol release is involved in metabolic function and osmoregulation (Mommsen et al., 1999). In particular, several studies support that cortisol would be implicated in the salmonid adaptation to freshwater by stimulating the hyperosmoregulatory mechanisms (Mc Cormick, 2001; Kiilerich et al., 2007). Also, the gonadal maturation is accompanied with changes in metabolic parameters known to be driven by cortisol, such as enhanced liver amino acid catabolism and gluconeogenesis leading to glucose increase for coping with stress during migration (Kubokawa et al., 1999). It means that this prolonged hypersecretion of cortisol may be due to reproduction but also to face with the dramatic changes in their physiology.

Except for cortisol, the plasma levels of most of the corticosteroids appear low or undetectable outside the reproductive season. However, at the mating period, substantial plasmatic amounts were measured for most of them in some fish species without any clear sex influence (Table 1). In salmonids and other fish families, corticosteroids like

11-deoxycorticosterone (DOC) and 11-deoxycortisol were reported to be highly up-regulated in the plasma at the time of reproduction, in both sexes. For instance, a 38-fold increase of plasma DOC levels was measured in *Tilapia aurea* during ovulation (Katz and Eckstein, 1974). However, these elevations (11-deoxycorticosterone, 11-deoxycortisol) were not observed in all species, highlighting probable species differences which remain to be established (Table 1). In any case, when this peak occurs, its synchronicity with blood MIS (Maturation-Inducing Steroid) increase is in agreement with a role of these corticosteroids in the final stages of reproduction (Noaksson et al., 2005; Milla et al., 2008).

5. The role of corticosteroids in the reproduction of female fish

In fish, stress causes adverse effects on female reproductive performances. Depending on the period and the intensity of stressor application, it may cause follicular atresia, advance or delay oocyte maturation and ovulation or affect egg size, fertilization success, spawning behaviour and progeny quality (Clearwater and Pankhurst, 1997; Coward et al., 1998; Schreck et al., 2001; Okumura et al., 2002). Deleterious effects of stress on vitellogenesis have been extensively studied in salmonids. For example, female brook trout *Salvelinus fontinalis* facing with acid stress had lower vitellogenin levels (Roy et al., 1990). In rainbow trout, females subjected to repeated acute stress during oogenesis produced smaller eggs, in accordance with vitellogenesis disruption (Campbell et al., 1992). These effects may be mediated by cortisol, which has been shown to interfere with vitellogenesis (Fig. 3). In Arctic charr *Salvelinus alpinus*, cortisol affected the vitellogenin production controlled by estrogen (Berg et al., 2004) and also directly inhibited the production of estrogen in rainbow trout and tilapia *Oreochromis mossambicus* (Carragher and Sumpter, 1990; Foo and Lam, 1993; Reddy et al., 1999; Pankhurst and Van Der Kraak, 2000). These results could be explained by some types of interactions between the glucocorticoid receptor and the estrogen receptor (ER). *In vivo* cortisol treatment caused a drop of cytosolic E2-binding sites in the liver and a reduction in plasma vitellogenin quantity in trout (Carragher et al., 1989; Pottinger and Pickering, 1990). Glucocorticoid receptor activation prevents estradiol from positively regulating the ER expression (Lethimonier et al., 2000). Furthermore, the glucocorticoid and estrogen receptor distributions overlap in the trout brain and pituitary indicating further potential interferences between the glucocorticoids and estrogen pathways (Teitsma et al., 1999). The information about the effects of the other corticosteroids is very scarce. Their weak ability to transactivate *in vitro* the glucocorticoid receptors (Bury et al., 2003) does not support such effect on vitellogenesis depletion.

The reported effects of corticosteroids on GnRH and gonadotropins productions are also consistent with the interference of corticosteroids with the Hypothalamus–Pituitary–Gonad (HPG) axis. The widespread expression of rainbow trout glucocorticoid receptor in the brain and pituitary shows that neurons and pituitary cells, involved in the control of the reproductive axis, are probably targets for glucocorticoids (Teitsma et al., 1999). In immature fish, cortisol treatments induced elevation of pituitary LH in eel *Anguilla anguilla* and rainbow trout. These results evoke a positive function of glucocorticoids in the first sexual maturation (Crim et al., 1981; Dufour et al., 1983; Huang et al., 1999). By contrast, in maturing brown trout, the pituitary and plasma gonadotropins were depleted by cortisol treatment (Carragher et al., 1989). In female fish, androgens are suspected to be implicated in the regulations of final stages synchronization and GnRH/gonadotropins secretion (Redding and Patino, 1993; Nagahama et al., 1994). The effects of cortisol or stress on plasma androgens suppression (Carragher et al., 1989; Campbell et al., 1994; Cleary and Pankhurst, 2000) also support that corticosteroids indirectly disrupt the HPG axis. Collectively, these works suggest that corticosteroids not only affect estrogens but also probably disrupt GnRH and gonadotropin actions. Therefore, it appears that corticosteroids may exert inhibitory effects on vitellogenesis. Nevertheless, as these works mainly focused on salmonids, it would be interesting to investigate that scheme in other fish families.

Conversely, sex-steroids regulate corticosteroid productions further supporting their reciprocal interaction along the reproductive cycle. Estradiol suppressed *in vitro* cortisol production in interrenals collected at spawning time in chinook salmon *Oncorhynchus tshawytscha*, and collected from immature kokanee salmon *Oncorhynchus nerka* (McQuillan et al., 2003), which confirms the reciprocal antagonism between both steroids even if that result was not observed in immature rainbow trout (Barry et al., 1997; McQuillan et al., 2003). By contrast, *in vivo*, estrogens were reported to promote cortisol production in immature trout (Pottinger et al., 1996). It is conceivable that this

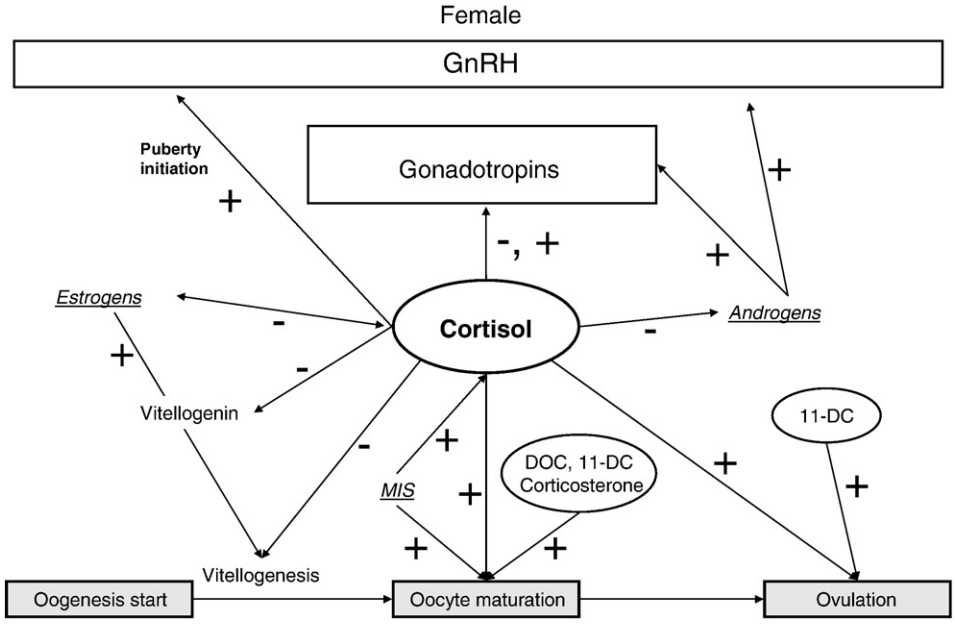

Fig. 3. Schema of the main corticosteroid implications in female teleost reproduction. MIS: Maturation-Inducing Steroid; 11-DC: 11-deoxycortisol; DOC: 11-deoxycorticosterone.

discrepancy is related to cortisol target within the Hypothalamus–Pituitary–Interrenal (HPI) axis. Also in rainbow trout, 17α-20ß-dihydroxyprogesterone which is the MIS was effective to stimulate cortisol production in the interrenal of immature fish (Barry et al., 1997). Therefore, sex-steroids may act on tissue implicated in cortisol production to modulate the whole physiological corticosteroid response.

The transient rise of plasma corticosteroids around ovulation is in agreement with their biological effects on peri-ovulatory mechanisms. In the 70s, numerous investigators attempted to identify the major steroids involved in oocyte maturation and ovulation. When identifying 17α-20ß-dihydroxyprogesterone and 17α-20ß-trihydroxyprogesterone as the MIS, corticosteroids were also intensively tested for their ability to participate in the final stages of oocyte maturation and ovulation. Treatments with LH stimulated *in vitro* follicular and interrenal synthesis of DOC and cortisol (Sundararaj and Goswami, 1969; Colombo et al., 1973) in agreement with peri-ovulatory role of both steroids. *In vivo* hCG injection in catfish *Heteropneustes fossilis* induced oocyte maturation coincidently with plasma cortisol and corticosterone increase (Mishra and Joy, 2006). Cortisol was demonstrated to be quite effective in inducing oocyte meiotic maturation in several fish species (see for review Goetz, 1983). *In vitro* tests also showed that DOC but also 11-deoxycortisol and corticosterone were able to trigger oocyte maturation in numerous freshwater species even if high doses were sometimes necessary to observe these effects (Goetz, 1983 for review; Rahman et al., 2001). In a recent work, the corticosteroid 11-deoxycortisol was even more potent than MIS to induce oocyte maturation (Unal et al., 2008). Moreover, when testing some steroid combinations, a synergy in the maturational response was demonstrated between corticosteroids and MIS. For example, *in vitro* treatments with cortisol increased the sensitivity of oocytes to the MIS in rainbow trout (Jalabert and Fostier, 1984). In the view of these interactions, it has been hypothesized that the binding of corticosteroids to plasma proteins during oocyte maturation would free unbound MIS and facilitates its action (Goetz, 1983). Thus, although the MIS are today identified, these *in vitro* results support that corticosteroids might be directly and/or indirectly involved in final oocyte maturation control. But, the plasma concentrations of corticosteroids, not markedly higher than MIS levels during this period, raise the question about the extent of this physiological significance. Associated with oocyte maturation, oocyte hydration is a biological process shown to be induced *in vitro* by cortisol in rainbow trout (Milla et al., 2006). Injections of high doses of cortisol promoted ovarian tissue hydration associated with an increase of sodium content in the ayu (*Plecoglossus altivelis*) (Hirose et al., 1974). But, except DOC, no other corticosteroids have ever been tested. Finally, ovulation was induced by treatments with corticosteroids (cortisol, 11-deoxycortisol and DOC) in diverse fish species (Hirose et al., 1974; Goetz and Theofan, 1979; Haider and Rao, 1994; Small, 2004). So, corticosteroids may be actors of the endocrine control of the final stages of reproduction, in several fish species.

Until now, the majority of the studies were dedicated to understand the oviparous model. Still, in the ovoviviparous model guppy *Poecilia reticulata*, the cortisol level dropped during fertilization followed by a rise during gestation and then a strong decline at the periparturition period (Venkatesh et al., 1990). Associated with the cortisol effect on gestation prolongation, it is also hypothesized that cortisol is also important in maintaining gestation (Venkatesh et al., 1991).

To sum-up (Fig. 3), the current state of knowledge is in favour of harmful effects of cortisol during vitellogenesis by interference with ovarian sex-steroids signaling. However, cortisol might participate in the fish puberty stimulation by enhancing pituitary gonadotropins at the onset of oogenesis. Moreover, the main corticosteroids, including cortisol, seem to be involved in the regulation of peri-ovulatory mechanisms despite a disruption of the HPG axis in the case of high cortisol levels.

6. The role of corticosteroids in the reproduction of male fish

Similarly to females, corticosteroids influence male reproduction (Fig. 4). But, the available investigations mainly focused on cortisol involvement. Low plasma cortisol levels were observed during spermatogenesis in male fish (Pickering and Christie, 1981; Hou et al., 2001). This could be a physiological adaptation to protect the testes against the adverse effects of cortisol (Pottinger et al., 1995). Indeed, this steroid has numerous deleterious effects on male reproduction. When facing stressful situations or cortisol treatment, a delay in the testicular development was observed, marked by smaller gonads, retardation in spermatogenesis and lower sperm quality (Campbell et al., 1992; Consten et al., 2001). In common carp, the administration of RU486, a potent glucocorticoid receptor antagonist, prevented the decrease of gonad growth and the delay of the spermatogenesis time-course,

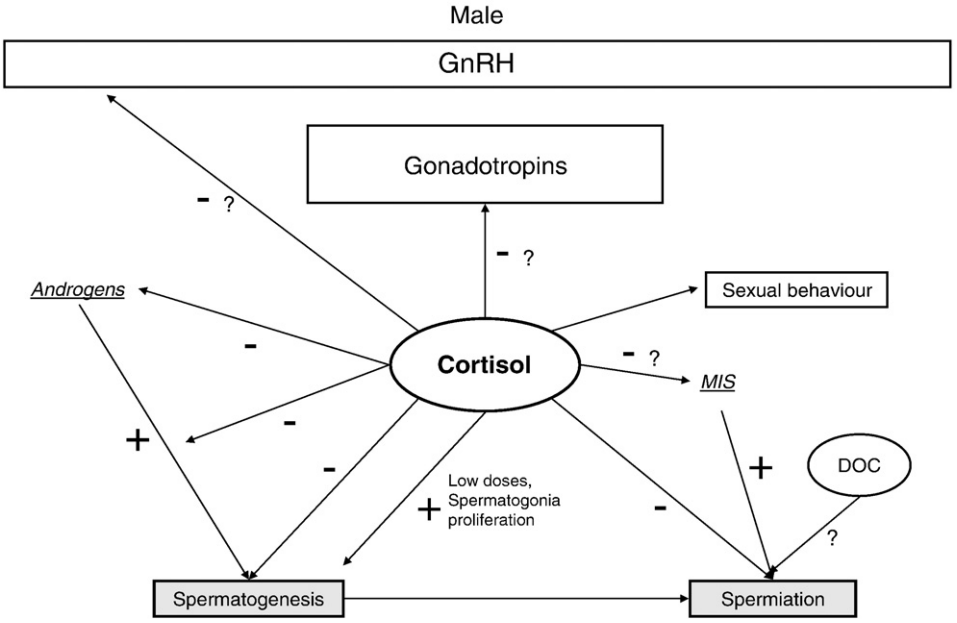

Fig. 4. Schema of the main corticosteroid implications in male teleost reproduction. MIS: Maturation-Inducing Steroid; DOC: 11-deoxycorticosterone.

showing that these cortisol impacts are managed *via* the glucocorticoid receptor pathway (Goos and Consten, 2002). Nevertheless, using a testis incubation test in the Japanese eel (*Anguilla japonica*), Ozaki et al. (2006) pointed out that cortisol directly enhances DNA replication and mitosis in spermatogonia when added at moderate doses (0.1–100 ng/mL). However, excess of cortisol (100 ng/mL) inhibited the 11-ketotestosterone effect on spermatogonia proliferation. In accordance with a positive regulatory effect of cortisol on spermatogenesis, administration of that steroid in immature knifefish *Notopterus notopterus* increased their gonado-somatic index (GSI) and activated spermatogenesis (Shankar and Kulkarni, 2000). These discrepancies between studies could be related to dose and/or species differences and underline the complexity to draw strong conclusions about the adverse cortisol effects on fish spermatogenesis.

Cortisol also inhibits the sex-steroid secretion at least during the spermatogenesis process. After stressor applications or cortisol administration during spermatogenesis, the plasma androgen levels were lower in treated fish than in controls (Pickering et al., 1987; Carragher et al., 1989; Pottinger, 1999; Goos and Consten, 2002; Consten et al., 2002; Lister et al., 2008). These observations suggest that cortisol effect on spermatogenesis retardation is partly caused by inhibition of androgen productions (Consten et al., 2002; Pickering et al., 1987; Scott and Baynes, 1982). But, we cannot preclude any inhibitory effects on the brain–pituitary–gonad axis by depressing brain GnRH content, pituitary FSH and LH and plasma gonadotropin levels as shown in common carp (Consten et al., 2001).

During the spermiation period, cortisol has also negative effects on some reproductive parameters such as testis growth for example (Carragher et al., 1989). Domesticated male striped bass *Morone saxatilis* selected as high cortisol stress responders tended to begin their spermiation earlier and exhibited a longer spermiation period (Castranova et al., 2005). In rainbow trout, a direct negative cortisol effect on the MIS testicular production was reported (Milla et al., 2008). As this latter is probably implicated in spermiation induction and/or amplification, this supports the hypothesis that cortisol affects the achievement of the last steps of male reproduction.

Even if few positive effects on spermatogenesis have been reported (see above), the great majority of the studies indicate that cortisol is an inhibitory hormone of male reproductive physiology over the whole reproductive cycle (Fig. 4). There are also some evidences of the relationship between the corticosteroid plasma levels and the mating behaviour. In *Neolamprologus pulcher*, the cortisol level was found to be higher in dominant fish compared to subordinates (Bender et al., 2006). Conversely, in *H. burtoni*, dominant territorial males which predominantly access to females exhibit lower cortisol levels associated with higher phenotypical ability to reproduction (Fox et al., 1997). Similarly, subordinate Arctic charr showed more elevated plasma concentrations of cortisol (Elofsson et al., 2000). But, it is difficult to know whether this plasma level is the result or the cause of the social position. Indeed, this plasma cortisol level may serve to provide a quick burst of energy to face with male intraspecies confrontations, either to fight or escape. Otherwise, this differential plasma corticosteroid level might reflect an ability to communicate during mating. Corticosteroids are released in the water, notably at the reproduction period (Lower et al., 2004; Ellis et al., 2005) and the sensitivity of olfactory epithelium, even weak, to sulphated corticosteroids in mature fish (cortisol and 11-deoxycortisol) (Sorensen et al., 1995) prevent us from ruling out their implication as reproductive pheromonal factors.

Interestingly, a plasma cortisol increase is also observed in parental male bluegill sunfish *Lepomis macrochirus* after hatching at the time of parental care start suggesting some cortisol physiological implications in such behaviour (Magee et al., 2006). Based on the relations between corticosteroid levels and the parental care in birds (Wingfield and Ramenofsky, 1999), it would be interesting to test this relationship in the rare fish species providing this behaviour.

7. Future directions

Several reasons may explain the complexity to assess the corticosteroid implication in fish reproduction. First, the physiological state of the animal is highly modified during mating (final stages of oogenesis, reproduction). On the one hand, as cortisol is the main stress hormone in fish, its plasma variations correlate with the occurrence of various stressful situations. Some works also suggest that corticosteroid receptors are also regulated after different stresses (Terova et al., 2007; Stolte et al., 2008). Mating period is accompanied with huge modifications in the behaviour, leading to a higher sensitivity to stress. Focusing on cortisol during reproduction must be thus undertaken with higher caution regarding the potential interaction between plasma cortisol levels and mating activities. In consequence, blood sampling should be rapidly carried out to avoid any cortisol contamination due to a stressful physiological state. Regarding the other corticosteroids, if the effects of stress on their blood plasma release remain undetermined, one should pay the same attention to them. On the other hand, reproduction often corresponds to a fasting period, or is at least accompanied with a decrease of feeding. As the plasma glucocorticoid levels are linked to the fish nutritional status, it may therefore be difficult to distinguish between the glucocorticoid fluctuations linked to reproductive physiological state and those linked to the alteration of the energetic metabolism. More generally, as suggested earlier in the salmon example, all the potential interactions between the corticotropic axis and reproduction in fish should be appropriately considered.

Second, potential relationships between corticosteroids and sex-steroids should be taken into consideration. In the classically described corticosteroid transduction pathway, corticosteroids activate corticosteroid receptors to exert their physiological actions. However, some corticosteroids are able to bind to gonadal progestogen receptors. In Arctic charr, 11-deoxycorticosterone (DOC), the putative teleost mineralocorticoid, was shown to bind to an ovarian progestogen membrane receptor. But, in that study, no other corticosteroids were tested (Berg et al., 2005). Moreover, DOC and/or to a lesser extent 11-deoxycortisol bound substantially a nuclear and membrane progestogen receptor in spotted sea trout *Cynoscion nebulosus*. But, cortisol does not seem to be an agonist of these receptors (Pinter and Thomas, 1995; Zhu et al., 2003). In male Japanese eel, DOC also displayed a high affinity for a nuclear 17α-20ß-dihydroxyprogesterone (MIS) receptor in the testis and once more, cortisol did not bind or activate it (Todo et al., 2000). That steroid binding profile was also observed in sperm membrane progestogen receptor in Atlantic croaker *Micropogonias undulatus* (Thomas et al., 2005). These results lead us to hypothesize that some corticosteroids including DOC interfere with MIS in its transduction pathway, in accordance with the reported physiological interactions between these steroids and MIS. Consequently, future studies on corticosteroid involvements should take into account potential corticosteroid effects *via* the progestogen receptor signaling. Besides, the reported effect of DOC on sperm fluidity also suggests that this steroid would play some roles in fish reproduction, either acting on the mineralocorticoid receptor or *via* progestogen receptors (Milla et al., 2008).

More generally, in the view of the described corticosteroid effects (oocyte maturation, ovulation, spermatogonia proliferation, sperm hydration), it is observed that corticosteroids either act in combination with sex-steroids or exert effects previously demonstrated to be managed by other hormones. By analogy with their roles in maintaining energetic, immune and ionic homeostasis in fish biology, we can speculate that, contrary to sex-steroids which trigger reproductive process, corticosteroids are involved in their adjustment. Their potential link with the sex-steroids should be integrated in the future experimental designs.

Third, as shown by Ozaki et al. (2006), the effects of cortisol on spermatogonia proliferation may be dose-dependent, either

stimulatory at low physiological concentrations or inhibitory at high concentrations. Thus, studies on corticosteroid effects on reproduction should be undertaken with a broad range of steroid concentrations to make sure to grasp all possibilities. In that way, more attention should be paid to 11β-hydroxysteroid dehydrogenase action, which converts cortisol into cortisone in the case of elevated levels in the plasma, as shown in mammals. The study of its activity could inform us about the need for the fish to reestablish the cortisol levels at stimulatory concentrations in the case of too high plasma concentrations. Besides, the other conditions of cortisol application (doses, timing and duration of application…) should also be accurately appreciated to better clarify the potential cortisol actions.

Finally, if the use of the mammalian model to explain the teleost physiology is often unsuitable, such comparison may allow exploring new area in fish. For several decades, the apparent absence of aldosterone and mineralocorticoid receptor in fish conferred them a corticosteroid scheme very different from mammals. Still, the discovery of mineralocorticoid receptor and its putative ligand DOC reopened the debate about the similarities between fish and mammals. If we attempt to highlight some similarities between those models, we note that the implications of corticosteroids in female reproduction (oogenesis inhibition, peri-ovulatory process stimulation, gonadotropins regulation) are similar. In male fish, whereas the adverse effects of glucocorticoids on reproduction are close to mammals, their controversial effects on spermatogonia proliferation between the fish and mammalian models may be related to dose effects. Regarding mineralocorticoids, the effects of aldosterone on sperm quality including spermatozoa concentration or androgen production may be sufficient arguments to hypothesize such roles of mineralocorticoids in teleost reproduction. More generally, the recent findings about the roles of cortisol and aldosterone during reproduction in mammals may open new perspectives regarding the endocrine control of reproduction in other vertebrates like fish.

In summary, teleost gonads are able to produce and respond to corticosteroids stimulation. Cortisol, the main corticosteroid, mainly displays direct deleterious effects on female and male gametogenesis (Figs. 3 and 4). But, the observations of these negative impacts might be related to elevated plasma concentrations. The relationship between corticosteroids and sex-steroids shows that corticosteroids also indirectly interfere with the HPG axis in both sexes (Figs. 3 and 4). By contrast, they exhibit a spectrum of direct positive activities during the final stages, particularly in females (oocyte maturation, ovulation). The changes in corticosteroid levels noted at this moment may be related to these multiple functions. Taken together, the available information strongly indicates that corticosteroid hormones may highly participate to the modulation of the reproductive endocrine control.

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References

Acosta, T.J., Tetsuka, M., Matsui, M., Shimizu, T., Berisha, B., Schams, D., Miyamoto, A., 2005. In vivo evidence that local cortisol production increases in the preovulatory follicle of the cow. J. Reprod. Dev. 51, 483–489.

Andersen, C.Y., 2002. Possible new mechanism of cortisol action in female reproductive organs: physiological implications of the free hormone hypothesis. J. Endocrinol. 173, 211–217.

Andersen, C.Y., 2003. Effect of glucocorticoids on spontaneous and follicle-stimulating hormone oocyte maturation in mouse oocytes during culture. J. Steroid Biochem. Mol. Biol. 85, 423–427.

Barry, T.P., Riebe, J.D., Parrish, J.J., Malison, J.A., 1997. Effects of 17,20β-dihydroxy-4-pregnen-3-one on cortisol production by rainbow trout interrenal tissue in vitro. Gen. Comp. Endocrinol. 107, 172–181.

Barry, T.P., Unwin, M.J., Malison, J.A., Quinn, T.P., 2001. Free and cortisol levels in semelparous and iteroparous chinook salmon. J. Fish Biol. 59, 1673–1676.

Bender, N., Heg, D., Hamilton, I.M., Bachar, Z., Taborsky, M., Oliveira, R.F., 2006. The relationship between social status, behaviour, growth and steroids in male helpers and breeders of a cooperatively breeding cichlid. Horm. Behav. 50, 73–82.

Berg, A.H., Modig, C., Olsson, P.E., 2004. 17beta-estradiol induced vitellogenesis is inhibited by cortisol at the post-transcriptional level in Arctic charr (*Salvelinus alpinus*). Reprod. Biol. Endocrinol. 2, 62.

Berg, A.H., Thomas, P., Olsson, P.E., 2005. Biochemical characterization of the Arctic charr (*Salvelinus alpinus*) ovarian progestin membrane receptor. Reprod. Biol. Endocrinol. 3, 64.

Bradford, C.S., Taylor, M.H., 1987. Semilunar changes in estradiol and cortisol coincident with gonadal maturation and spawning in the killifish *Fundulus heteroclitus*. Gen. Comp. Endocrinol. 66, 71–78.

Brann, D.W., Mahesh, V.B., 1991. Role of corticosteroids in female reproduction. FASEB J. 5, 2691–2696.

Bry, C., 1985. Plasma cortisol levels of female rainbow trout (*Salmo gairdneri*) at the end of the reproductive cycle: relationship with oocyte stages. Gen. Comp. Endocrinol. 57, 47–52.

Bury, N.R., Sturm, A., Le Rouzic, P., Lethimonier, C., Ducouret, B., Guiguen, Y., Robinson-Rechavi, M., Laudet, V., Rafestin-Oblin, M.E., Prunet, P., 2003. Evidence for two distinct functional glucocorticoid receptors in teleost fish. J. Mol. Endocrinol. 31, 141–156.

Campbell, C.M., Walsh, J.M., Idler, D.R., 1976. Steroids in the plasma of the winter flounder (*Pseudopleuronectes americanus* Walbaum). A seasonal study and investigation of steroids involvement in oocyte maturation. Gen. Comp. Endocrinol. 29, 14–20.

Campbell, C.M., Fostier, A., Jalabert, B., Truscott, B., 1980. Identification and quantification of steroids in the serum of rainbow trout during spermiation and oocyte maturation. J. Endocrinol. 85, 371–378.

Campbell, P.M., Pottinger, T.G., Sumpter, J.P., 1992. Stress reduces the quality of gametes produced by rainbow trout. Biol. Reprod. 47, 1140–1150.

Campbell, P.M., Pottinger, T.G., Sumpter, J.P., 1994. Preliminary evidence that chronic confinement stress reduces the quality of gametes produced by brown and rainbow trout. Aquaculture 120, 151–169.

Canario, A.V.M., Scott, A.P., 1990. Scott identification of, and development of radioimmunoassays for 17α,21-dihydroxy-4-pregnene-3,20-dione and 3α,17α,21-trihydroxy-5β-pregnan-20-one in the ovaries of mature plaice (*Pleuronectes platessa*). Gen. Comp. Endocrinol 78, 273–285.

Carragher, J.F., Sumpter, J.P., 1990. The effect of cortisol on the secretion of sex-steroids from cultured ovarian follicles of rainbow trout. Gen. Comp. Endocrinol. 77, 403–407.

Carragher, J.F., Sumpter, J.P., Pottinger, T.G., Pickering, A.D., 1989. The deleterious effects of cortisol implantation on reproductive function in two species of trout, *Salmo trutta* L. and *Salmo gairdneri* Richardson. Gen. Comp. Endocrinol. 76, 310–321.

Carruth, L.L., Dores, R.M., Maldonado, T.A., Norris, D.O., Ruth, T., Jones, R.E., 2000. Elevation of plasma cortisol during the spawning migration of landlocked kokanee salmon (*Oncorhynchus nerka kennerlyi*). Comp. Biochem. Physiol. C 127, 123–131.

Castranova, D.A., King, W., Woods, L.C., 2005. The effects of stress on androgen production, spermiation response and sperm quality in high and low cortisol responsive domesticated male striped bass. Aquaculture 246, 413–422.

Challis, J.R.G., Sparague, C., Patrick, J.E., 1983. Relation between diurnal changes in peripheral plasma progesterone, cortisol and estriol in normal woman at 30–31, 34–35, and 38–39 weeks of gestation. Gynecol. Obstet. Invest. 16, 33–44.

Clearwater, S.J., Pankhurst, N.W., 1997. The response to capture and confinement stress of plasma cortisol, plasma sex steroids and vitellogenic oocytes in the marine teleost, red gurnard. J. Fish Biol. 50, 429–441.

Cleary, J.J., Pankhurst, N.W., 2000. The effect of capture and handling stress on plasma steroid levels and gonadal condition in wild and farmed snapper *Pagrus auratus* (Sparidae). J. World Aquacult. Soc. 31, 558–567.

Colombe, L., Fostier, A., Bury, N.R., Pakdel, F., Guiguen, Y., 2000. A mineralocorticoid-like receptor in the rainbow trout, *Oncorhynchus mykiss*: cloning and characterization of its steroid binding domain. Steroids 65, 319–328.

Colombo, L., Bern, H.A., Pieprzk, J., Johnson, D.W., 1973. Biosynthesis of 11-deoxycorticosteroids by teleost ovaries and discussion of their possible role in oocyte maturation and ovulation. Gen. Comp. Endocrinol. 21, 168–178.

Colombo, L., Colombo Belvedere, P., Arcarese, G., 1978. Emergence of ovarian 11-deoxycorticosteroids biosynthesis at ovulation time in the sea bass, *Dicentrarchus labrax* L. Ann. Biol. Anim. Biochim. Biophys. 18, 937–941.

Consten, D., Bogerd, J., Komen, J., Lambert, J.G., Goos, H.J., 2001. Long-term cortisol treatment inhibits pubertal development in male common carp, *Cyprinus carpio* L. Biol. Reprod. 64, 1063–1071.

Consten, D., Lambert, J.G., Komen, H., Goos, H.J., 2002. Corticosteroids affect the testicular androgen production in male common carp (*Cyprinus carpio* L.). Biol. Reprod. 66, 106–111.

Cook, A.F., Stacey, N.E., Peter, R.E., 1980. Periovulatory changes in serum cortisol levels in the goldfish, *Carassius auratus*. Gen Comp Endocrinol. 40, 507–510.

Coward, K., Bromage, N.R., Little, D.C., 1998. Inhibition of spawning and associated suppression of sex steroid levels during confinement in the substrate-spawning *Tilapia zillii*. J. Fish Biol. 52, 152–165.

Crim, L.W., Peter, R.E., Billard, R., 1981. Onset of gonadotropic hormone accumulation in the immature trout pituitary gland in response to estrogen or aromatizable androgen steroid hormones. Gen. Comp. Endocrinol. 44, 374–381.

De Weerth, C., Buitelaar, J.K., 2005. Physiological stress reactivity in human pregnancy — a review. Neurosci. Biobehav. Rev. 29, 295–312.

Doyon, C., Leclair, J., Trudeau, V.L., Moon, T.W., 2006. Corticotropin-releasing factor and neuropeptide Y mRNA levels are modified by glucocorticoids in rainbow trout, *Oncorhynchus mykiss*. Gen. Comp. Endocrinol. 146, 126–135.

Dufour, S., Delerve-Le Belle, N., Fontaine, Y.A., 1983. Effects of steroid hormones on pituitary immunoreactive gonadotropin in European freshwater eel, *Anguilla anguilla* L. Gen. Comp. Endocrinol. 190–197.

Duprez, D.A., 2007. Aldosterone and the vasculature: mechanisms mediating resistant hypertension. J. Clin. Hypertens. 9, 13–18.

Ellis, T., James, J.D., Scott, A.P., 2005. Branchial release of free cortisol and melatonin by rainbow trout. J. Fish Biol. 67, 535–540.

Elofsson, U.O., Mayer, I., Damsgård, B., Winberg, S., 2000. Intermale competition in sexually mature arctic charr: effects on brain monoamines, endocrine stress responses, sex hormone levels, and behaviour. Gen. Comp. Endocrinol. 118, 450–460.

Escher, G., Mohaupt, M., 2007. Role of aldosterone available in preeclampsia. Mol. Asp. Med. 28, 245–254.

Farman, N., Rafestin-Oblin, M.E., 2001. Multiple aspects of mineralocorticoid selectivity. Am. J. Physiol. Renal Physiol. 280, F181–F192.

Fateh, M., Ben-Rafael, Z., Benadiva, C.A., Mastroianni, L., Flickinger, G.L., 1989. Cortisol levels in human follicular fluid. Fertil. Steril. 51, 538–541.

Fejes-Toth, G., Naray-Fejes-Toth, A., 2007. Early aldosterone-regulated genes in cardiomyocytes: clues to cardiac remodeling? Endocrinology 148, 1502–1510.

Filby, A.L., Tyler, C.R., 2007. Cloning and characterization of cDNAs for hormones and/or receptors of growth hormone, insulin-like growth factor-I, thyroid hormone, and corticosteroid and the gender-, tissue-,and developmental-specific expression of their mRNA transcripts in fathead minnow (*Pimephales promelas*). Gen. Comp. Endocrinol. 41, 563–565.

Fiore, C., Sticchi, D., Pellati, D., Forzan, S., Bonanni, G., Bertoldo, A., Massironi, M., Calo, L., Fassina, A., Rossi, G.P., Armanini, D., 2006. Identification of the mineralocorticoid receptor in human spermatozoa. Int. J. Mol. Med. 18, 649–652.

Foo, J.T.W., Lam, T.J., 1993. Serum cortisol response to handling stress and the effect of cortisol implantation on testosterone level in the tilapia, *Oreochromis mossambicus*. Aquaculture 115, 145–158.

Fox, H.E., White, S.A., Kao, M.H.F., Fernald, R.D., 1997. Stress and dominance in a social fish. J. Neurosci. 17, 6463–6469.

Gao, H.B., Tong, M.H., Hu, Y.Q., You, H.Y., Guo, Q.S., Ge, R.S., Hardy, M.P., 2003. Mechanisms of glucocorticoid-induced Leydig cell apoptosis. Mol. Cell. Endocrinol. 199, 153–163.

Gazola, R., Borella, M.I., Donaldson, E.M., Val-Sella, M.V., Sukumasavin, N., Fava-de-Moraes, F., Bernardino, G., 1996. Braz. J. Med. Biol. Res. 29, 659–664 title missing.

Ge, R.S., Dong, Q., Niu, E.M., Sottas, C.M., Hardy, D.O., Catterall, J.F., Latif, S.A., Morris, D.J., Hardy, M.P., 2005a. 11β-hydroxysteroid dehydrogenase 2 in rat Leydig cells: its role in blunting glucocorticoid action at physiological levels of substrate. Endocrinology 146, 2657–2664.

Ge, R.S., Dong, Q., Sottas, C., Latif, S.A., Morris, D.J., Hardy, M.P., 2005b. Stimulation of testosterone production in Leydig cells by aldosterone is mineralocorticoid receptor mediated. Mol. Cell. Endocrinol. 243, 35–42.

Gekle, M., Mildenberger, S., Freudinger, R., Grossmann, C., 2007. Altered collagen homeostasis in human aortic smooth muscle cells (HAoSMCs) induced by aldosterone. Pflugers Arch. 454, 403–413.

Gilmour, K.M., 2005. Mineralocorticoid receptors and hormones: fishing for answers. Endocrinology 146, 44–46.

Goetz, F.W., 1983. Hormonal control of oocyte final maturation and ovulation in fishes. Fish Physiol. 9B, 117–170.

Goetz, F.W., Theofan, G., 1979. In vitro stimulation of germinal vesicle breakdown and ovulation of yellow perch (*Perca flavescens*) oocytes. Effects of 17α-hydroxy-20-dihydroprogesterone and prostaglandins. Gen. Comp. Endocrinol. 37, 273–285.

Goos, H.J.T., Consten, D., 2002. Stress adaptation and pubertal development in the male common carp, *Cyprinus carpio*. Mol. Cell. Endocrinol. 197, 105–116.

Greenwood, A.K., Butler, P.C., White, R.B., DeMarco, U., Pearce, D., Fernald, R.D., 2003. Multiple corticosteroid receptors in a teleost fish: distinct sequences, expression patterns, and transcriptional activities. Endocrinology 144, 4226–4236.

Haddy, J.A., Pankhurst, N.W., 1999. Stress-induced changes in concentrations of plasma sex steroids in black bream. J. Fish Biol. 55, 1304–1316.

Haider, S., Rao, N.V., 1994. Induced spawning of maturing Indian catfish, *Clarias batrachus* (L.), using low doses of steroid hormones and salmon gonadotropin. Aquacult. Res. 25, 401–408.

Hane, S., Robertson, H., 1959. Changes in plasma 17-hydroxycorticosteroids accompanying sexual maturation and spawning of the pacific salmon (*Oncorhynchus tschawytscha*) and rainbow trout (*Salmo gairdnerii*). Proc. Natl. Acad. Sci. U. S. A. 45, 886–893.

Harlow, C.R., Jenkins, J.M., Winston, R.M.L., 1997. Increased follicular fluid total and free cortisol levels during the luteinizing hormone surge. Fertil. Steril. 68, 48–53.

Hillier, S.G., 2001. Gonadotropic control of ovarian follicular growth and development. Mol. Cell. Endocrinol. 179, 39–46.

Hirose, K., Hirano, T., Ishida, R., 1974. Effects of salmon gonadotropin on ovulation in the ayu, *Plecoglossus altivelis*, with special reference to water balance. Comp. Biochem. Physiol. A 47, 283–289.

Hou, Y.Y., Han, X.D., Yuzuri, S., 2001. Annual changes in plasma levels of cortisol and sex steroid hormones in male rainbow trout, *Oncorhynchus mykiss*. Chin. J. Ocean. Limnol. 19, 217–221.

Huang, Y.S., Rousseau, K., Sbaihi, M., Le Belle, N., Schmitz, M., Dufour, S., 1999. Cortisol selectively stimulates pituitary gonadotropin beta-subunit in a primitive teleost, *Anguilla anguilla*. Endocrinology 140, 1228–1235.

Idler, D.R., Freeman, H.C., Truscott, B., 1964. Steroid hormones in the plasma of spawned Atlantic salmon (*Salmo salar*) and a comparison of their determination by chemical and biological assay methods. Can. J. Biochem. 42, 211–218.

Jalabert, B., Fostier, A., 1984. The follicular sensitivity in vitro to maturation-inducing hormones in rainbow trout *Salmo gairdneri*: role of estradiol-17-beta. Aquaculture 42, 1–11.

Jimena, P., Castilla, J.A., Peran, F., Ramirez, J.P., Vergara, F., Molina, R., Vergara, F., Herruzo, A., 1992. Adrenal hormones in human follicular fluid. Acta Endocrinol. 127, 403–406.

Katz, Y., Eckstein, B., 1974. Changes in steroid concentration in blood of female *Tilapia aurea* (teleostei, Cichlidae) during initiation of spawning. Endocrinology 95, 963–967.

Kazeto, Y., Ijiri, S., Matsubara, H., Adachi, S., Yamauchi, K., 2003. Molecular cloning and characterization of 3β-hydroxysteroid dehydrogenase/Δ5–Δ4 isomerase cDNAs from Japanese eel ovary. J. Steroid Biochem. Mol. Biol. 85, 49–56.

Kiilerich, P., Kristiansen, K., Madsen, S.S., 2007. Cortisol regulation of ion transporter mRNA in Atlantic salmon gill and the effect of salinity on the signaling pathway. J. Endocrinol. 194, 417–427.

Kime, D.E., Dolben, I.P., 1985. Hormonal changes during induces ovulation of the carp *Cyprinus carpio*. Gen. Comp. Endocrinol. 58, 137–149.

Kime, D.E., Scott, A.P., Canario, A.V., 1992. In vitro biosynthesis of steroids, including 11-deoxycortisol and 5 alpha-pregnane-3 beta,7 alpha,17,20 beta-tetrol, by ovaries of the goldfish *Carassius auratus* during the stage of oocyte final maturation. Gen. Comp. Endocrinol. 87, 375–384.

Kobayashi, T., Nakamura, M., Kajiura-Kobayashi, H., Young, G., Nagahama, Y., 1998. Immunolocalization of steroidogenic enzymes (P450scc, P450c17, P450arom, and 3b-HSD) in immature and mature testes of rainbow trout (*Oncorhynchus mykiss*). Cell Tissue Res. 292, 573–577.

Koldkjær, P., Pottinger, T.G., Perry, S.F., Cossins, A.R., 2004. Seasonality of the red blood cell stress response in rainbow trout (*Oncorhynchus mykiss*). J. Exp. Biol. 207, 357–367.

Kubokawa, K., Watanabe, T., Yoshioka, M., Iwata, M., 1999. Effects of acute stress on plasma cortisol, sex steroid hormone and glucose levels in male and female sockeye salmon during the breeding season. Aquaculture 172, 335–349.

Kusakabe, M., Kobayashi, T., Todo, T., Mark Lokman, P., Nagahama, Y., Young, G., 2002. Molecular cloning and expression during spermatogenesis of a cDNA encoding testicular 11beta-hydroxylase (P45011beta) in rainbow trout (*Oncorhynchus mykiss*). Mol. Reprod. Dev. 62, 456–469.

Kusakabe, M., Nakamura, I., Young, G., 2003. 11beta-hydroxysteroid dehydrogenase complementary deoxyribonucleic acid in rainbow trout: cloning, sites of expression, and seasonal changes in gonads. Endocrinology 144, 2534–2545.

Lethimonier, C., Flouriot, G., Valotaire, Y., Kah, O., Ducouret, B., 2000. Transcriptional interference between glucocorticoid receptor and estradiol receptor mediates the inhibitory effect of cortisol on fish vitellogenesis. Biol. Reprod. 62, 1763–1771.

Lewicka, S., Von Hagens, C., Hettinger, U., Grunwald, K., Vecsei, P., Runnebaum, B., Rabe, T., 2003. Cortisol and cortisone in human follicular fluid and serum and the outcome of IVF treatment. Hum. Reprod. 18, 1613–1617.

Li, Y.Y., Inoue, K., Takei, Y., 2003. Interrenal steroid 21-hydroxylase in eels: primary structure, progesterone-specific activity and enhanced expression by ACTH. J. Mol. Endocrinol. 31, 327–340.

Lister, A., Nero, V., Farwell, A., Dixon, D.G., Van Der Kraak, G., 2008. Reproductive and stress hormone levels in goldfish (*Carassius auratus*) exposed to oil and sands process-affected water. Aquat. Toxicol. 87, 170–177.

Lower, N., Scott, A.P., Moore, A., 2004. Release of sex steroids into the water by roach. J. Fish Biol. 64, 16–33.

Lu, W., Worthington, J., Riccardi, D., Balment, R.J., McCrohan, C.R., 2007. Seasonal changes in peptide, receptor and ion channel mRNA expression in the caudal neurosecretory system of the European flounder (*Platichthys flesus*). Gen. Comp. Endocrinol. 153, 262–272.

Magee, S.E., Neff, B.D., Knapp, R., 2006. Plasma levels of androgens and cortisol in relation to breeding behavior in parental male bluegill sunfish, *Lepomis macrochirus*. Horm. Behav. 49, 598–609.

Mc Cormick, S.D., 2001. Endocrine control of osmoregulation in fish. Am. Zool. 41, 781–794.

McQuillan, H.J., Lokman, P.M., Young, G., 2003. Effects of sex steroids, sex, and sexual maturity on cortisol production: an *in vitro* comparison of chinook salmon and rainbow trout interrenals. Gen. Comp. Endocrinol. 133, 154–163.

Michael, A.E., 2003. Life after liquorice: the link between cortisol and conception. Reprod. Biomed. Online 7, 683–690.

Michael, A.E., Papageorghiou, A.T., 2008. Potential significance of physiological and pharmacological glucocorticoids in early pregnancy. Hum. Reprod. Update 14, 497–517.

Michael, A.E., Pester, L.A., Curtis, P., Shaw, R.W., Ewards, C.R., Cooke, B.A., 1993. Direct inhibition of ovarian steroidogenesis by cortisol and the modulatory role of 11β-hydroxysteroid dehydrogenase. Clin. Endocrinol. 38, 641–644.

Milla, S., Jalabert, B., Rime, H., Prunet, P., Bobe, J., 2006. Hydration of rainbow trout oocyte during meiotic maturation and in vitro regulation by 17,20{beta}-dihydroxy-4-pregnen-3-one and cortisol. J. Exp. Biol. 209, 1147–1156.

Milla, S., Terrien, X., Sturm, A., Ibrahim, F., Giton, F., Fiet, J., Prunet, P., Le Gac, F., 2008. Plasma 11-deoxycorticosterone (DOC) and mineralocorticoid receptor testicular expression during rainbow trout *Oncorhynchus mykiss* spermiation: implication with 17alpha, 20beta-dihydroxyprogesterone on the milt fluidity? Reprod. Biol. Endocrinol. 6, 19.

Mishra, A., Joy, K.P., 2006. Effects of gonadotrophin in vivo and 2-hydroxyoestradiol-17 in vitro on follicular steroid hormone profile associated with oocyte maturation in the catfish *Heteropneustes fossilis*. J. Endocrinol. 189, 341–353.

Mommsen, T.P., Vijayan, M.M., Moon, T.W., 1999. Cortisol in teleosts: dynamics, mechanisms of action, and metabolic regulation. Rev. Fish Biol. Fish 9, 211–268.

Nacharaju, V.L., Muneyyirci, O., Kahn, N., 1997. Presence of 11 beta-hydroxysteroid dehydrogenase in human semen: evidence of correlation with semen characteristics. Steroids 62, 311–314.

Nagahama, Y., Yoshikuni, M., Yamashita, M., Tanaka, M., 1994. Regulation of oocyte maturation of fish. In: Hoar, W.S., Randall, D.J., Donaldson, E.M. (Eds.), Fish Physiology, vol. 13. Academic Press, New York, pp. 393–439.
Noaksson, E., Linderoth, M., Gustavsson, B., Zebühr, Y., Balk, L., 2005. Reproductive status in female perch (*Perca fluviatilis*) outside a sewage treatment plant processing leachate from a refuse dump. Sci. Total Environ. 340, 97–112.
Okumura, S., Okamoto, K., Oomori, R., Nakazono, A., 2002. Spawning behavior and artificial fertilization in captive reared red spotted grouper, *Epinephelus akaara*. Aquaculture 206, 165–173.
Ozaki, Y., Higuchi, M., Miura, C., Yamaguchi, S., Tozawa, Y., Miura, T., 2006. Roles of 11ß-hydroxysteroid dehydrogenase in fish spermatogenesis. Endocrinology 147, 5139–5146.
Pankhurst, N.W., Conroy, A.M., 1988. Endocrine changes during gonadal maturation and spawning in the Orange roughy (*Hoplostethus atlanticus* Collett), a teleost from the midslope waters off New Zealand. Gen. Comp. Endocrinol. 70, 262–273.
Pankhurst, N.W., Van Der Kraak, G., 2000. Evidence that acute stress inhibits ovarian steroidogenesis in rainbow trout in vivo, through the action of cortisol. Gen. Comp. Endocrinol. 117, 225–237.
Park, C.B., Takemura, A., Aluru, N., Park, Y.J., Kim, B.H., Lee, C.H., Lee, Y.D., Moon, T.W., Vijayan, M.M., 2007. Tissue-specific suppression of estrogen, androgen and glucocorticoid receptor gene expression in feral vitellogenic male Mozambique tilapia. Chemosphere 69, 32–40.
Pickering, A.D., Christie, P., 1981. Changes in the concentrations of plasma cortisol and thyroxine during sexual maturation of the hatchery-reared brown trout, *Salmo trutta*. Gen. Comp. Endocrinol. 44, 488–496.
Pickering, A.D., Pottinger, T.G., Carragher, J., Sumpter, J.P., 1987. The effects of acute and chronic stress on the levels of reproductive hormones in the plasma of mature male brown trout, *Salmo trutta* L. Gen. Comp. Endocrinol. 68, 249–259.
Pinter, J., Thomas, P., 1995. Characterization of a progestogen receptor in the ovary of the spotted sea-trout, *Cynoscion nebulosus*. Biol. Reprod. 52, 667–675.
Pottinger, T.G., 1999. The impact of stress on animal reproductive activities. In: Balm, P.H.M. (Ed.), Stress Physiology in Animals. Sheffield Academic Press, Sheffield, pp. 130–177.
Pottinger, T.G., Pickering, A.D., 1990. The effect of cortisol administration on hepatic and plasma estradiol-binding capacity in immature female rainbow trout (*Oncorhynchus mykiss*). Gen. Comp. Endocrinol. 80, 264–273.
Pottinger, T.G., Balm, P.H.M., Pickering, A.D., 1995. Sexual maturity modifies the responsiveness of the pituitary–interrenal axis to stress in male rainbow trout. Gen. Comp. Endocrinol. 98, 311–320.
Pottinger, T.G., Carrick, T.R., Hughes, S.E., Balm, P.H., 1996. Testosterone, 11-ketotestosterone, and estradiol-17 beta modify baseline and stress-induced interrenal and corticotropic activity in trout. Gen. Comp. Endocrinol. 104, 284–295.
Prunet, P., Sturm, A., Milla, S., 2006. Multiple corticosteroid receptors in fish: from old ideas to new concepts. Gen. Comp. Endocrinol. 147, 17–23.
Rahman, M.A., Ohta, K., Chuda, H., Nakano, S., Maruyama, K., Matsuyama, M., 2001. Gonadotropin-induced steroidogenic shift towards maturation-inducing hormone in Japanese yellowtail during final oocyte maturation. J. Fish Biol. 58, 462–474.
Redding, J.M., Patino, R., 1993. Reproductive physiology. In: Evans, D.H. (Ed.), Physiology of Fishes. CRC Press, Boca Raton, pp. 503–534.
Reddy, P.K., Renaud, R., Leatherland, J.F., 1999. Effects of cortisol and triiodo-L-thyronine on the steroidogenic capacity of rainbow trout ovarian follicles at two stages of oocyte maturation. Fish Physiol. Biochem. 21, 129–140.
Robertson, O.H., Wexler, B.C., 1959. Hyperplasia of the adrenal and cortical tissue in Pacific salmon (genus *Oncorhynchus*) and rainbow trout (*Salmo gairdneri*) accompanying sexual maturation and spawning. Endocrinology 65, 225–238.
Rocha, M.J., Reis-Henriques, M.A., 1999. Plasma levels of C-18-, C-19- and C-21-steroids in captive and feral female sea bass. J. Fish Biol. 55, 25–34.
Roy, R.L., Ruby, S.M., Idler, D.R., Ying, S., 1990. Plasma vitellogenin levels in pre-spawning rainbow trout, *Oncorhynchus mykiss*, during acid exposure. Arch. Environ. Contam. Toxicol. 19, 803–806.
Sarkar, P., Bergman, K., Fisk, N.M., O, 'Connor, T.G., Glover, V., 2007. Ontogeny of foetal exposure to maternal cortisol midtrimester amniotic fluid as a biomarker. Clin. Endocrinol. 66, 636–640.
Schreck, C.B., Contreras-Sanchez, W., Fitzpatrick, M.S., 2001. Effects of stress on fish reproduction, gamete quality, and progeny. Aquaculture 197, 3–24.
Scott, A.P., Baynes, S.M., 1982. Plasma levels of sex steroids in relation to ovulation and spermiation in rainbow trout (*Salmo gairdneri*). In: Richter, C.J.J., Goos, H.J. (Eds.), Proceedings of the 2nd International Symposium on Reproductive Physiology of Fish: 2–6 august 1982. Pudoc, Wageningen, pp. 103–106.
Scott, A.P., Canario, A.V., 1990. Plasma levels of ovarian steroids, including 17 alpha,21-dihydroxy-4-pregnene-3,20-dione and 3 alpha,17 alpha,21-trihydroxy-5 beta-pregnan-20-one, in female plaice (*Pleuronectes platessa*) induced to mature with human chorionic gonadotrophin. Gen. Comp. Endocrinol. 78, 286–298.
Scott, A.P., Mackenzie, D.S., Stacey, N.E., 1984. Endocrine changes during natural spawning in the white sucker, *Catostomus commersoni*. Gen. Comp. Endocrinol. 56, 349–359.
Scott, A.P., Sherwood, N.M., Canario, A.V.M., Warby, C.M., 1991a. Identification of free and conjugated steroids, including cortisol and 17α,20β-dihydroxy-4-pregnen-3-one, in the milt of Pacific herring, *Clupea harengus pallasi*. Can. J. Zool. 69, 104–110.
Scott, A.P., Canario, A.V.M., Sherwood, N.M., Warby, C.M., 1991b. Levels of steroids, including cortisol and 17α,20β-dihydroxy-3-one, in plasma, seminal fluid, and urine of pacific herring (*Clupea harengus pallasi*) and north sea plaice (*Pleuronectes platessa* L.). Can. J. Zool. 69, 111–116.
Seckl, J.R., Chapman, K.E., 1997. The 11β-hydroxysteroid dehydrogenase system, a determinant of glucocorticoid and mineralocorticoid action. Eur. J. Biochem. 249, 361–364.
Shankar, D.S., Kulkarni, R.S., 2000. Effect of cortisol on testis of freshwater fish *Notopterus notopterus* (Pallas). Indian J. Exp. Biol. 38, 1227–1230.
Small, B.C., 2004. Effect of dietary cortisol administration on growth and reproductive success of channel catfish. J. Fish Biol. 64, 589–596.
Socorro, S., Martins, R.S., Delovre, L., Mylonas, C.C., Canario, A.V.M., 2007. A cDNA for European sea bass (*Dicentrachus labrax*) 11βhydroxylase: gene expression during the thermosensitive period and gonadogenesis. Gen. Comp. Endocrinol. 150, 164–173.
Sorensen, P.W., Scott, A.P., Stacey, N.E., Bowdin, L., 1995. Sulfated 17,20 beta-dihydroxy-4-pregnen-3-one functions as a potent and specific olfactory stimulant with pheromonal actions in the goldfish. Gen. Comp. Endocrinol. 100, 128–142.
Stolte, E.H., De Mazon, A., Leon, K., Jesiak, M., Bury, N.R., Sturm, A., Savelkoul, H., Van Kemenade, L., Flik, G., 2008. Corticosteroid receptors involved in stress regulation in common carp, *Cyprinus carpio*. J. Endocrinol. 198, 403–417.
Struthers, A.D., 1995. Mineralocorticoid receptor blockade in chronic heart failure. J. Hum. Hypertens. 9, 443–446.
Sturm, A., Bury, N., Dengreville, L., Fagart, J., Flouriot, G., Rafestin-Oblin, M.E., Prunet, P., 2005. 11-deoxycorticosterone is a potent agonist of the rainbow trout (*Oncorhynchus mykiss*) mineralocorticoid receptor. Endocrinology 146, 47–55.
Sundararaj, B.I., Goswami, S., 1969. Role of interrenal in luteinizing hormone-induced ovulation and spawning in the catfish, *Heteropneustes fossilis* (Bloch). Gen. Comp. Endocrinol. Suppl. 2, 374–384.
Sun, K., Adamson, S.L., Yang, K., Challis, J.R.G., 1999. Interconversion of cortisol and cortisone by 11β-hydroxysteroid dehydrogenases type 1 and 2 in the perfused human placenta. Placenta 20, 13–19.
Teitsma, C.A., Anglade, I., Lethimonier, C., Le Dréan, G., Saligaut, D., Ducouret, B., Kah, O., 1999. Glucocorticoid receptor immunoreactivity in neurons and pituitary cells implicated in reproductive functions in rainbow trout: a double immunohistochemical study. Biol. Reprod. 60, 642–650.
Terova, G., Gornati, R., Rimoldi, S., Bernardini, G., Saroglia, M., 2007. Quantification of a glucocorticoid receptor in sea bass (*Dicentrarchus labrax*, L.) reared at high stocking density. Gene 347, 144–151.
Tesone, M., Charreau, E.H., 1980. Steroid biosynthesis in the gonads of the teleost fish *Jenynsia lineata*. Comp. Biochem. Physiol. 65, 631–637.
Thomas, P., Tubbs, C., Detweiler, C., Das, S., Ford, L., Breckenridge-Miller, D., 2005. Binding characteristics, hormonal regulation and identity of the sperm membrane progestin receptor in Atlantic croaker. Steroids 70, 427–433.
Tilbrook, A.J., Turner, A.I., Clarke, I.J., 2000. Effects of stress on reproduction in non-rodent mammals: the role of glucocorticoids and sex differences. Rev. Reprod. 5, 105–113.
Todo, T., Ikeuchi, T., Kobayashi, T., Kajiura-Kobayashi, H., Suzuki, K., Yoshikuni, M., Yamauchi, K., Nagahama, Y., 2000. Characterization of a testicular 17α,20β-dihydroxy-4-pregnen-3-one (a spermiation-inducing steroid in fish) receptor from a teleost, Japanese eel (*Anguilla japonica*). FEBS Letts. 465, 12–17.
Unal, G., Karakisi, H., Mahmut, E.L.P., 2006. Levels of some ovarian hormones in the pre- and post spawning periods of *Chalcalburnus tarichi* Pallas, 1811, and the postovulatory structure of follicles. Turk. J. Vet. Anim. Sci. 30, 427–434.
Unal, G., Erdogan, E., Oguz, A.R., Kaptaner, B., Kankaya, E., Elp, M., 2008. Determination of hormones inducing oocyte maturation in *Chalcalburnus tarichi* (Pallas, 1811). Fish Physiol. Biochem. 34, 447–454.
Venkatesh, B., Tan, C.H., Lam, T.J., 1990. Steroid hormone profile during gestation and parturition of the guppy (*Poecilia reticulata*). Gen. Comp. Endocrinol. 77, 476–483.
Venkatesh, B., Tan, C.H., Lam, T.J., 1991. Progestins and cortisol delay while estradiol-17 beta induces early parturition in the guppy, *Poecilia reticulata*. Gen. Comp. Endocrinol. 83, 297–305.
Wagner, A., Claus, R., 2004. Involvement of glucocorticoids in testicular involution after immunization of boars against GnRH. Reproduction 127, 275–283.
Weber, M.A., Groos, S., Hopfl, U., Spielmann, M., Aumüller, G., Konrad, L., 2000. Glucocorticoid receptor distribution in rat testis during postnatal development and effects of dexamethasone on immature peritubular cells in vitro. Andrologia 32, 23–30.
Wendelaar Bonga, 1997. The stress response in fish. Physiol. Revs. 77, 591–625.
Westring, C.G., Ando, H., Kitahashi, T., Bhandari, R.K., Ueda, H., Urano, A., Dores, R.M., Sher, A.A., Danielson, P.B., 2008. Seasonal changes in CRF-I and urotensin I transcript levels in masu salmon: correlation with cortisol secretion during spawning. Gen. Comp. Endocrinol. 155, 126–140.
Wingfield, J.C., Grimm, A.S., 1977. Seasonal changes in plasma cortisol, testosterone and oestradiol-17β in the plaice, *Pleuronectes platessa* L. Gen. Comp. Endocrinol. 31, 1–11.
Wingfield, J.C., Ramenofsky, M., 1999. Hormones and the behavioral ecology of stress. In: Balm, P.H.M. (Ed.), Stress Physiology in Animals. Sheffield Academic Press, pp. 1–51.
Woodhead, A.D., 1975. Endocrine physiology of fish migration. Ocean. Mar. Ann. Rev. 13, 287–382.
Yang, J.G., Chen, W.Y., Li, P.S., 1999. Effects of glucocorticoids on maturation of pig oocytes and their subsequent fertilizing capacity in vitro. Biol. Reprod. 60, 929–936.
Zhou, L.Y., Wang, D.S., Shibata, Y., Bindhu, P.P., Suzukia, A., Nagahama, Y., 2007. Characterization, expression and transcriptional regulation of P450c17-I and -II in the medaka, *Oryzias latipes*. Biochem. Biophys. Res. Commun. 362, 619–625.
Zhu, Y., Rice, C.D., Pang, Y., Pace, M., Thomas, P., 2003. Cloning, expression, and characterization of a membrane progestin receptor and evidence it is an intermediary in meiotic maturation of fish oocytes. Proc. Natl. Acad. Sci. U. S. A. 100, 2237–2242.