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Review

The mechanism of reproduction and hormonal function in finfish species: A review

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The number of aquatic species currently under domestication efforts is rising rapidly, due to the development of commercial aquaculture. For domestication and the establishment of a sustainable aquaculture industry, the control reproduction processes of fish in captivity, and to acquire high quality seed, is necessary. The fish reproductive cycle is separated in gametogenesis (of growth) and oocyte maturation and spermiation (of maturation). These are controled by the reproductive hormones of the brain, pituitary and gonad. Environmental and endogenous physiological information reach neuroendocrine system that regulates pituitary and gonadal functions. The most commonly reproduction dysfunctions in cultured fish are the unpredictability of final oocyte maturation (FOM) in females, and the lack and less quality of sperm in males. These problems are due to the fact that the fish in captivity does not have the same conditions in the spawning grounds, and as a result, there is a failure of the proper hormonal regulation. Failure of the pituitary to release gonadotropin (GTH-II), one of the hormones involved in the regulation of reproduction, is the result of unsucssesful ovulation. Actually two type of GTH are identified. They are differing from structural and chemical roles. GTH-I also name as FSH, involve in vitellogenesis or spermatogenesis (initial stage of gametogenesis), and GTH-II name as LH are involve in FOM and spermiogenesis and spermiation.The control role and function of the hormone involve in reproduction are described and reviewed in the present paper.

Key word: GTH-I, GTH-II, GNRH, teleost, gametogenesis.

INTRODUCTION

Aquaculture has been practiced in extensive forms, more than several centuries, but approaching the intensive form has been done only in the last few decades. During this period, the number of aquatic species is rising under domestication efforts. A sustainable aquaculture industry is the capacity to control reproduction processes of fish in capacity and to acquire high quality seed in future, aquaculture production and development is based on the control of aquaculture reproduction. The rearing of many finfish species still rely on the capture of eggs or juveniles in the wild. Many brakish water fish such as species in cyprinid family are readily reproduce when migrated to river. The South Caspian Kutum (*Rutilus frisii kutum*) as example that spawn very easily just by a gentle force at the belly that release the milt and eggs at spawning time (Yousefian and Mosavi, 2008). But collecting the eggs or juveniles in spawning season is unreliable. Shafiei sabet et al. (2010) mentioned close interaction between environmental cues and endocrine control of reproduction. They reported that endocrine control cannot continue without the appropriate environmental cues required to stimulate reproduction.To have an industrialization of aquaculture, reproduction should be controlled and a steady supply of seed be produced all around the year. Although a series of culture fishes today are reproduce under control in captivity but there are some new cultured fish that depend on the collection of juveniles or adults from the wild. The management of the technologies for gamete production in captivity is one of the essential step for aquaculture that would ensure the growth to this sector (Bromage, 1992).

Unfortunately, most fish when reared in captivity condition, exhibit some degree of reproduction dysfunction. Many species of captive fish are able to reach reproduction

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maturity in aquaculture conditions and gonadal growth occurs normally (Mylonas and Zohar, 2001). However, some of female species often fail final oocyte maturation stage (FOM) and do not spawn (Zohar, 1989; Peter et al., 1993), while males exhibit diminished production or low quality of milt (Billard, 1986, 1989; Mylonas and Zohar, 2001).

For management of reproduction in captive fish, we may control or provoke the reproduction. These are approached by environmental or hormonal stimulation. The possibility of collecting and storage of milt without further manipulations and seasonal depending is very useful for the spread of induced spawning in aquaculture (Mylonas and Zohar, 2001).

Environmental manipulation to induce ovulation and spawning in fish has been reviewed by Lam (1983) and Lam and Munro (1987). The lack of natural environmental stimuli, and the unavoidable stress by captivity, often causes the lack of the final oocyte maturation stage (FOM) and of ovulation in females, inhibiting also their natural spawning. In males, instead, a decrease of the seminal liquid volume and a worsening of its quality can be observed (Rainis et al., 2003). After isolation of hypothalamus LH-releasing factors by (Amoss et al., 1971; Matsuo et al., 1971), this decapeptide named LHRH, its primary structure was identified. This decapeptide stimulates FSH release and therefored rename GnRH (Zohar et al., 2010).

In some species hormonal treatments are the only means of controlling reproduction reliably. Over the years, a variety of hormonal approaches have been used successfully (Peter et al., 1993; Zohar, 1989; Tucker, 1994; Podhorec and Kouril, 2009).

The use of hormones, with regard to the possibilities of hormonal therapy of reproduction, has been referred to by Burgas et al. (1971), and they discovered the primary structure of mammalian GnRH neurode capeptide.

The possibility of direct stimulation of gonadotropin cells secreting the fish's own luteinizing hormone (Lam et al., 1975) was added to a previously used type of hormonal therapy, which replaced the insufficient production of endogenous luteinizing hormone (von Ihering, 1937).

Along with the identification of LH inhibition factor (Peter et al., 1986) dopamine and use of DA antagonists, effective stimulation methods of LH secretion, the so-called hypothalamic approach (Peter et al., 1988), were developed which can be applied to a wide range of fish species (Podhorec and Kouril, 2009).

Traditional methods of induced spawning for cultured fish are based on the injection of GTH-II from different sources, including crude extract of carp pituitary gland (CPE), partially purified fish GTH-II and mammalian GTH, especially human chorionic hormone (Lam, 1982; Donaldson and Hunter, 1983; Peter et al., 1988). However, the use of this technique has number problems. GtH-II and HCG is highly species specific that are effective in some species while ineffective in others (Zohar et al., 1987; Lin et al., 1986). On the other hand when crude pituitary extract are used to induce spawning, the fish is treated with a mixture of hormones which may have side effects on gametogenesis or other functions (Lin and Peter, 1996).

The use of hormones should be at a proper time other wise it will have a worse effect. Actually after successfully completing vitellogenesis fish are not capable of undergoing the next steps of gametogenesis and subsequent ovulation (Mylonas and Zohar, 2007). There is a need for maturation induction technologies. Sustained-release delivery systems for gonadotropin- releasing hormone (GnRH) agonists have been increasingly employed in various culture situations during the past two decades, in order to control the reproduction of commercially important finfish (Mylonas and Zohar, 2001). The rule and function of different hormones and passway in the gonad and body of fish have been investigated by many authors (Harvey and Hoar, 1979; Lam, 1982, 1985; Donaldson and Hunter, 1983; Crim et al., 1987).

Ovulation and spawning in teleosts as in other vertebrates are controlled by several interacting factors, environmental stimuli are translated by the brain into neural signals which result in release of gonadotropin releasing hormone (GnRH) and /or inhibition of release of gonadotropin release inhibiting factor (GnRIF) causing the pituitary to secrete gonadotropins (GTHs) (Peter et al., 1986; Lin and Peter, 1996). When a certain GTH level in reached, vitellogenic oocytes undergo the process of final oocyte maturation, the germinal vesicle migrates to the periphery, theca and granulosa cells of the follicle are stimulated to secrete a maturation-inducing steroid (MIS), and the MIS induces germinal vesicle breakdown (GVBD) (Nagahama, 1983; Fostier and Jalabert, 1982; Goetz, 1983).

Therefore, the use of hormones have their own various problems and the fish have to be at proper time of treatment, advanced stage of gonad development, on the other hand for most of fish two or more hormone is necessary for successful response.

THE REPRODUCTIVE CYCLES IN FISH

The endocrine control system of the reproduction in finfish is based on the hypothalamus-hypophysis (pituitary gland) - gonads axis, similarly to mammals. hypothalamus produces the gonadotropin-releasing factor (GnRH) which acts on the pituitary gland. This gland controls synthesis and release of the gonadotropic hormones (GtHs), whose role is to lead the gonads (ovaries and testicles) to produce the gametes. The pituitary gland produces also dopamine, which, on the contrary, has an inhibiting effect on the process (Rainis et al., 2003) (Figure 1).

In all vertebrates, the pituitary is attached to the hypothalamus by a short stalk that, fish, consists of neurosecretory fibers passing from the brain to the pituitary. These are in fact axons from neurons located in

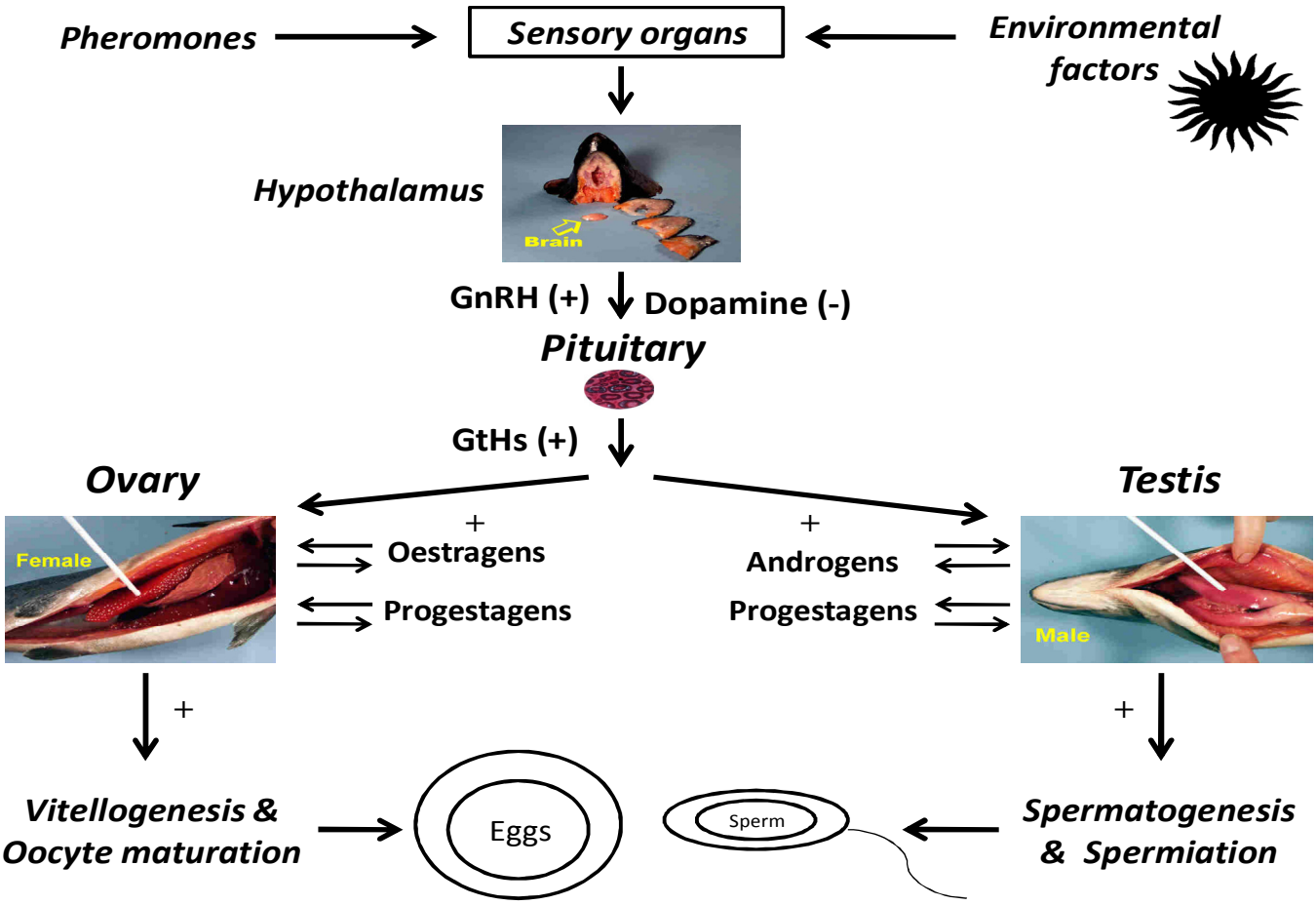

Figure1. Schematic representation of the reproductive axis in fish, its major components and phases, and its environmental and endocrine control.

the hyphothalamus and sending projections to the pituitary. The pituitary gland, or hypophysis, consists of the adenohypophysis and the neurohypophysis (Zohar et al., 2010). The production of these hormones is based on the environmental stimulation that will be discussed. Actually two type of GTH are identified. They differ in structural and chemical role. GTH-I also name as FSH, is involve in vitellogenesis or spermatogenesis (initial stage of gametogenesis), and GTH-II name as LH are involve in FOM and spermiogenesis and spermiation.

During the process of gametogenesis and based on the stage of egg development, one of these hormones is secreted. The process in male and female are well described by Rainis et al. (2003). GtH-II increase in plasma just before spermiation and FOM, determining the switch from the steroidogenic and androgenic production (Testosterone, T and 11-Keto-Testosterone, 11-KT, in males and Oestradiol-17β and Oestradiol, in females) to the progestinic production (like 17α-20β-dihydroxy-4-pregnen-3-one, 17α20βP and 17-hydroxyprogesterone) respectively in the interstitial testicular cells and the ovarian theca cells. The progestagens cause the FOM in females, which regulate the spermiation in males (stimulating the production of seminal fluid, the spermatozoa maturation) and influence the sexual behavior (Pankhurst and Thomas, 1998). Most of the milt hydration process is GtH-dependent, because these hormones stimulate spermiation and increase the total volume of spawned milt (Pankhurst, 1994; Rainis et al., 2003).

The production of reproduction hormones is studied from 1964 by Oliveau and Ball and they were found in verteberate. In all vertebrates including fish, the anterior lobe contains the corticotrophs (ACTH cells), the mammotrophs (prolactin cells, PRL cells), the somatotrophs (growth hormone cells, GH cells), the thyrotrophs (THS cells) and the gonadotrophs (LH/FSH cells). Many pioneering studies on the pituitary of fish were conducted by Madeleine Firstly two gonadotroph cell type distinguished in salmonids using conventional staining techniques (Olivereau, 1976) following the purification of FSH and LH in coho salmon, this was later confirmed

using specific antibodies to the β subunits of the gonadotropins (Dickey and Swanson, 1998; Nozaki et al., 1990; Shimizu et al., 2003; Zohar et al., 2010).

GnRH RECEPTORS IN FISH

Confounding the effects of GnRH on gonadotropin synthesis and release in fish illustrate the presence of multiple isoforms of GnRH in teleosts (Kah et al., 2007; Lethimonier et al., 2004). Studies comparing the relative LH releasing effects of the native GnRH isoforms in teleost species, including goldfish, catfish and seabream, as well as *in vitro* experiments directly testing receptor activation demonstrated that the most potent GnRH form in terms of bioactivity is GnRH-II, while the species-specific Type I isoform is generally the least potent (Zohar et al., 2010). In agreement with the higher potency GnRH-II compared to Type I GnRH, apart from the mammalian Type I GnRH (Reinhat et al., 1992) receptor, all GnRH receptors cloned in vertebrates and in case of many species have a higher sensitivity to GnRH-II (Kah et al., 2007). The lower doses of GnRH-I that were insufficient to elicit a response alone were otherwise able to enhance the effects of stimulatory doses of GnRH-II. Coadministration of intermediate doses of both peptides generally had synergistic effects on gonadotroph function. Studies of this sort, in which physiologically relevant combinations of the different GnRH forms are tested using experimental models shows the coordinated effects of multiple GnRHs on pituitary function in fish and other vertebrates (Zohar et al., 2010).

DOPAMINE INHIBITS GONADOTROPIN RELEASE IN FISHES

Dopamine (DA) possesses a strong inhibitory action on gonadotropin secretion in some teleosts species and that it was possible to overcome this inhibitory action of DA and to potentiate the action of GnRH by the application of specific D2 dopamine receptor antagonists (Zohar and Mylonas, 2001). In fact, dopamine strongly inhibits gonadotropin release in a mechanism way that differs in different fish. Neuroendocrine regulation of GTH-II secretion in teleosts is mainly under a dual neurohormonal system. GTH-II release is stimulated by a gonadotropin-releasing hormone (GnRH) and inhibited by dopamine, which functions as gonadotropin releasing-inhibitiry factor (GRIF). Dopamine acts directly at the level of the pituitary to modulate the actions of GnRH as well as the spontaneous release of GTH-II, and inhibits release of GnRH (Peter et al., 1991). Dopamine is a small neurotransmitter that is synthesized from tyrosine through a two step reaction involving the step-limiting enzyme tyrosine hydroxylase and DOPA-decarboxylase (Zohar et al., 2010). The distribution of dopamine in the brain of fish has been extensively studied using different techniques showing the existence of a well developed dopaminergic system (Nieuwenhuys et al., 1998). Dopamine receptors belong to the G-protein coupled receptor (GPCR) family. There are two main classes of DA receptors that differ in their ability to activate (D1) or to inhibit (D2) the enzyme adenylyl cyclase, with each class containing various subtypes (Cardinaud et al., 1998; Kebabian and Calne, 1979; Zohar et al., 2010).

Dopamine, one of the catecholamine neurotransmitters, is the only known factor having an inhibitory effect on LH secretion in the family Cyprinidae. Dopamine exerts its inhibitory activity via receptors belonging to members of seven transmembrane domain GPCRs, which are separated into D1and D2 receptor classes. Secretion of dopamine from nerve terminals in the pituitary and its binding to D2 receptors localized on gonadotrophs results in inhibition of basal and GnRH-stimulated release of LH (Podhorec and Kouril, 2009). The acute direct effect of DA induces the disruption of intracellular GnRH signal transduction pathways whereas the long-term effects account for a reduction in the number of GnRH receptors on the surface of LH tropic cells and a reduction in GnRH peptide release from nerve terminals in the pituitary. The inhibitory effect of DA on LH secretion changes over the course of the reproductive cycle, with the maximum DA inhibition occurring during the final stages of gametogenesis. This feature is utilised in aquaculture of Cyprinidae by using dopamine antagonists in ovulation-inducing therapies, example, domperidon, pimozide, reserpin, metoclopramide, haloperidol, isofloxythepin (Podhorec and Kouril, 2009).

Several years ago, (Chang and Peter, 1983) demonstrated that dopamine (DA) inhibits GTH release from dispersed pituitary cells or pituitary fragments, suggesting that dopamine could be the gonadotropin inhibitory factor. Further investigation established that DA was acting directly at the pituitary cell level, thus indicating that gonadotrophs carry dopamine receptors (Chang and Peter, 1983). It was shown that dopamine acts directly onto the gonadotrophs through D2 receptors. This was shown first in goldfish and then confirmed in other species such as carp, African catfish, trout, tilapia, eel and gray mullet. The rainbow trout is the only species in which dopamine is known to inhibit both LH and FSH release acting through D2 receptors (Vacher et al., 2002, 2000; Zohar et al., 2010). Dopamine in some fishes such as goldfish (Trudea, 1997) has two different effects. One is inhibiting gonadotropin release by direct action on the gonadotropin and the second is reducing GnRH secration in the vicinity of the gonadotrophes. The presence of dopamine inhibitor of gonadotrpin in some species, absence in many others and partially in marine species illustrate that it has different physiological and adaptative maner to different species. In goldfish, dopamine prevents ovulation, although environmental conditions, such as the presence of phermon, vegetation, proper temperature and females as partners are not appropriate.

For this reason, in cyprinids, silurids and tilapia, combined treatment with GnRH agonist and dopamine D2 antagonist, such as pimozide, are extremely efficient to induce ovulation and spermiation (Peng et al., 1994; Zohar et al., 2010). Inducton of ovulation in endemic Chalcaburnus chalcoides, living in the Caspian Sea is also achived by the use of LRH-Aa combined with methoclopramide (Yousefian et al., 2008). Surprising without the use of Methoclopramide no successful results were obtained

THE STEROID FEEDBACK IN REPRODUCTION CYCLE

There is a communication between the brain/pituitary complex and the the gonads. This relation allows the activity of the different components of the brain–pituitary–gonad axis to be synchronized at all steps of the life cycle, which is crucial for coordinated responses. Of particular importance are sexual steroids, produced by the gonads, which are used by the brain and pituitary as indicators of the sexual status. Sexual steroids modulate the activity of the neuronal systems influencing the reproductive axis.They notably affect expression of neuropeptides and neurotransmitters, as well as that of their corresponding receptors in the brain and the pituitary. These mechanisms are essential components of the hormonal communication along the brain–pituitary–gonadal axis. Classically, both positive and negative feedback effects have been reported on the synthesis and release of LH in teleosts, using gonadectomy and/or steroid replacement. Negative feedback is documented in many species including salmonids, cyprinids, silurids and perciforms (Aroua et al., 2007; Trudeau, 1997). However, there is also evidence in juvenile fish for a positive feedback of sex steroids on LH content and release (Crim and Evans, 1983). The mechanisms mediating these effects are likely to be extremely complex and can be caused by direct effects of steroids at the pituitary or the hypothalamic levels. Indeed, both regions contain a high density of estrogen receptors and androgen receptors. This complexity is even increased by the fact that the brain of fish is well known for its high capacity to convert aromatizable androgens into estrogens (Zohar et al., 2010).

STIMULATION FACTOR OF LH SECRETION

In finfish species of Teleostei the neurodecapeptide GnRH is the central regulator of the reproductive hormonal cascade regulating the synthesis and release of LH secretion from the pituitary gland (Somoza et al., 2002; Yaron et al., 2003; Millar et al., 2004; Kah et al., 2007; Yousefian et al., 2009). The hypophysiotropic GnRH is processed in the hypothalamic neurons by enzymatic cleavage of a precursor polypeptide and packaged in storage granules (Yaron and Sivan, 2006).

GnRH was first isolated from the mammalian hypothalamus as mammalian GnRH with the following amino acid structure: pGlu-His-Trp-Ser- Tyr-Gly-Leu-Arg-Pro-Gly.NH2 (Burgus et al., 1971).

The first GnRH form identified in teleost fish was a salmon GnRH in chum salmon (*Oncorhynchus keta*) whose stucture was similar to that of mGnRH, differing only in amino acids at Positions 7 (Trp) and 8 (Leu), (Sherwood et al., 1983). Among vertebrates Teleostei is the group where the highest number of GnRH forms occurs (Chen and Fernald, 2008). A total of eight GnRH forms have been identified until now. Research until now using diagnostic methods like RIA, HPLC, *in situ* hybridization, etc., has confirmed the occurrence of only two GnRH formes (GnRH2, GnRH3) in the members of the family Cyprinidae, example, goldfish, roach and zebra danio. However, in some species the three GnRH forms were detected simultaneously, example, gilthead seabream (Podhorec and Kouril, 2009). Based on the classification proposed by Fernald and White (1999), the determined GnRH forms are divided into three branches. Despite great effort, the occurrence of the GnRH1 line has not been detected in the cyprinids. It seems that it is mainly GnRH3 line, which compensates for the LH inducing role of the missing GnRH1 line in Cyprinidae.

GONADOTROPIN SECRETION AND CATECHOLAMINE METABOLISM IN TELEOSTS

Teleost species possess two distinct gonadotropins, GtH-I and GtH-II. This is the case of seabream, striped bass, salmon and trout, whereas only one gonadotropin could be found in catfish. GtH I and GtH II have distinct temporal expression and release profile in teleosts. In rainbow trout, β GtH- I mRNAs are present throughout all stages of vitellogenesis, while β GtH- II transcripts increase mainly at the time of ovulation. In sexually immature salmon, *in vivo* E2 treatment increases GtH-II b transcript levels, but did not alter GtH-I β or α subunits. GtH-I is released over the entire vitellogenesis, whereas GtH-II remains low during vitellogenesis and exhibits a sharp peak before ovulation (Davies et al., 1995). These different patterns of expression and release demonstrate that there is obviously a differential control of GtH-I and GtH-II (Saligaut et al., 1999).

REPRODUCTIVE CYCLE AND THE CONTROL OF GtHS RELEASE

To describe reproduction controll by hormone, we may first describe GTH-II which is more important than GTH-I in most fishes. Actually GTH-I and GTH-II are equipotent in stimulating 11-ketotestosterone (11KT) and 17α, 20βP production (Planas et al., 1991). Later, however, GTH-II was more potent than GTH-I in stimulating 17α, 20βP

synthesis. Thus, early GnRH treatment may have had a relatively greater impact on GTH-I production while the effectiveness of GnRHa injection at the height of the spawning season is consistent with increase impact on GTH-II production and 17α,20βP synthesis at that stage of the reproduction cycle.

A DA inhibitory tone on GtH-II secretion has been reported in goldfish, common carp, European eel and catfish. A bimodal inhibitory mechanism of DA, direct on gonadotropic cells and indirect via GnRH, has been suggested (De leeuw al., 1986; Zohar et al., 2010; Trudeau et al., 1993).

In salmon, DA modulated GTH release only after administration of a LHRH analoge (Van der Kraak et al., 1986). However, Billard et al. (1983) demonstrated in trout that pimozide (a DA antagonist) alone could increase plasma GtH. Some changes of catecholamine metabolism occurred during the reproductive cycle of the rainbow trout: decrease of DA turnover in pituitary and hypothalamus during the periovulatory period, increase of DA contents in pituitary and hypothalamus during the periovulatory period, increase of DA contents in preoptic area as vitellogenesis proceeds (Saligaut et al., 1992; Saligaut et al., 1999). In using the reproductive cycle by the control, the GtH-I release blood levels in immature and vitellogenic trout are much higher than those of GtH-II and can be compared with those found in salmon in the literature (Dickey and Swanson, 1995). E2 implantation decreases blood GtH I levels in immature rainbow trout. In vitellogenic trout, ovariectomy significantly increases blood GtH I levels, like coho salmon (Larsen and Swanson, 1997), and E2-supplementation prevents this increase (Saligaut et al., 1988). GtH release depends then in immature and vitellogenic trout, like in salmon, upon a negative oestrogenic feedback (Saligaut et al., 1999).

Gametogenesis and final maturation in fish is Similar to other vertebrates (Mylonas et al., 2010).In mammals, spermiation is known as a complex process by which elongated spermatids undergo their final maturation and are released from supporting Sertoli cells into the tubule lumen, which is open at both ends (Beardsley and O'Donnell, 2003). This process has been described at the morphological level, but its control remains poorly understood, although it is known that it requires the actions of both gonadotropins (follicle- stimulating hormone, FSH and luteinizing hormone, LH), as well as intratesticular testosterone (T) (Saito et al., 2000). Similar to mammals, there is a close morphological and functional intercellular communication between Sertoli cells and germ cells during fish spermatogenesis (De Montgolfier et al., 2007; Mylonas et al., 2010; Loir et al., 1995).

ENDOCRINOLOGY OF FISH REPRODUCTION

Gametogenesis (spermatogenesis and vitellogenesis) and final maturation (spermiation and OM) are regulated by a cascade of hormones along the brain–pituitary–gonad (BPG) axis. In this axis, the secretion of the pituitary gonadotropins FSH and LH is controlled by the brain via the stimulatory action of the GnRHs (Peter and Yu, 1997; Yu et al., 1997), which are the primary neuropeptides regulating reproduction, acting as integrators of external information (example, environment, temperature, water fall and social interactions). Dopamine (DA) as mentioned before in some fishes exerts a negative ef-fect on the functions of GnRH on the pituitary gonadotrophs (Chang and Jobin, 1994). The FSH and LH are released into the bloodstream to act on the gonad, where they stimulate the synthesis of the sex steroid hormones (androgens, estrogens and progestogens), which are the ultimate effectors of gonadal development.

Hormonal regulation of fish spermatogenesis and spermiation has been described (Mylonas et al., 2010). Testicular spermatogenesis, as well as spermiation, is regulated by pituitary FSH and LH secretion through the action of the sex steroid hormones, as well as other growth factors. Before the onset of spermatogenesis, spermatogonial stem cell renewal seems to be regulated by E2 acting on Sertoli cells (Miura and Miura, 2003). The androgen 11-keto testosterone (11KT) is the major regulator of spermatogenesis, while the maturation inducing steroid (MIS) regulates sperm capaciation and spermiation (Miura and Miura, 2003).

Both steroids are synthesized by the somatic Leydig cells of the testes, after GtH stimulation. The LH is mainly involved in the stimulation of androgen production in Leydig cells, whereas FSH seems to exert more complex functions in the male testes, stimulating androgen production from the Leydig cells, as well, but also regulating Sertoli cell activity during spermatogenesis.

The onset of spermatogenesis is a process controlled by the secretion of pituitary GtHs (mainly FSH). The FSH acts on Sertoli cells and stimulates 11KT biosynthesis, which in turn regulates the full process of spermatogenesis, mediated also by growth factors (example, insulin-like growth factor I, IGF-I or activin B) secreted by the Sertoli cells.

In males, FSH levels are high at early spermatogenesis; on the other hand, LH is low during early spermatogenesis. In males, androgen production (T and 11KT) remains high through the entire spawning period, even while MIS levels are high, since spermatogenesis, spermiogenesis and spermiation occur concurrently. In females, a predominant role has been suggested for FSH during vitellogenesis in fishes with synchronous ovarian development. On the other hand, in fish with asynchronous ovarian development the role of FSH in vitellogenesis is less clear and a possible function has been ascribed also to LH.

The control of reproduction with the use LH has been worked in bakish water fishes. Grey mullet, Mugil cephalus L. is a marine teleost suitable for culture in brackish water. Todate, very little report on artificial propagation of grey mullet has ever been recorded because of its difficulty to

artidicially reproduction and still seed stocking relies on wild recruitment. The use of mammalian gonadotrophins and synthetic analog of luteinizing hormone releasing hormone (LRH-A2) for initiation and advancement and ovarian development is achived when it was used at proper time of eggs development. For successful spawning, a priming injection for females should be when an average egg diameter was 600 mm or more. Riped ova have a thin layer cytoplasm covering the yolk and have a single oil droplet. In this case, the egg diameter was 960 µm and oil globule 360 µm (Yousefian et al., 2006).

At the conclusion of vitellogenesis, OM is triggered by the action of LH on the follicle cells, which synthesize and secrete the maturation inducing hormone (MIH) or maturation inducing steroid (MIS) (Nagahama et al., 1994; Suwa and Yamashita, 2007). In salmonids (*Onchorhynchus and Salmo spp*.), and a few freshwater and marine fishes the MIS is the progestin 17,20b-dihydroxy-4pregnen-3one (17,20bP). In some other marine species, a derivative of 17,20bP the 17a, 20b, 21-trihydroxy-4-pregnen-3-one (20bS) has been described to act as MIS. Both 17,20bP and 20bS are acting as MIH in European sea bass, striped bass and red seabream (Suwa and Yamashita, 2007; Mylonas et al., 2010). The MIS binds to specific receptors on the oocyte plasma membrane and the signal received in the oocyte surface is transduced to the cytoplasm to finally result in the formation and activation of the maturation-promoting factor (MPF), which is responsible for the resumption of meiosis and completion of oocyte maturation (Mylonas et al., 2010; Nagahama et al., 1994).

REPRODUCTIVE DYSFUNCTIONS IN CULTURED FINFISH

By using a series of selected articles, Mylonas and Zohar (2001) described reproductive dysfunctions in cultured finfish.Reproduction is regulated by the brain via the release of GnRH from the hypothalamus and it stimulates the release of gonadotropin (GtH) from the pituitary. Dopamine provides a negative control of pituitary GtH release, mostly freshwater species. Fish possess two or three different variants of GnRH, and 14 variants have been so far identified from various vertebrates (Mylonas and Zohar, 2001). Pituitary control of reproduction is via a dual GtH system (Schulz, 1995), with follicle stimulating hormone (FSH or GtH I) regulating vitellogenesis and spermatogenesis, and luteinizing hormone (LH or GtH II) regulating FOM and spermiation.

In female, most cultured species exhibit some degree of reproductive dysfunction when reared in captivity. Problems are more widespread in female broodstock and can vary from inconsistent spawning only, to the complete failure of oogenesis (Mylonas and Zohar, 2001). The most commonly observed reproductive dysfunction in captive fish, especially marine species, is the unpredictable occurrence or absence of FOM. The failure of captive fish to undergo FOM in captivity in some fish was found to be the absence of LH release during the spawning season. Fish that exhibit this type of dysfunction undergo normal vitellogenesis, but with the onset of the spawning season the developing oocytes fail to initiate FOM; instead they undergo atresia. Treatment of such broodstock with exogenous GtH or GnRHa at the completion of vitellogenesis stimulates gonadal steroidogenesis FOM and ovulation.

The final and most severe form of reproductive dysfunction of captive female broodstock is the failure to undergo vitellogenesis. During vitellogenesis, FSH or LH stimulate the production of testosterone (T) by the theca cells and its aromatization to 17β -estradiol (E2) in the granulosa (Nagahama, 1994). In response to stimulation by E2 the liver produces vitellogenin, which is sequestered by the oocytes in a receptor-mediated process enhanced by FSH. At the completion of vitellogenesis a surge in plasma LH stimulates a drop in plasma E2, a transient increase in plasma T during GV migration, and a dramatic elevation in the plasma levels of the maturation inducing steroid (MIS), which acts at the level of the oocyte membrane to induce FOM (Nagahama, 1994; Nagahama et al., 1994; Mylonas and Zohar, 2001; Peter and Yu, 1997). In male gonadotropins regulate spermatogenesis via the production of androgens by the testes, mainly 11-ketotestosterone, since T is the precursor of 11-KT, the levels of the two androgens covary during most of the reproductive season. Plasma 11-KT levels peak during spermiogenesis and decline just prior to, or during the spermiation period (Mylonas and Zohar, 2001).

ENDOCRINE CONTROL OF GAMETOGENESIS AND fiNAL MATURATION

The most common dysfunctions include the production of lower quantity of milt and/or sperm during the spermiation period and the failure to undergo OM at the completion of vitellogenesis. The endocrine cause of the failure of female fish to undergo OM has been identified to be a dysfunctional release of LH from the pituitary at the end of vitellogenesis (Mylonas *et al*., 2010).

However, LH was synthesized and stored in the pituitary during vitellogenesis, since levels of LH and its mRNA in the pituitary did not differ between wild and captive females, demonstrating that the problem is one of lack of release and not synthesis in captivity. The disruption in LH release from the pituitaries of captive fish is not due to a dysfunction in pituitary responsiveness, but may be related to the control of pituitary function by the reproductive brain (Mylonas et al., 2010). In fact, differences were observed between wild and captive females undergoing OM, when comparing the pituitary content of the endogenous GnRHs.

The GnRH mRNA levels within the brain, however, were

similar between the two groups, indicating that the altered pituitary content of GnRH in captive fish may be a result of altered release from the hypothalamus, rather than deficient synthesis (Steven, 2000; Steven et al., 2000). There were differences between GnRH measured in the pituitary and the same values of GnRH mRNA in the brain of wild fishes and farmed organisms (Steven et al., 2000). These data suggest that GnRH synthesis in the hypothalamus is not disrupted, but that the problem concerns GnRH secretion from nerve terminals in adenohypophysis.

In male reproductive dysfunctions of captive fishes are not restricted to females, since males may produce a reduced amount of milt and of lower quality, even though they do undergo complete spermatogenesis and spermiation in captivity (Mylonas et al., 2010). Lower plasma levels of LH during the spermiation period have been suggested as the cause of the reduced amount of milt produced by some fishes (Mañanos et al., 2002; Mylonas and Zohar, 2001a). The amount of LH in the pituitary or the ability of the pituitary to synthesize LH in response to treatment with exogenous GnRHa is not affected in these fishes, suggesting that again the reproductive dysfunction in the males may be identified in the brain control of GtH synthesis and/or release (Mylonas et al., 2010).

REFERENCES

Amoss M, Burgus R, Blackwell R, Vale W, Fellows R, Guillemin R (1971). Purification, amino acid composition and N-terminus of the hypothalamic luteinizing hormone releasing factor (LRF) of ovine origin. Biochem. Biophys. Res. Commun., 44: 205 - 210.

Aroua S, Weltzien FA, Le Belle N, Dufour S (2007). Development of real-time RT-PCR assays for eel gonadotropins and their application to the comparison of in vivo and in vitro effects of sex steroids. Gen. Comp. Endocrinol., 153: 333 - 343.

Burgus R, Butcher M, Ling N, Monahan M, Rivier J, Fellows R, Amoss M, Blackwell R, Vale W, Guillemin R (1971). Molecular structure of the hypothalamic factor (LRF) of ovine origin monitoring the secretion of pituitary gonadotropic hormone of luteinization (LH). Weekly reports of meetings of the Academy of Sciences. Serie D: Sciences Naturelles, 273: 1611-1613.

Bromage N (1992). Propagation and stock improvement. In: J. Sheperd and N.Bromage (eds.) Intensive Fish Farming. Blackwell Science Ltd., Oxford, UK, pp. 103-153.

Billard R, Alagarswami K, Peter RE, Breton B (1983). Potentialisation par le pimozide des effets du LHRH-A sur la se´cre´tion gonadotrope hypophysaire, l'ovulation et la spermiation chez la carpe commune, *Cyprinus carpio*. C.R. Acad. Sci. Paris., 296: 181-184.

Billard R (1986). Spermatogenesis and spermatology of some teleost fish species. Reprod. Nutr. Dev., 26: 877-920.

Billard R (1989). Endocrinology and fish culture. Fish Physiol. Biochem., 7: 49-58.

Beardsley A, O'Donnell L (2003). Characterization of normal spermiation and spermiation failure induced by hormone suppression in adult rats. Biol. Reprod., 68: 1299-1307.

Chen CC, Fernald RD (2008). GnRH and GnRH receptors: distribution, function and evolution. J. of Fish Biol., 73: 1099-1120.

Chang JP, Peter RE (1983). Effects of dopamine on gonadotropin release in female goldfish, *Carassius auratus*. Neuroendocrinol., 36: 351-357.

Chang JP, Jobin RM (1994). Regulation of gonadotropin release in vertebrates: a comparison of GnRH mechanisms of action. In: Davey, K.G., Peter, R.E., Tobe, S.S. (Eds.), Perspectives in Comparative Endocrinology. National Research Council of Canada, Ottawa, pp. 41–51.

Cardinaud B, Gilbert JM, Liu F, Sugamori KS, Vincent JD, Niznik HB, Vernier P (1998). Evolution and origin of the diversity of dopamine receptors in vertebrates. Adv. Pharmacol., 42: 936-940.

Crim LW, Evans DM (1983). Influence of testosterone and/or luteinizing hormone releasing hormone analogue on precocious sexual development in the juvenile rainbow trout. Biol. Reprod., 29: 137-142.

Crim LW, Peter RE, Van Der Kraak GV (1987). The use of LHRH analogues in aquaculture. In*:* B.H. Vickery and JJ. Nestor Jr, editors. LHRH and its analog: Contraceptive and therapeutic applications, part 2, MTP Press, Boston, MA. pp 489-498.

Davies B, Swanson P, Bromage N (1995). The effects of photoperiod and temperature on serum GtH I and GtH II and the timing of maturation in the female rainbow trout. In: Goetz, F.W., Thomas, P. (Eds.), Reproductive Physiology of Fish V. Proceedings of the Fifth International Symposium, 02 July–08 July 1995. Fish Symposium 95, Austin, USA, pp. 185-186.

De Leeuw R, Goos HJTh, Van Oordt PGWJ (1986). The dopaminergic inhibition of the gonadotropin-releasing hormone-induced gonadotropin release: an in vitro study with fragments and cell suspensions from pituitaries of African catfish, *Clarias gariepinus,* Burchell. Gen. Comp. Endocrinol., 63: 171-177.

De Montgolfier B, Dufresne J, Letourneau M, Nagler J, Fournier A, Aude TC, Cyr DG (2007). The expression of multiple connexins throughout spermatogenesis in the rainbow trout testis suggests a role for complex intercellular communication. Biol. Reprod., 76: 2-8.

Dickey JT, Swanson P (1998). Effects of sex steroids on gonadotropin (FSH and LH) regulation in coho salmon (*Oncorhynchus kisutch*). J. Mol. Endocrinol., 21: 291-306.

Donaldson EM, Hunter GA (1983). Induced final maturation, ovulation and spermiation in cultured fishes. In: Hoar, W.S., Randall, D.J., Donaldson, .M. (Eds.), Fish Physiology. Academic Press, Orlando, FL, pp. 351-403.

Fernald RD, White RB (1999). Gonadotropin-releasing hormone genes: phylogeny, structure, and functions. Frontiers in Neuroendocrinol., 20: 224-240.

Fostier A, Jaiabert B (1982). Physiological basis of practical means to induce ovulation in fish, *In:* C.JJ. Richter, HJ.Th. Goos, compilers. Proceedings of the International Symposium of Reproductive Physiology of Fish. Wagenigen, the Netherlands, 2-6 August 1982, Pudoc, Wagenigen. pp. 164-173.

Goetz FW (1983). Hormonal control of oocyte maturation and ovulation in fishes. In: Fish Physiology Vol IX, Part B. Eds. W.S. Hoar, D.J. Randall and E.M. Donaldson. Academic Press, New York, pp. 117-169.

Harvey B, Hoar WS (1979). The theory and practise of induced breeding in fish. IDRC-TS2le, P. 48. ..

Kah O, Lethimonier Ch, Somoza G, Guilguir L, Vail-lant C, Lareyre JJ (2007). GnRH and GnRH receptors in Metazoa: A historical, comparative and evolutive perspective. Gen. Comparat. Endocrinolol., 153: 346-364.

Kebabian JW, Calne DB (1979). Multiple receptors for dopamine. Nature, 277: 93-96.

Larsen DA, Swanson P (1997). Effects of gonadectomy on plasma gonadotropins I and II in coho salmon, *Oncorhynchus kisutch*. Gen. Comp. Endocrinol., 108: 152-160.

Lam TJ, Pandey S, Hoar WS (1975). Induction of ovulation in goldfish by synthetic luteinizing hormone-releasing hormone (LHRH). Can. J. Zool., 53: 1189-1192.

Lam TJ (1982). Applications of endocrinology to fish culture. Can. J. Aquat. Fish. Sci., 39: 11-137.

Lam TJ (1983). Environmental influences on gonadal activity in fish. In: Hoar, W.S., Randall, D.J., Donaldson, E.M. (Eds.). Fish Physiology. Reproduction, Part B, vol. IX, Academic Press, New York, pp. 65-116.

Lam TJ (1985). Induced spawning in fish,. In*:* C.S. Lee and I.C. Liao, editors. Reproduction and culture of milkfish. The Oceanic Institute, HI, and Tungkang Marine Laboratory, Taiwan. pp. 14-56.

Lam T JMunro AD (1987). Environmental control of reproduction in teleosts: an overview. pp. 279-288. In: D. R. Idler, L.W. Crim, and J.M. Walsh, editors. Proceeding of the Third International Symposium on Reproductive Physiology of Fish. MSRL Memorial Univ. of Newfoundland. St. John's Newfoundland, Canada.

Lethimonier C, Madigou T, Munoz-Cueto JA, Lareyre JJ, Kah O (2004). Evolutionary aspects of GnRHs, GnRH neuronal systems and GnRH

receptors in teleost fish. Gen. Comp. Endocrinol., 135: 1-16.
Loir M, Sourdaine P, Mendis-Handagama SM, Jegou B (1995). Cell–cell interactions in the testis of teleosts and elasmobranchs. Microsc. Res. Tech., 32: 533-552.
Lin HR, Kraak GVD, Liang JY, Peng C, Li GY, Lu LZ, Zhou XJ, Chang ML, Peter RE (1986). The effects of LHRH analogue and drugs which block the effects of dopamine on gonadotropin secretion and ovulation in fish cultured in China. In: Bil-lard R., Marcel J. (eds.): Aquaculture of Cyprinids. INRA, Paris, 502: 139-150.
Lin HR, Peter RE (1996). Hormones and Spawning in fish. Asian Fisheries Science Manila, Philippines, 9: 21-33.
Matsuo H, Baba Y, Nair RM, Arimura A, Schally AV (1971). Structure of the porcine LH- and FSH-releasing hormone. I. The proposed amino acid sequence. Biochem. Biophys. Res. Commun., 43: 1334-1339.
Mañanos E, Carrillo M, Sorbera LS, Mylonas CC, Asturiano JF, Bayarri MJ, Zohar Y, Zanuy S (2002). Luteinizing hormone and sexual steroid plasma levels after treatment of European sea bass with sustained-release delivery systems for gonadotropin-releasing hormone analogue. J. Fish Biol., 60: 328-339.
Millar RP, Lu ZL, Pawson AJ, Flanagan CA, Morgan K, Maudsley SR (2004). Gonadotropin-releasing hormone receptors. Endocrine Reviews, 25: 235- 275.
Miura T, Miura CI (2003). Molecular control mechanisms of fish spermatogenesis. Fish Physiol. Biochem., 28: 181-186.
Mylonas C, Zohar Y (2001). Use GnRH-delivery systems for the control of reproduction in fish, reviews in fish biology and fisheries, 10: 463-49.
Mylonas CC, Zohar Y (2001a). Endocrine regulation and artificial induction of oocyte maturation and spermiation in basses of the genus Morone. Aquaculture, 202: 205-220.
Mylonas CC, Zohar Y (2007). Promoting oocyte maturation, ovulation and spawning in farmed fish. In: Babin, P.J., Cerdá, J., Lubzens, E. (Eds.), The Fish Oocyte: from Basic Studies to Biotechnological Applications. Kluwer Academic Publishers, Dordrecht, the Netherlands, pp. 433-470.
Mylonas CC, Fostier A, Zanuy S (2010). Broodstock management and hormonal manipulations of fish reproduction. Gen. Comp. Endocrinol., 165: 516-534.
Nozaki M, Naito N, Swanson P, Miyata K, Nakai Y, Oota Y, Suzuki K, Kawauchi H (1990). Salmonid pituitary gonadotrophs. I. Distinct cellular distributions of two gonadotropins, GTH I and GTH II. Gen. Comp. Endocrinol., 77: 348-357.
Nieuwenhuys R, Donkelaar HJt, Nicholson C (1998). In: The Central Nervous System of Vertebrates. Springer, Berlin, New York.
Nagahama Y (1983). The functional morphology of teleost gonads. *In*: Hoar, W.S., Randall, D.J. and Donaldson, E.M. (eds.), *Fish Physiology. Vol. IX, Part A: Reproduction*. Academic Press, Orlando, pp. 223-275.
Nagahama Y (1994). Endocrine control of gametogenesis. Int. J. Dev. Biol., 38: 217-229.
Nagahama Y, Yoshikuni M, Yamashita M, Tanaka M (1994). Regulation of oocyte maturation in fish. *In*: Farrel, A.P. and Randall, D.J. (eds.), *Fish Physiology. Vol. XIII:* Molecular Endocrinology of Fish. Academic Press, San Diego, California, pp. 393-439.
Olivereau M, Ball JN (1964). Contribution to the histophysiology of the pituitary gland of teleosts, particularly those of the *Poecilia* species. Gen. Comp. Endocrinol., 47: 523-532.
Olivereau M (1976). Gonadotropin secreting cells of the pituitary gland of the Atlantic salmon: unity or duality? Gen. Comp. Endocrinol., 28: 82-95.
Peng C, Gallin W, Peter RE, Blomqvist AG, Larhammar D (1994). Neuropeptide-Y gene expression in the goldfish brain: distribution and regulation by ovarian steroids. Endocrinology, 134: 1095-1103.
Peter RE, Chang JP, Nahorniak CS, Omeljaniuk RJ, Sokolowska M, Shih SH, Billard R (1986). Interactions of catecholamines and GnRH in regulation of gonadotropin secretion in teleost fish. Recent Progress in Hormone Research, 13: 229-239.
Peter RE, Lin HR, Van der Kraak G (1987). Drug/hormone induced breeding of Chinese teleosts, *In*: D, R. Idler, L.W. Crim, and J.M. Walsh, editors. Proceedings of the Third International Symposium on Reproductive Physiology of Fish, MSRL, Memorial University of New found l and, St.John's, Newfoundland, Canada. pp. 120-123.
Peter RE, Lin HR, Van Der Kraak G (1988). Induced ovulation and spawning of cultured freshwater fish in China: advances in application of GnRH analogues and Dopamine antagonists. Aquaculture, 74: 1-10.
Peter RE, Trudeau VL, Sloley BD (1991). Brain regulation of reproduction in teleosts. Bulletin of the Institute of Zoology, Academia Sinica, 16: 89-118.
Peter RE, Lin HR, van der Kraak G, Little M (1993). Releasing hormones, dopamine antagonists and induced spawning. *In*: Muir, J.F. and Roberts, R.J. (eds.), *Recent Advances in Aquaculture. Vol. IV*. Blackwell Scientific Publications, Oxford, pp. 25-30.
Peter RE, Yu KL (1997). Neuroendocrine regulation of ovulation in fish: Basic and applied aspects. Rev. Fish Biol. Fish. 7: 173-197.
Podhorec P, Kouril J (2009). Induction of final oocyte maturation in cyprinidae fish by hypothalamic factors: a review.Veterinarni medicina, 54 (3): 97-110.
Pankhurst NW (1994). Effects of gonadotropin releasing hormone analogue, human chorionic gonadotropin and gonadal steroids on milt volume in the New Zealand snapper, *Pagrus auratus (Sparidae)*. Aquaculture, 125:185-197.
Pankhurst NW, Thomas PM (1998). Maintenance at elevated temperature delays the steroidogenic and ovu-latory responsiveness of rainbow trout Oncorhynchus mykiss to luteinizing hormone releasing hormone analogue. Aquaculture, 166: 163-177.
Planas JV, Swanson P, Dickhoff WW (1991). Regulation of testicular steroidogenesis by coho salmon gonadotropins, GTH I and GTH II, in vitro. In: Scott, A.P., Sumpter, J.P., Kime, D.E., Rolfe, M.S (Eds.), Proceedings of the Fourth International Symposium on Reproductive Physiology of Fish, University of East Anglia, Norwich, UK, p. 103.
Rainis S, Mylonas CC, Kyriakou Y, Divanach P (2003). Enhancement of spermiation in European sea bass *(Dicentrarchus labrax)* at the end of the reproductive season using GnRHa implants. Aquaculture, 219: 873-890.
Schulz RW (1995). Physiology, morphological, and molecular aspects of gonadotropins in fish with special reference to the African catfish, *Clarias gariepinus*. *In*: Goetz, F.W. and Thomas, P. (e d s.), *Reproductive Physiology of Fish*. Fish Symposium 95, Austin, Texas, pp. 2-6.
Suwa K, Yamashita M (2007). Regulatory mechanisms of oocyte maturation and ovulation. In: Babin, P.J., Cerdá, J., Lubzens, E. (Eds.), The Fish Oocyte: From Basic Studies to Biotechnological Applications. Kluwer Academic Publishers, Dordrecht, the Netherlands.
Saito K, O'Donnell L, McLachlan RI, Robertson DM (2000). Spermiation failure is a major contributor to early spermatogenic suppression caused by hormone withdrawal in adult rats. Endocrinol., 141: 2779-2785.
Saligaut C, Salbert G, Bailhache T, Bennani S, Jego P(1992). Serotonin and dopamine turnover in the female rainbow trout, *Oncorhynchus mykiss*. Brain and pituitary: changes during the annual reproductive cycle. Gen. Comp. Endocrinol., 85: 261-268.
Saligaut C, Linard B, Mananos EL, Kah O, Breton B, Govoroun M (1998). Release of pituitary gonadotrophins GtH I and GtH II in the rainbow trout (*Oncorhynchus mykiss*): modulation by estradiol and catecholamines. Gen. Comp. Endocrinol., 109: 302-309.
Saligaut C, Linard B, Breton B, Anglade I, Bailhache T, Kah O, Jego P (1999). Brain aminergic systems in salmonids and other teleosts in relation to steroid feedback and gonadotropin release. Aquaculture, 177:13-20.
Shafiei Sabet S, Imanpour MR, Aminian Fatideh B, Gorgin S (2009). Relation between gonadal hormones and sexual maturity of female kutum (*Rutilus frisii kutum* Kamenskii, 1901) during spawning season. J. cell and mol. Res., 1(2): 96-104.
Steven C (2000). Studies on the GnRH–GtH system of female striped bass (*Morone saxatilis*): effects of GnRH agonist therapy and comparison of reproductive endocrine parameters between wild and captive fish. Marine Estuarine and Environmental Sciences. University of Maryland, College Park.
Steven C, Gothilf Y, Holland MCH, Stubblefield J, Mylonas CC, Zohar Y (2000). Differential expression of the three GnRH genes in wild and captive striped bass, *Morone saxatilis*, in response to natural and hormonally induced maturation. p. 66. In: Norberg B., Kjesbu O.S., Taranger G.L., and Andersson E., Stefansson S.O. (eds.): Reproductive Physiology of Fish. University of Bergen, Bergen, Norway. pp. 326.
Steven C, Lehnen N, Kight K, Ijiri S, Klenke U, Harris WA, Zohar Y (2003). Molecular characterization of the GnRH system in zebrafish (*Danio*

rerio): cloning of chicken GnRH-II, adult brain expression patterns and pituitary content of salmon GnRH and chicken GnRH-II. General and Comparative Endocrino, 133: 27-37.
Shimizu A, Tanaka H, Kagawa H (2003). Immunocytochemical applications of specific antisera raised against synthetic fragment peptides of mummichog GtH subunits: examining seasonal variations of gonadotrophs (FSH cells and LH cells) in the mummichog and applications to other acanthopterygian fishes. Gen. Comp. Endocrinol., 132: 35-45.
Somoza GM, Miranda LA, Strobl-Mazzulla P, Guilgur LG (2002). Gonadotropin-Releasing Hormone (GnRH): From fish to mammalian brains. Cellular and Molecular Neurobiology, 22: 589-609.
Sherwood NM, Eiden L, Brownstein M, Spiess J, Rivier J, Vale W (1983). Characterization of a teleost gonadotropin-releasing hormone. Proceedings of the National Academy of Sciences of the United States of America, 80: 2794-2798.
Trudeau VL, Sloley BD, Wong AOL, Peter RE (1993). Interactions of gonadal steroids with brain dopamine and gonadotropin-releasing hormone in the control of gonadotropin-II secretion in the goldfish. Gen. Comp. Endocrinol., 89: 39-50.
Trudeau VL (1997). Neuroendocrine regulation of gonadotrophin II release and gonadal growth in the goldfish, Carassius auratus. Rev. Reprod., 2: 55-68.
Tucker JW (1994). Spawning of captive serranid fishes: a review. J. World Aquat. Soc., 25: 345-359.
Vacher C, Mananos EL, Breton B, Marmignon MH, Saligaut C (2000). Modulation of pituitary dopamine D1 or D2 receptors and secretion of follicle stimulating hormone and luteinizing hormone during the annual reproductive cycle of female rainbow trout. J. Neuroendocrinol., 12: 1219-1226.
Vacher C, Ferriere F, Marmignon MH, Pellegrini E, Saligaut C (2002). Dopamine D2 receptors and secretion of FSH and LH: role of sexual steroids on the pituitary of the female rainbow trout. Gen. Comp. Endocrinol., 127: 198–206.
Van Der Kraak G, Donaldson EM, Chang JP (1986). Dopamine involvement in the regulation of gonadotropin secretion in coho salmon. Can. J. Zool., 64(6): 1245-1248.
Von Ihering R (1937). A Method for inducing fish to spawn. The Progressive Fish-Culturist, 34: 15-16.
Van der Kraak G, Donaldson EN, Chang JP (1986). Dopamine involvement in the regulation of gonadotropin secretion in coho salmon. Can. J. Zool., 64: 1245-1248.
Yaron Z, Gal G, Melamed P, Rosenfeld H, Elizur A, Levavi-Sivan B (2003). Regulation of fish gonadotropins. Int. Rev. Cytol., 255: 131-185.
Yaron Z, Sivan B (2006). Reproduction. In: Evans D.H., Claibourne J.B. (eds.): The Physiology of Fishes. CRC Press, Boca Raton, 601: 343-386.
Yousefian M, Ghanei M, Pourgolam R, Rostami HKh (2009). Gonad Development and Hormonal Induction in Artificial Propagation of Grey Mullet, *Mugil Cephalus* L. Res. J. Fisheries Hydrobiol., 4(2): 35-40.
Yousefian M, Mosavi H (2008). Spawning of South Caspian Kutum (*Rutilus frisii kutum*) in most Migratory River of south Caspian Sea. Asian Journal of Animal and Veterinary Advance. p.6.
Yousefian M, Gezel HG, Hedayatifard M (2008). Induce of ovulation in endemic *Chalcaburnus chalcoides*, living in the Caspian Sea, using LRH-Aa combined with methoclopramide. Afr. J. Biotechnol., 7: 22.
Yu KL, Lin XW, da Cunha Bastos J, Peter RE (1997). Neural regulation of GnRH in teleost fishes. In: Parhar, I.S., Sakuma, Y. (Eds.), GnRH Neurons: Gene to Behavior. Brain Shuppan, Tokyo, pp. 277-312.
Zohar Y, Pagelson G, Tosky M, Finkelman Y (1987). GnRH control of gonadotropin secretion, ovulation and spawning in the gilthead seabream, Sparus aurata. Proceeding of the Third International Symposium on Reproductive Physiology of Fish: p. 106.
Zohar Y (1989). Fish reproduction: Its physiology and artificial manipulation. *In*: Shilo, M. and Sarig, S. (eds.), *Fish Culture in Warm Water Systems: Problems and Trends*. CRC Press, Boca Raton, pp. 65-119.
Zohar Y, Mylonas C (2001). Endocrine manipulations of spawning in cultured fish: from hormones to genes. Aquaculture 197: 99-136.
Zohar Y, Muñoz-Cueto JA, Elizur A, Kah O (2010). Neuroendocrinology of reproduction in teleost fish. Gen. Comp. Endocrinol., 165: 438-455.