Dopaminergic Regulation of Gonadotropin-II Secretion in Testosterone-treated Precocious Male and Immature Rainbow Trout *Oncorhynchus mykiss*

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The present work examined the role of gonadotropin-releasing hormone (GnRH) and dopaminergic drugs on the secretion of maturational gonadotropin (GTH II) in relation to testosterone (T) treatment. This study provides evidence that the plasma GTH II levels are increased by T treatment in precocious males, but not in the immature animal. In addition, GnRH analogue (GnRHa) alone significantly increased the plasma GTH II secretion in immature rainbow trout treated with T, as well as in T-treated and T-untreated precocious males. However, injection with either dopamine (DA) or domperidone (DOM; DA D2 receptor antagonist) alone did not alter the basal plasma GTH II secretion in all experimental groups. The secretion of GTH II in the T-treated precocious males was remarkably influenced by GnRHa or combination of dopaminergic drugs. Notably, the effects of dopaminergic drugs on GnRHa-induced GTH II secretion was prolonged by T in precocious males. In T-treated immature animals, GnRHa-induced GTH II secretion was increased only by a dose DOM (10 μg/g body wt) but not by higher dose DOM (100 μg/g body wt). In the T-untreated immature rainbow trout, however, plasma GTH II secretion was not influenced by the same treatments. Therefore, these results indicate that DA may be acting indirectly by blocking the effect of GnRH on GTH II secretion *in vivo*. T may act to modulate the relative contribution by the stimulatory (GnRH) and inhibitory (DA) neuroendocrine factors, which would ultimately determine the pattern of GTH II secretion.

In teleosts, secretion of maturational gonadotropin (GTH II) from the pituitary is under the control of several regulatory mechanisms which include an excitatory gonadotropin-releasing hormone (GnRH) system, facilitatory or inhibitory central monoaminergic systems, and positive or negative feedback regulation of gonadal steroids (reviewed by Peter et al., 1986, 1991; Saligaut et al., 1999). Among the central monoamines, dopamine (DA) might act on the gonadotrophs directly by inhibiting basal GTH II secretion and indirectly by blocking the effect of GnRH on GTH II secretion in goldfish, *Carassius auratus* (Chang and Peter et al., 1983). In Cyprinids, intraperitional injection of pimozide, the DA D1 and D2 receptor antagonist, has been shown to potentiate the effects of GnRH, such as GTH II secretion (reviewed by Peter et al., 1986). Furthermore, studies on DA receptors have shown that dom-

peridone, the DA D2 receptor antagonist, enhances pituitary GnRH binding capacity in the goldfish and African catfish *Clarias gariepinus* (De Leeuw et al., 1988, 1989).

Gonadal steroids were reported to potentiate the effect of exogenously administered GnRH analogue (GnRHa) in a season-dependent manner (Sokolowska et al., 1985). It has been further shown that circulating gonadal steroids and brain catecholamines modulate the GnRH activity at the level of the brain or pituitary (Trudeau et al., 1993). Gonadal steroids enhanced both GnRH-induced GTH II secretion and the GTH II response to domperidone in goldfish (Trudeau et al., 1993). It was reported that testosterone (T) stimulated the synthesis of salmon GnRH (sGnRH) and the accumulation of pituitary GTH II in masu salmon *Oncorhynchus masou* (Amano et al., 1994). Also, plasma GTH II secretion of immature rainbow trout *Oncorhynchus mykiss* treated with T was stimulated by native forms of GnRH or GnRHa (Crim et al., 1988). Saligaut et al. (1992) found that the aminergic turnover

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rate in the brain of rainbow trout changes according to the endogenous steroid conditions. Notably, the inhibitor of catecholamine synthesis, α-methyl-*p*-tyrosine (MPT), caused an increase in the plasma GTH II levels in 17β-estradiol (E_2)-implanted immature and vitellogenic rainbow trout (Linard et al., 1995; Saligaut et al., 1998). Therefore, it is assumed that gonadal steroids act on the brain-pituitary axis by modulating a number of neuronal systems directly involved in the control of GTH II secretion.

Although the involvement of brain catecholamines in gonadal steroid feedback control of GTH II secretion in teleosts has been suggested by several investigators (De Leeuw et al., 1987; Trudeau et al., 1993; Senthilkumaran and Joy, 1996; Saligaut et al., 1998), no direct evidence for such interactions has been provided. In a certain strain of rainbow trout, females mature at three years of age, while most males mature precociously in the first and second autumns. Using this strain of rainbow trout, we demonstrate the existence of a tonic DA inhibition of GTH II secretion and a potential role of T in such regulation in precocious male and immature rainbow trout.

Materials and Methods

General

One-year-old precocious male and immature rainbow trout, weighing about 100 g, were selected from stock held in a recirculating freshwater tanks and fed on pellets containing T or free of T for one month at 14°C.

Pellets containing T were prepared by spraying T ethanol solution, 25 mg/100 ml, on 1 kg commercial pellets for rainbow trout, followed by evaporation at room temperature overnight. Final content of T was 25 µg/g dry pellets. Fishes were fed with these pellets twice at a ration of 1.5% of body weight per day. The control group was fed with T-free pellets sprayed with only ethanol.

Des-Gly[10][D-Ala[6]] GnRH ethylamide (GnRHa; Sigma) and DA (Sigma) were dissolved in physiological saline (PS) consisting of 0.75% NaCl. Domperidone (DOM; Research Biochemicals International) was suspended in a vehicle consisting of 0.75% NaCl and 0.1% sodium metabisulphite.

The fish were rapidly anaesthetized in 2-phenoxyethanol (1 ml/2 l). All drugs were injected intraperitoneally. Blood samples of precocious and immature males were taken at $t=0$, 6 and 12 h post-injection, and at $t=0$, 6, 12 and 24 h post-injection, respectively, by puncturing the caudal vasculature using heparinized syringes. After centrifugation, the plasma was stored at -40°C until assayed for GTH II level. GTH II measurements were carried out by radioimmunoassay, developed by Kobayashi et al. (1987).

Data were analyzed for significance ($P<0.05$) using one-way ANOVA and Duncan's new multiple range test. In Experiments 1 and 2, the plasma GTH II levels

of control (T-untreated) and T-treated fish for each treatment during the same time period were tested for significance ($P<0.05$) using Student's *t* test.

Experimental design

Experiment I. In order to examine the influence of DA on GnRHa-stimulated GTH II secretion in T-untreated and T-treated precocious males, groups of 8 animals each were injected with PS, DA alone (10 and 100 µg/g body wt), GnRHa (0.1 µg/g body wt) alone, or a combination of GnRHa (0.1 µg/g body wt) and DA (10 and 100 µg/g body wt).

Experiment II. Effect of the DA D2 receptor antagonist DOM on GnRHa-stimulated GTH II secretion was invesigated in T-untreated and T-treated precocious males. Groups of 8 animals each were injected with PS plus vehicle, vehicle plus GnRHa (0.1 µg/g body wt), PS plus DOM (10 and 100 µg/g body wt), or a combination of GnRHa (0.1 µg/g body wt) and DOM (10 and 100 µg/g body wt).

Experiment III. In order to examine the influence of DOM on GnRHa-stimulated GTH II secretion in T-untreated and T-treated immature rainbow trout, groups of 10 animals each were injected with PS plus vehicle, vehicle plus GnRHa (0.1 µg/g body wt), PS plus DOM (10 and 100 µg/g body wt), or a combination of GnRHa (0.1 µg/g body wt) and DOM (10 and 100 µg/g body wt).

Results

Injection of the physiological saline (PS) or PS plus vehicle, did not influence the plasma GTH II levels.

Effects of dopamine (DA) on the plasma GTH II response to GnRHa in T-untreated and T-treated precocious males (EXP. I).

As demonstrated in Fig. 1A, plasma GTH II levels in T-untreated precocious males were about 1.5 ng/ml in each group of fish sampled at the time of injection. DA at 10 or 100 µg/g body wt did not significantly alter the plasma GTH II levels. In contrast, injection of GnRHa (0.1 µg/g body wt) caused significantly higher plasma GTH II levels than that of PS alone- and DA alone-injected fish at 6 and 12 h post-injection. In addition, a low dose DA (10 µg/g body wt) did not have an effect on GnRHa-induced GTH II levels at 6 and 12 h post-injection. However, a high dose DA (100 µg/g body wt) significantly decreased these levels more than the GnRHa-alone did at 6 h post-injection, but the GnRHa-induced GTH II levels were not significantly influenced by DA at this concentration at 12 h post-injection.

In the T-treated precocious males (Fig. 1B), plasma GTH II levels were about 3.2 ng/ml in each group of fish sampled at the time of injection. In fish injected with DA (10 and 100 µg/g body wt) alone the levels were not changed. On the other hand, those injected injected with GnRHa (0.1 µg/g body wt) alone had a
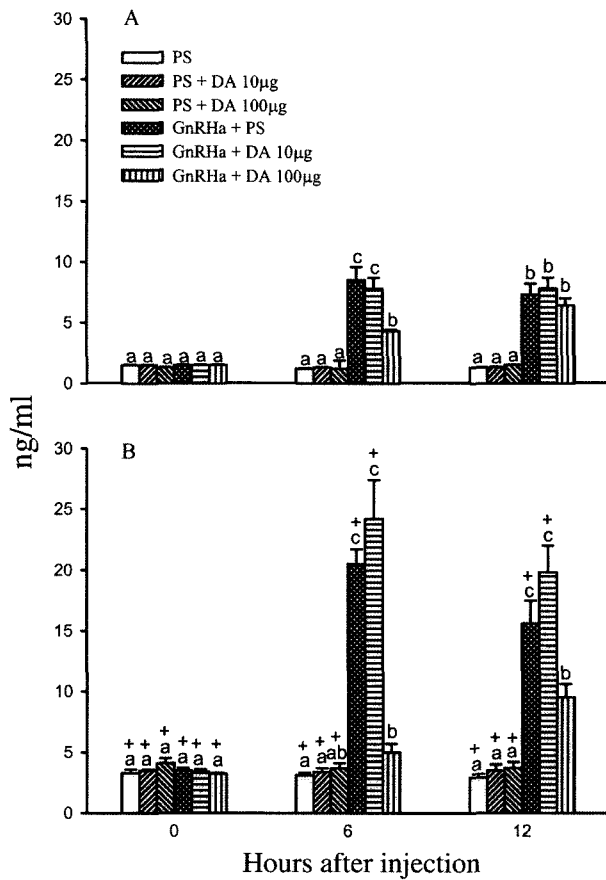
Fig. 1. Effects of GnRHa and DA on plasma GTH II levels in control (T-untreated; A) and T-treated (B) precocious males. Each group of 8 fish was given a single intraperitonial injection of physiological saline (PS), DA (10 or 100 µg/g body wt), GnRHa (0.1 µg/g body wt) or GnRHa (0.1 µg/g body wt) plus DA (10 and 100 µg/g body wt). Blood samples were taken at 0, 6 and 12 h post-injection. Results are expressed as mean ± SEM. A significant difference (p<0.05) was observed between columns indicated by different letters at each sampling time. + indicates significant differences from control (T-untreated) and T-treated precocious males at the same sampling time, p<0.05.

Fig. 2. Effects of GnRHa and domperidone (DOM) on plasma GTH II levels in control (untreated; A) and T-treated (B) precocious males. Each group of 8 fish was given a single intraperitonal injection of physiological saline (PS) plus vehicle (VEHI), PS plus DOM (10 and 100 µg/g body wt), GnRHa (0.1 µg/g body wt) plus VEHI or GnRHa (0.1 µ g/g body wt) plus DOM (10 and 100 µg/g body wt). Blood samples were taken at 0, 6 and 12 h post-injection. Results are expressed as mean ± SEM. A significant difference (p<0.05) was observed between columns indicated by different letters at each sampling time. + indicates significant differences from control (T-untreated) and T-treated precocious males at the same sampling time, p<0.05.

significantly higher plasma GTH II level than those injected with PS- or DA alone. Treatment of the T-treated fish with a high dose of DA (100 µg/g body wt) further decreased the GnRHa-induced GTH II levels at 6 and 12 h compared to the GTH II levels in T-untreated fish, but a low dose of DA (10 µg/g body wt) did not.

Effects of domperidone (DOM) and GnRHa on plasma GTH II secretion in T-untreated and T-treated precocious males (EXP. 2).

In the T-untreated precocious males (Fig. 2A), DOM at 10 or 100 µg/g body wt did not significantly alter the plasma GTH II levels at all sampling times. The GTH II levels elevated by GnRHa-alone injection were significantly maintained for up to 12 h post-injection. A high dose DOM (100 µg/g body wt) enhanced the GnRHa-induced GTH II levels at 6 h, but not at 12 h, post-injection. At a low dose of DOM, however, the levels did not significantly change compared to GnRHa alone.
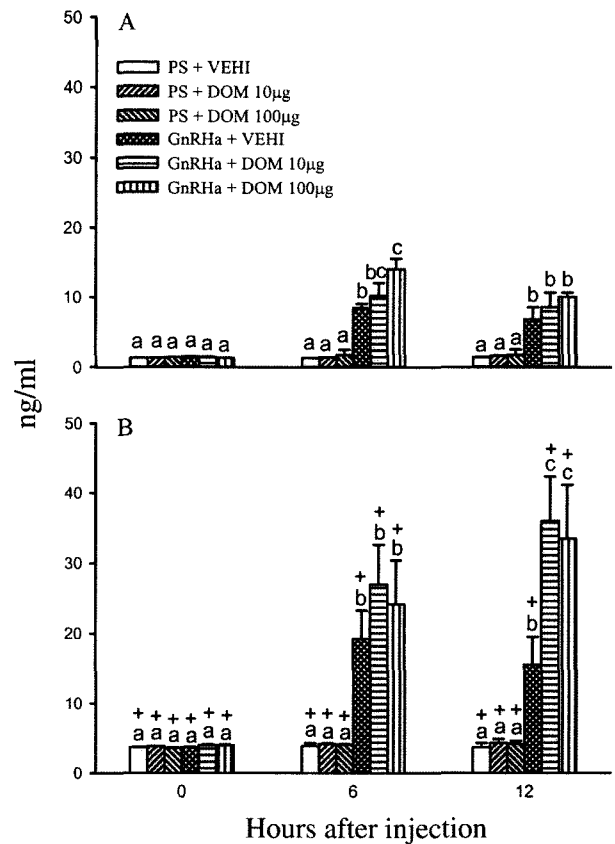
In the T-treated precocious males (Fig. 2B), DOM (10 and 100 µg/g body wt) alone did not influence the plasma GTH II levels. At 6 h post-injection, the levels were significantly influenced by the GnRHa alone, but GnRHa-induced GTH II levels were not influenced by DOM (10 and 100 µg/g body wt). At 12 h post-injection, the GnRHa-induced GTH II levels was equally and significantly increased by the both doses of DOM.

Effects of DOM and GnRHa on plasma GTH II secretion in T-untreated and T-treated immature males (EXP. 3).

In the T-treated immature rainbow trout (Fig. 3), plasma GTH II levels were about 0.9 ng/ml in each group of fish sampled at the injection time. DOM (10 and 100 µg/g body wt) alone did not influence plasma GTH II levels. In contrast, GnRHa alone increased and maintained the plasma GTH II levels for up to 12 h when compared with plasma GTH II levels induced by DOM alone or high dose (100 µg/g body wt) DOM plus
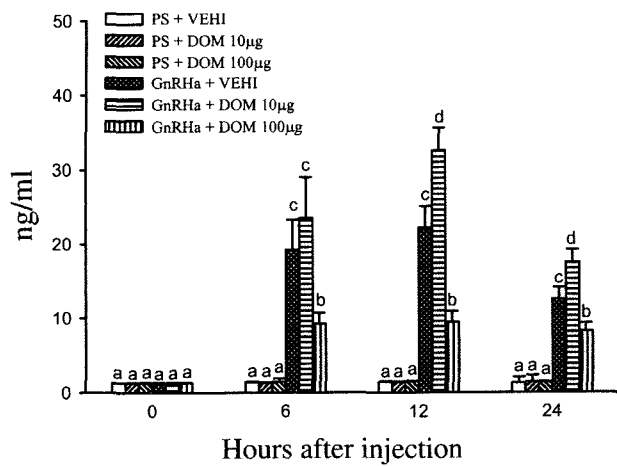
Fig. 3. Effects of GnRHa and domperidone (DOM) on plasma GTH II levels in T-treated immature rainbow trout. Each group of 10 fish was given a single intraperitonial injection of physiological saline (PS) plus vehicle (VEHI), PS plus DOM (10 and 100 μg/g body wt), GnRHa (0.1 μ g/g body wt) plus VEHI or GnRHa (0.1 μg/g body wt) plus DOM (10 and 100 μg/g body wt). Blood samples were taken at 0, 6, 12, 24 h post-injection. Results are expressed as mean ± SEM. A significant difference (p<0.05) was observed between columns indicated by different letters at each sampling time.

GnRHa. Fish receiving a combined injection of GnRHa plus low dose (10 μg/g body wt) DOM had higher plasma GTH II levels than those receiving GnRHa alone at 12 h post-injection. Although the plasma GTH II levels induced by high dose DOM plus GnRHa were significantly lower than those by GnRHa alone and by low dose DOM plus GnRHa for up to 12 and 24 h, they were significantly higher than those by PS or DOM alone for up to 24 h. The increase in plasma GTH II levels induced by GnRHa alone or a combination of a low dose DOM and GnRHa sharply decreased from 12 h post-injection. However, when induced by GnRHa plus high dose DOM, the levels were constantly maintained at 24 h post-injection.

The plasma GTH II levels in T-untreated immature rainbow trout were about 0.9 ng/ml in each group of fish sampled at the injection time. However, the plasma GTH II levels were not significantly influenced by GnRHa (0.1 μg/g body wt), DOM (10 and 100 μg/g body wt) or a combination of GnRHa (0.1 μg/g body wt) and DOM (10 and 100 μg/g body wt) through out the duration of sampling (data not shown).

Discussion

The present study demonstrates that T enhanced GTH II secretion by potentiating the GTH II response to GnRHa, affected basal GTH II secretion, and increased dopaminergic inhibitory system in precocious males. In contrast, T did not affect basal GTH II secretion in immature rainbow trout, but clearly enhanced GnRHa-induced GTH II secretion as well as the GTH II response to the DA D2 receptor antagonist.

Plasma GTH II levels were increased by T treatment

in precocious males, but not in immature rainbow trout in this investigation. Gielen and Goos (1984) reported that GTH II secretion was not stimulated by T administration, although T stimulated GTH II accumulation in the pituitary in immature rainbow trout. However, a long-term T treatment at high doses increased plasma GTH II levels, and this is also reflected by the enhanced pituitary responsiveness to GnRH in immature rainbow trout (Crim and Evans, 1983). Therefore, an increase in plasma GTH II levels in this study is possibly due to a higher T level in the serum of precocious male which induced GnRH synthesis in the brain as a result of steroid positive feedback.

Most of the evidence concerning the dopaminergic inhibition of GTH II secretion comes from studies in goldfish and other cyprinid species (reviewed by Peter et al., 1986, 1991) and also in African catfish (De Leeuw et al., 1985, 1987). In the goldfish, DA or DA agonists can abolish the pituitary's response to GnRHa as can lesioning of dopaminergic tracts in the pituitary stalk and preoptic nucleus (Chang and Peter, 1983). Conversely, DA antagonists such as pimozide (D1 and D2 receptor antagonist) and DOM can increase GTH II secretion or if administered in combination with GnRHa, can markedly potentiate the response to GnRHa (reviewed by Peter et al., 1986, 1991). In the present study, DA- and DOM-alone did not influence the basal secretion of GTH II, but caused a marked potentiation of the GTH II secretion stimulated by GnRHa in T-treated and T-untreated precocious males and in T-treated immature fish. In addition, DA at a lower concentration (10 μg/g body wt), did not influence the effect of GnRHa. At a higher dose (100 μg/g body wt) however, this drug inhibited the GnRHa-induced GTH II secretion. Apparently, only at a higher dose can exogenous DA overrule the inhibitory effect of the endogenous DA. The effects of DA in salmonids, however, were much less pronounced than in the goldfish, and salmonids were capable of ovulation in response to GnRHa alone (Billard et al., 1984; Van der Kraak et al., 1986). Omeljaniuk (1995) reported that an outstanding difference between the goldfish and rainbow trout was that the pituitaries of rainbow trout consistently had significantly smaller numbers of DA receptor sites than goldfish pituitaries. In the previous study, furthermore, we demonstrated that DA directly inhibited the sGnRH-induced release of GTH II, and that inhibition by DA of the release of GTH II was probably mediated by D2-like receptors in dispersed pituitary cells of T-treated immature rainbow trout (Kim et al., 1995). Therefore, a stronger DA inhibitory tone in goldfish compared with rainbow trout could account for this variation, although difference in pituitary sensitivity to GnRHa could also play a role. In contrast, there is no evidence for DA inhibitory regulation on GTH II secretion in the Atlantic croaker, *Micropogonias undulatus*: In a series of *in vivo* experiments, effects of a number of DA agonist and antagonist drugs on GTH

II secretion were tested, and no evidence of inhibition or potentiation, was found (Copeland and Thomas, 1989). It appears, therefore, that dopaminergic inhibition of GTH II secretion may not be a universal phenomenon in the teleost fish.

The most interesting finding in the present study was that the effects of DA neuroleptics on GnRHa-induced GTH II secretion were prolonged in T-treated fish compared with T-untreated fish in precocious males. De Leeuw et al. (1987) have proposed a model to explain the relationship between steroid feedback and dopaminergic inhibition of GTH II in the African catfish, whereby aromatizable androgen and estrogens are converted to catecholestrogens which are metabolized preferentially by catechol o-methyl transferase, thus allowing the dopamine level to increase. In this way, increased circulating steroid levels would indirectly inhibit GTH II secretion. Trudeau et al. (1993) showed that implants of T and 17β-estradiol (E_2) significantly potentiated both plasma GTH II responses to sGnRHa and DOM elevation of plasma GTH II levels in sexually regressed female goldfish. Dufour et al. (1984) also found an increase in plasma GTH II levels following a series of injections of GnRHa plus pimozide, but not when any of the drugs were injected alone in E_2-pretreated sexually immature eels. Furthermore, gonadal steroids are also involved in the regulation of synthesis in GTH and GnRH (Amano et al., 1994). These results suggest that exogenous T treatment may regulate GTH II secretion by potentiating the responsiveness of gonadotrophes to GnRH receptors as well as DA receptors.

In this investigation, the injection of a combination of GnRHa plus lower dose DOM was more effective in increasing GTH II levels in T-treated immature rainbow trout, although no dose dependence was found in precocious males. Omeljaniuk et al. (1989) reported that injection of goldfish with DOM caused an increase in GnRH receptor capacity in the goldfish pituitary 24 h later. De Leeuw et al. (1989) demonstrated the dose- and time-dependency of this up-regulation. These results suggest that the GnRH receptor capacity of the rainbow trout pituitary could also be influenced by DOM, indicating that it is due to direct effects on the gonadotrophs. Furthermore, another possible explanation for these dose-related differences might be attributable to differences in down-regulation of the GnRH receptors according to DOM concentration in T-treated rainbow trout, although the physiological significance of this difference is not clear.

In summary, the present study has provided evidence that GnRH-induced GTH II levels were enhanced by DOM and depressed by DA in rainbow trout. Furthermore, the effects of DA and DOM on GnRH-induced GTH II secretion was prolonged by exogenous T treatment. Whether DA receptor modulation by steroids exists in rainbow trout is not clear and DA modulation of GTH II secretion by steroids still awaits

further investigation.

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