The Effect of 17β-Estradiol on Growth and Survival of Sword Tail (Xiphophorus helleri)

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Abstract: Effect of dietary 17β-estradiol supplementation was investigated on growth and survival of sword tail (Xiphophorus helleri). Fish (1.27±0.23 g) were offered control diet (without 17β-estradiol supplementation) and control diet supplemented with 50, 25 and 10 mg/kg 17β-estradiol, over a 60-day period. Fish were fed based on 3% of total biomass at the early and 2% at the late experiment period. Results showed that there were no significant differences in growth performance and survival rate between different treatments (P > 0.05).

Key words: 17β-Estradiol · Growth · Survival · Sword tail Fish

INTRODUCTION

The role of sex steroids as inducers of sexual development in fish was first proposed by Yamamoto [1]. His decisive research led to many experiments to better understand the role of steroids on fish sexual differentiation, much of these studies have been economic and aquaculture-based, as attributes such as growth, appearance and timing of maturity are often more pleasing in one sex than the other [2].

17β-estradiol (E2) is the natural estrogen has been shown to be a useful feminization hormone in some fish [3], it is synthesized by the follicular layer of the oocyte, with both thecai and granulosa cells being concerned in steroidogenesis [4-6]. In fact, E2 produced by the follicle cells in the ovary, persuade VTG gene copy and translation by liver cells [7].

17β-estradiol (E2) plays a principle role in female fish, promoting gonadal growth and development [8]. In oviparous vertebrates, E2 acts on the liver by the estrogen receptor (ER) to induce production of vitellogenins (Vgs), yolk pioneer proteins found mainly in mature females. Interactions among the mechanisms regulating growth and reproduction in mammals are well studied but fewer are well characterized in fish [9]. However, the presence of GH/IGF-I axis components in both testis and ovary of some fish species, as well as receptors for gonadal steroids in somatic tissues indicate interaction between the regulatory mechanisms that govern reproduction and somatic growth in fishes [10-12]. The attendance of estrogen and estrogen-like compounds in the environment has become a subject of great interest in recent years. A lot of the research centers studied the effects of these compounds on animal development and reproduction. By contrast, few studies have been undertaken to explain how endocrine disruptors influence on growth or its regulation [13].

It has been reported by some researchers that the effects of sex hormones on fish might be different based on the fish age (application time), amount of hormone and fish species [14-16].

Some previous studies Schreck and Fowler [17] and Degani [14] reported that the direct feminization via estrogens caused some raise in fish size. Some other researchers reported conflicting findings [18, 19].

Degani [14] reported that direct feminization by estrogens caused some increase in fish size and 17β-estradiol improved the growth in ells (Anguilla anguilla). Güzel et al. [20] found that estriadiol valerate increased the condition factor, had no effects on the growth and FCR, but decreased the survival rate. Johnstone et al. [21] concluded that 17β-estradiol had no significant effect on rainbow trout and Atlantic salmon (Salmo salar) growth. Goryczko et al. [18] reported that the application of 17β-estradiol delayed rainbow trout growth. Arsenault et al. [22], Johnstone et al. [23] and Komen et al. [24] concluded that E2 had negative effects on parr-smolt Atlantic salmon (Salmo salar L.), brook trout (Salvelinus fontinalis) and carps (Cyprinus carpio) growth performance.

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The present study was conducted to investigate the effect of dietary 17β-estradiol supplementation on growth performance and survival in sword tail (Xiphophorus helleri).

MATERIALS AND METHODS

This study was conducted in 2011 by using 12 glass aquaria (60×40×30 cm) ready with single central air pump. Water temperature was maintained at 27-29 °C using under water heaters. A total of 72 fry sword tail fish (1.27±0.23 g) were randomly distributed into the aquaria. Fish were fed on commercial trout pellet (Biomare, France, 0.5 mm in diameter) over a 14-day period for adaptation. Thereafter, the aquaria assigned as 4 triplicate treatments. One of the treatments received Biomare diet (control group), whereas the remaining were received Biomare supplemented with 50, 25 and 10 mg/kg 17β-estradiol. To achieve the 17β-estradiol-supplemented diets, E2 was dissolved in absolute ethanol and sprayed onto Biomare and then pellets were dried over night. Fish were fed based on 3 % of body weight in the first month and 2 % at the second month. The fish were fed 2 times daily. Fish were biometry at the trial initiation as well as fortnightly thereafter and the food amount was adjusted accordingly. Dissolved oxygen, total hardness and pH were 7.8, 270±0.2 mg/l and 7.5±0.3.

At the end of the trial, growth performance was evaluated by calculating weight gain (WG), weight gain percentage (WG %), food conversion ratio (FCR) and specific growth rate (SGR) as follow:

$$WG = W_f - W_i$$

$$WG(\%) = \frac{W_f - W_i}{W_i} \times 100$$

$$FCR = \frac{CF}{WG}$$

$$SGR = 100 \times \frac{\ln W_f - \ln W_i}{T}$$

Where $W_i$ was the initial weight, $W_f$ was final weight, $CF$ was total consumed food and $T$ was the experiment duration.

Survival rate was also calculated by counting the number of remaining fish divided to the initial fish number multiplied to 100.

Data were analyzed using statistical software SPSS v. 18. Data were subjected to one way ANOVA and Duncan's test to find significant effect of diets on growth and survival. $P < 0.05$ considered to be significantly different. Data are presented as treatments mean ± SD.

RESULTS

Table 1 shows the growth performance and survival of different treatments after 60 day. There were no significant differences in growth performance and survival rate between the treatments ($P > 0.05$). Growth pattern of different groups is presented in figure 1.

DISCUSSION

In the present study the obtained results showed no significant differences in growth performance and survival rate between treatments ($P > 0.05$) but growth factors is better in treatments that received 17β-estradiol than control group.

As seen from the present study, several other researchers such as Güzel et al. [20], Johnstone et al. [21] and Parks and Parks [25] in Rainbow Trout (Oncorhynchus mykiss), Atlantic salmon (Salmo salar) and Brook Trout (Salvelinus fontinalis) reported that using of estradiol had no effects on the weight increase. Johnstone et al. [21, 23] reported that the growth decreased during the application, but there were no significant growth variations after the use in time, therefore, the application had no effect on growth.

Some other researchers stated that estradiol delayed the fish growth. Górczyko et al. [18] reported that the use of 17β-estradiol (5 and 20 mg kg$^{-1}$) for 120 days retarded the rainbow trout growth. Komen et al. [24] applied 25, 75 and 125 ppm doses of 17β-estradiol to carps (Cyprinus carpio) and reported negative impacts of the applications on carp's growth. Hendry et al. [26] noted physical deformity and decreased growth in Atlantic halibut (Hippoglossus hippoglossus), fed E2 at 10 mg/kg for

<table>
<thead>
<tr>
<th>17β-estradiol content (mg/kg)</th>
<th>WG</th>
<th>WG(%)</th>
<th>SGR</th>
<th>FCR</th>
<th>Survival(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>0.89 ± 0.13</td>
<td>67.83 ± 14.52</td>
<td>0.86 ± 0.14</td>
<td>6.18 ± 1.57</td>
<td>90 ± 14.14</td>
</tr>
<tr>
<td>10</td>
<td>0.94 ± 0.09</td>
<td>78.54 ± 9.29</td>
<td>0.96 ± 0.09</td>
<td>5.19 ± 0.21</td>
<td>100 ± 0.00</td>
</tr>
<tr>
<td>25</td>
<td>0.96 ± 0.21</td>
<td>82.52 ± 17.48</td>
<td>1.00 ± 0.16</td>
<td>5.86 ± 1.89</td>
<td>90 ± 14.14</td>
</tr>
<tr>
<td>50</td>
<td>1.06 ± 0.16</td>
<td>77.70 ± 9.07</td>
<td>0.96 ± 0.08</td>
<td>4.98 ± 0.38</td>
<td>90 ± 14.14</td>
</tr>
</tbody>
</table>
45 days all through the range of doses administered, it became obvious that the negative effects of E2 on feeding behavior and following growth increased with dose.

Davis et al. [27] obviously indicated that, simultaneous with Vtg induction, E2 suppresses the Gh/IgfI axis. These results completely support this hypothesis that E2 represses somatic growth while promoting vitellogenesis during gonadal recrudescence in females.

The growth and plasma IGF-I concentrations in Arsenault et al. [22] trial provide insight into the mechanisms by which E2 and 4-NP influence the growth of Atlantic salmon smolt. These results recommended that their mechanisms of action involve a disruption of the GH/IGF-I axis and that their pathways leading to its disruption may be diverse from each other. Studies have shown that E2 is able of affecting the thyroid axis [28-30] and Ikuta et al. [31] suggested that E2 could potentially interfere with the cross-talk between the thyroid and GH/IGF-I axis. E2 could potentially affect the hypothalamus, causing inhibition of GH and/or TSH secretion from the pituitary. Miwa and Inui [32] indicated that E2 has a suppressive action on pituitary somatotrophic function.

Some others reported significant weight increase in fish by the estradiol use [14, 17]. Güzel et al. [20] concluded that the condition factor of E2 applied group was significantly (P<0.05) higher than that of the control group. This result was in agreement with some other studies working with 17β-estradiol or 17α- methyltestosterone [33-37]. Degani [14] applied 17β-estradiol (50 mg kg⁻¹) for 75 days to eels (Anguilla anguilla) and reported that the use of 17 β-estradiol improved the fish growth. Hiroaki et al. [38] showed 17β-estradiol significantly increased the growth rate of Japanese eel (Anguilla japonica) treated with 25 and 50 mg/kg E2 diet at the early juvenile step, the amount of protein in muscle decreased and that of fat increased in the E2-treated groups except in the early juvenile step fed with 25 mg/kg E2.

Some researchers reported that estradiol use did not influence the survival rate [39, 17] but in several other studies reported that E2 application had negative effect on fish survival rate [18, 20, 23, 25, 40].

The difference between the growth, FCR and survival rate findings of the present study and the other studies could be explained by the differences in application time, dose and rate, as well as fish species of the mentioned studies [14, 15, 20, 41].

At the end of this study, we concluded that the use of 17 β-estradiol, applied with feed by the rate of 10, 25, 50 mg kg⁻¹ for 60 days had no significant effects on growth performance and survival rate. Therefore, it is not important to recommend this kind of estradiol application for sword tail (Xiphophorus helleri) because of its insignificant effect on growth and survival rate.

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