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Effects of dietary chenodeoxycholic acid on growth performance, body composition and related gene expression in large yellow croaker (*Larimichthys crocea*) fed diets with high replacement of fish oil with soybean oil

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ABSTRACT

An experiment was conducted to determine the effects of dietary chenodeoxycholic acid (CDCA) on the growth performance, body composition and lipid deposition of juvenile large yellow croaker (*Larimichthys crocea*) (initial weight, 10.03 ± 0.02 g) fed a soybean oil diet. The practical diet with 6% soybean oil (SO) was used as the control, and a fish oil (FO) diet was designed with 6% fish oil. On the basis of the soybean oil diet, 300 mg and 900 mg/kg (CDCA) were added, respectively. After 10-week growth trial, results showed that survival rate (SR) and condition factor (CF) were not significantly different among dietary treatments. Compared with the FO group, final weight (FW) and weight gain rates (WGR) in the SO group significantly decreased while the groups with the supplementation of CDCA show significantly better growth performance than the SO group and FO group. The lipid content of liver was significantly increased by the replacement of FO with SO but decreased with the increase of the supplementation of CDCA. The activity of lipoprotein lipase was significantly higher in fish fed the diet with 900 mg/kg CDCA supplementation. The expression of PPARα decreased while the expression of SREBP-1 increased significantly in fish fed the SO diet compared with fish fed the FO diet. Meanwhile, dietary 900 mg/kg CDCA significantly upregulated the expression of PPARα and FXR while the expression of SREBP-1 was decreased by the supplementation of CDCA. The results suggested that the supplementation of CDCA could improve the growth performance and lipid deposition of liver in large yellow croaker which were negatively affected by the replacement of dietary fish oil with soybean oil.

1. Introduction

Continually developing of aquafeed industry increased the demand for fish oil and has caused great pressure on the fishery resources (Barlow, 2000; Leaver et al., 2008). Soybean oil is the world's largest source of vegetable oil (Figueiredo Silva et al., 2005) and has been looked upon as a candidate to replace fish oil for relatively considerable output and acceptable price (Bell et al., 2005; Tan et al., 2016). However, high dietary soybean oil, mainly due to the high content of linoleic acid (18:2n-6) and the lack of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) (Nutrition, 1993), reduced the growth and survival and increased the lipid accumulation in some marine fish (Montero et al., 2008; Fountoulaki et al., 2009; Wang et al., 2011; Peng et al., 2014). There needs to be more researches to explore viable methods that can enhance growth or improve metabolism to reverse the negative effects of the replacement of fish oil.

As non-nutrient additive, bile acids could enhance growth, improve metabolism, prevent diseases and help digestion (Deshimaru et al., 1982; Pullen and Polin, 1984; Maita et al., 1996). Bile acids have different molecular forms that are produced by liver but the different structures cause varied function (Hofmann et al., 2009), then it may be hard to investigate the effects and mechanism of bile acids. As one of main bile acids, chenodeoxycholic acid (CDCA) is synthesized in liver (Russell, 2003) and has been shown to be the most potent natural agonist that can activate farnesoid X receptor (FXR) (Parks et al., 1999). As a nuclear receptor, FXR could regulate numbers of genes expression involved in lipid metabolism (Zhang and Edwards, 2008). However, the lipid metabolism in fish remains to be elucidated, but it is always an interaction of fatty acid transport, synthesis and oxidation which is similar to mammals (Tocher, 2003). Lipoprotein lipase (LPL) and hepatic

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lipase (HL) play important role in hydrolyzing triglycerides in lipoproteins (Oku et al., 2006) and are required for the process of transferring free fatty acids into cell (Ma et al., 1994; Rinninger et al., 1998; Mead et al., 2002). Sterol regulatory element binding protein 1 (SREBP-1) is one part of the bHLH-Zip transcription factor family and a major metabolism regulator. Multiple genes related to fatty acid synthesis have been confirmed in mammals that could be upregulated by SREBP-1 (Matsuzaka et al., 2002; Yan et al., 2009; Dong et al., 2015). In addition, peroxisome proliferator activated receptor alpha (PPARα) has been confirmed to be important in reducing lipid accumulation. The activation of PPARα upregulated genes related to the transportation, binding, activation and β-oxidation of fatty acids (Escher et al., 2001; Matsuzaka et al., 2002; Mandard et al., 2004; Kersten, 2014). Meanwhile, recent advances in Nile tilapia (*Oreochromis niloticus*) suggested the molecular mechanisms of PPARα in fish were similar to mammals (Ning et al., 2016). Based on these observations, it was assumed that FXR, LPL, HL, SREBP-1 and PPARα may play import role in regulating the lipid metabolism of large yellow croaker. However, up to now, there are no reports of relevant studies on the influence of CDCA supplementation on the growth and lipid metabolism in fish.

Large yellow croaker (*Larimichthys crocea*) is an economically important species with delicious taste and high economic value. In recent years, the culture scale has been expanded rapidly in China but the growth retardation, increase of lipid deposition and decrease of immunity were observed in large yellow croaker fed a vegetable oil diet (Wang et al., 2011; Tan et al., 2016). The objective of the present study was to explore the effects and mechanism of the supplementation of CDCA on the growth performance and lipid deposition in large yellow croaker fed the diet with replacement of fish oil with soybean oil.

2. Materials and methods

2.1. Experiment diets

The practical diets contained 45% protein and 12% lipid were formulated to be appropriate for large yellow croaker (Yi et al., 2014). The fish oil diet (FO group) was designed with 6% fish oil while 6% soybean oil was used to replace the fish oil in the soybean oil diet (SO group). Based on the soybean oil diet, 300 mg/kg (SOL group, soybean oil with low CDCA supplementation) and 900 mg/kg (SOH group, soybean oil with high CDCA supplementation) CDCA were added into the basal diet, respectively (Table 1). All ingredients were smashed and filtrated through 80 mesh. The dry ingredients were mixed with oil and then appropriate amount of water was added. At last, pellets were produced and dried in an oven at 40 °C over a 12 h period, and stored at − 20 °C until used.

2.2. Feeding trial and sample collection

Same batch of juvenile large yellow croaker was obtained from Xiangshan Harbor Nusery Co., Ltd. (Ningbo, China). Prior to the experiment, commercial diet was used to feed the fish in floating sea cages for two weeks. Sixty fish (mean weight 10.03 ± 0.02 g) were distributed into each floating cage (1 m × 1 m × 1.5 m) after fasting for 24 h. Each diet was randomly distributed in triplicate (three replicates per treatment) and fed to fish two times a day at 05:00 and 17:00 until apparent satiation for ten weeks with appropriate conditions (temperature: 24.3–28.5 °C; salinity: 26.5–30.8‰; dissolved oxygen: 6.5–7.4 mg L^{-1}).

At the termination of feeding trial, proper concentrations of eugenol (1:10,000) were used to anesthetize the fish after fasting for 24 h. For each cage, total fish were taken for measuring the number and body weight and three fish were collected randomly and stored at − 20 °C for future analysis of biochemical. The body weight, length and liver weight were measured for analysis of hepatosomatic index (HSI) and Condition factor (CF). For enzyme activity and molecular analyses, the liver from twelve fish per cage were put to the liquid nitrogen immediately and stored at − 80 °C before analysis.

Table 1
Formulation and proximate analysis of the experimental diet for large yellow croaker larvae(% dry matter).

	Diets			
Ingredients %	FO	SO	SOL	SOH
Fish meal[a]	39	39	39	39
Soybean meal[b]	20	20	20	20
Wheat meal[c]	23	23	23	23
Wheat starch	6	6	6	6
Fish oil	6	0	0	0
Soybean oil	0	6	6	6
Soybean lecithin	1.5	1.5	1.5	1.5
Vitmal premix[d]	2	2	2	2
Mineral premix[e]	2	2	2	2
Attractant[f]	0.3	0.3	0.3	0.3
Mold inhibitor[g]	0.1	0.1	0.1	0.1
Chenodeoxycholic acid[h]			0.03	0.09
Proximate composition				
Crude protein	45.2	46.1	45.7	45.6
Crude lipid	11.5	11.4	10.8	11.1

[a] Fish meal: crude protein 74.4% dry matter, crude lipid 8.5% dry matter; Qingdao Great Seven Bio-Tech., China.
[b] Soybean meal: crude protein 54.3% dry matter, crude lipid 2.0% dry matter; Qingdao Great Seven Bio-Tech., China.
[c] Wheat meal crude protein 17.2% dry matter, crude lipid 2.5% dry matter; Qingdao Great Seven Bio-Tech., China.
[d] Vitamin premix (mg or g/kg diet): cholecalciferol, 5 mg; retinol acetate, 32 mg; thiamin 25 mg; vitamin B12 (1%), 10 mg; riboflavin, 45 mg; pyridoxine HCl, 20 mg; ascorbic acid, 2000 mg; alpha-tocopherol (50%), 240 mg; vitamin K3, 10 mg; pantothenic acid, 60 mg; inositol, 800 mg; niacin acid, 200 mg; folic acid, 20 mg; biotin (2%), 60 mg; choline chloride (50%), 4000 mg; microcrystalline cellulose, 12.47 g.
[e] Mineral premix (mg or g/kg diet): $CuSO_4{\cdot}5H_2O$, 10 mg; Ca $(IO_3)_2{\cdot}6H_2O$ (1%), 60 mg; $CoCl_2{\cdot}6H_2O$ (1%), 50 mg; $FeSO_4{\cdot}H_2O$, 80 mg; $MgSO_4{\cdot}7H_2O$, 1200 mg; $MnSO_4{\cdot}H_2O$, 45 mg; $NaSeSO_3{\cdot}5H_2O$ (1%), 20 mg; $ZnSO_4{\cdot}H_2O$, 50 mg; $CaH_2PO_4{\cdot}H_2O$, 10 g; Zeolite, 8.485 g.
[f] Attractants: glycine and betaine.
[g] Mold inhibitor: contained 50% calcium propionic acid and 50% fumaric acid.
[h] Chenodeoxycholic acid: ≥ 97%, sigma.

2.3. Chemical analysis

The samples dried constantly at 105 °C were prepared to analyze the biochemical composition in the reference of the Association of Official Analytical Chemists (AOAC, 1995). Kjeldahl method (N × 6.25) was used to measure and estimate the crude protein. Soxhlet extractor with petroleum ether was used to determine the crude lipid. The lipid content of the tissues were determined by using the Folch method (Yan et al., 2015) after the samples were freeze-dried.

2.4. Activity of LPL and HL in liver

Approximate 0.2 g of liver from each sample was mixed with nine times sample weight of phosphate buffered saline (Solarbio, China) and homogenized using a homogenizer (Kinematica, Switzerland) in ice water bath. After centrifuging at 4000g for ten minutes at 4 °C, the supernatant fraction was collected and stored at − 20 °C until use. A commercial kit (Nanjing Jiancheng Bioengineering Institute, China) was used to determine the activity of LPL and HL according to instructions.

2.5. RNA extraction and real-time quantitative PCR

Total RNA was isolated from liver according to the instruction of Trizol Reagent (Takara, Japan). The concentration and quality of total RNA were determined using a Nanodrop® 2000 (Thermo Fisher Scientific, USA) and agarose gel electrophoresis was used to test the integrity. Total RNA was reverse transcribed to cDNA using a kit from

Table 2
Primer pair sequences for real-time PCR.

Target genes	Forward (5′–3′)	Reverse (5′–3′)	References
SREBP-1	CCAAGACAGAGGAGTGCGAGAC	TCATTTGCTGGCAGTCGTGGAG	GenBank accession no. KP342262
PPARα	GTCAAGCAGATCCACGAAGCC	TGGTCTTTCCAGTGAGTATGAGCC	Zuo et al. (2013)
LPL	GAATTCAACGCGGAAACACAG	ACGCTCATAGAGGGCAGACAC	Yan et al. (2015)
HL	TCCGTCCATCTATTCATTGACTCTC	GCCACTGTGAACCTTCTTGATATTG	Cai et al. (2015)
FXR	TGGAGGAAAGGATACGCAAGAGTG	TGTCAGGATGGTTACGGTGGTG	GenBank accession no. XM_010737684.2
β-Actin	CTACGAGGGTTATGCCCTGCC	TGAAGGAGTAACCGCGCTCTGT	Yan et al. (2015)

SREBP-1, sterol-regulatory element binding protein-1; PPARα, peroxisome proliferators-activated receptor α; LPL, lipoprotein lipase; HL, hepatic lipase; FXR, farnesoid X receptor.

Transgen Biotech (Beijing, China) and diluting it with RNAase-free water to 100 ng/μL. The reaction of Real-time quantitative PCR was performed in a total volume of 20 μL (each primer: 0.4 μL; cDNA: 1 μL; 2 × qPCR SuperMix: 10 μL; water: 8.2 μL). The real-timePCR was programmed as follows: 95 °C for 2 min, followed by 40 cycles of 95 °C for 10 s, 58 °C for 15 s,72 °C for 10 s and melting curve analysis was performed to confirm the specificity of production. The primers sequence of β-actin, FXR, SERBP-1, PPARα, HL and LPL are listed in Table 2. In addition, a standard curve was obtained and used to calculate the amplification efficiency. The mRNA levels of gene were calculated and normalized via the delta-delta method (Livak and Schmittgen, 2001).

2.6. Calculations and statistical analysis

Survival rate ($SR\%$) = 100 × FN/IN
Weight gain rate (WGR%) = $100 \times (w_t - w_0)/w_0$
Feed intake (%/d) = $100 \times D_d/((w_t + w_0)/2)/t$
Hepatosomatic index(HSI,%) = 100 × liver weight (g)/body weight (g)
Condition factor (CF) = body weight (g)/body length $(cm)^3$.

where FN is the final number and IN is the initial number of fish in each cage. w_t is the final weight, w_0 is the initial weight of fish and D_d is the dry weight of diet in each cage and t is the experiment period.

SPSS 20.0 was used to perform the statistical analysis. Results were means ± S.E.M from at least three fish per cage. The dates were analyzed using one-way ANOVA and followed by Tukey's Test. The level of significance was set at $P < 0.05$.

3. Results

3.1. Survival rate and growth performance

The SR, FI and CF of large yellow croaker were independent of the dietary treatments ($P > 0.05$) (Table 3). The replacement of FO with SO significantly decreased the FW and WGR ($P < 0.05$) which were significantly higher in fish fed the diet with CDCA supplementation compared with fish fed soybean oil or fish oil ($P < 0.05$), while no differences were observed between the low CDCA supplementation and high CDCA supplementation group ($P > 0.05$) (Table 3).

3.2. Body composition

The crude protein and whole-body lipid content were not affected significantly by the dietary treatments ($P > 0.05$) (Table 4). Fish fed the SO diet had significantly higher liver lipid content than the fish fed the FO diet. The liver lipid content in the CDCA groups were significantly lower than the SO group but still higher than FO group ($P < 0.05$) (Table 4). Similarity, fish in the SO group showed significantly higher HSI compared with the FO group and high CDCA supplementation group ($P < 0.05$) (Table 4). Moreover, no significant differences of the muscle lipid content were observed ($P > 0.05$) (Table 4).

3.3. Activity of LPL and HL in liver

The high CDCA supplementation group significantly increased the activity of LPL compared with other group ($P < 0.05$) and no differences were observed between the SO and FO group ($P > 0.05$) (Fig. 1). However, no significant differences of the activity of HL were observed among all groups ($P > 0.05$) (Fig. 1).

3.4. Expression of genes related to lipid metabolism

The mRNA levels of FXR were significantly higher in the CDCA supplementation groups than the FO and SO group ($P < 0.05$) (Fig. 2a). The highest mRNA levels of SREBP-1 were observed in the fish fed the SO diet ($P < 0.05$) (Fig. 2b). The mRNA levels of PPARα were significantly decreased by the replacement of FO with SO, but increased by the supplementation of 900 mg/kg CDCA ($P < 0.05$) (Fig. 2c). The low CDCA supplementation group markedly increased the LPL expression compared with the SO group ($P < 0.05$) (Fig. 2d). The highest HL expression was observed in the fish fed the SO diet and the CDCA supplementation tended to decrease the HL expression even though the effects were not significant ($P > 0.05$) (Fig. 2e).

4. Discussion

Results of present study showed that the growth performance of large yellow croaker was significantly decreased when fish oil in diets was totally replaced by soybean oil. Previous studies have shown that

Table 3
Growth response and survival of large yellow croaker.

	Diets			
	FO	SO	SOL	SOH
Initial body weight (g)	10.03 ± 0.02	10.02 ± 0.01	10.03 ± 0.01	10.01 ± 0.03
Final body weight (g)	53.59 ± 0.82^{b}	50.28 ± 0.92^{c}	56.99 ± 0.15^{a}	54.51 ± 0.86^{b}
Weight gain rate (%)	439.82 ± 5.10^{b}	401.94 ± 9.20^{c}	468.37 ± 0.84^{a}	444.72 ± 8.89^{ab}
Feed intake (%/d)	2.25 ± 0.10	2.28 ± 0.05	2.22 ± 0.01	2.50 ± 0.11
Condition factor (g/cm^3)	1.66 ± 0.07	1.66 ± 0.01	1.68 ± 0.03	1.61 ± 0.05
Survival rate (%)	91.48 ± 3.65	86.11 ± 3.64	91.67 ± 3.33	91.11 ± 4.84

Mean values ± S.E.M ($n = 3$) within a row with a common superscript letter are not significantly different from other dietary groups ($P > 0.05$).

Table 4
Proximate composition (wet weight %) in whole body and lipid content of the liver and muscle in large yellow croaker.

	Diets			
	FO	SO	SOL	SOH
Crude protein (%)	15.60 ± 0.21	15.36 ± 0.14	15.68 ± 0.082	15.67 ± 0.11
Crude lipid (%)	8.63 ± 0.39	9.68 ± 0.46	9.76 ± 0.11	9.81 ± 0.31
Ash (%)	3.35 ± 0.12	3.26 ± 0.04	3.18 ± 0.12	3.43 ± 0.01
Hepatosomatic index (%)	1.20 ± 0.07[c]	1.86 ± 0.03[a]	1.71 ± 0.11[ab]	1.44 ± 0.03[b]
Liver lipid content (%)	14.27 ± 0.69[c]	21.73 ± 1.14[a]	18.98 ± 0.72[ab]	17.09 ± 0.78[b]
Muscle lipid content (%)	8.94 ± 1.40	9.23 ± 1.20	7.96 ± 0.97	10.47 ± 1.36

Mean values ± S.E.M (n = 3) within a row with a common superscript letter are not significantly different from other dietary groups (P > 0.05).

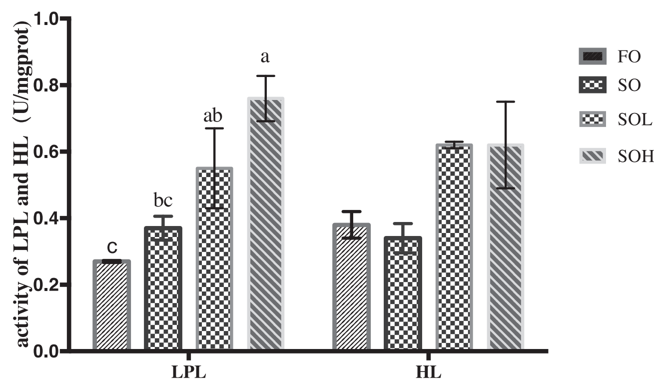

Fig. 1. Effects of dietary soybean oil and CDCA on activity of lipase in larval large yellow croaker. Data were presented as means ± S.E.M. (n = 3). The enzyme activity of lipase was expressed as specific activity (U/mg protein). Bars bearing the same letters are not significantly different (P > 0.05).

high amount of dietary soybean oil significantly decreased the growth of fish, such as gilthead sea bream (*Sparus aurata*) (Montero et al., 2008) and turbot (*Scophthalmus maximus*) (Peng et al., 2014). These negative effects may be partially due to the unbalanced fatty acid profile and poor palatability and digestibility of vegetable oil diet (Caballero et al., 2002; Halver and Hardy, 2002). In the present study, growth was significantly increased by dietary CDCA supplementation. However, no information about the effects of dietary CDCA on aquatic animals are available, although dietary ursodeoxycholic acid could improve the growth performance of Juvenile Eel (Maita et al., 1996) and suitable levels of bile acid could increase the WGR and feed efficiency of yellowtail (Deshimaru et al., 1982). Previous studies in mammals established that bile acids could enhance growth, improve metabolism, prevent diseases and improve digestion of lipids and the products of digestion including dietary cholesterol, phospholipids, bilayers and fatty acids coming from the enzymatic breakdown of triglycerides (Deshimaru et al., 1982; Maita et al., 1996; Pullen and Polin, 1984; Stamp and Jenkins, 2008).

In the present study, the supplementation of CDCA significantly decreased the lipid content of liver and HSI which were markedly increased by the replacement of FO with SO. Similar results have been reported in sharpsnout seabream (Piedecausa et al., 2007), large yellow croaker (Wang et al., 2011) and gilthead sea bream (Menoyo et al., 2007; Fountoulaki et al., 2009) that the replacement of fish oil with soybean oil could lead to the increase of HSI and lipid content of liver. However, there is a general lack of knowledge about the effects of CDCA on lipid metabolism in aquatic animals. Some researches on mammals indicated that bile acid treatment could improve the lipid metabolism such as reducing hypertriglyceridemia and preventing hepatic TG accumulation (Angelin et al., 1978; Bateson et al., 1978; Carulli et al., 1981; Kast et al., 2001; Cipriani et al., 2009). To further explore the effect of CDCA supplementation and dietary soybean oil on the lipid metabolism, the activity of hepatic LPL and HL and the related genes expression were analyzed.

Studies in mammals confirmed that LPL is important in promoting the cellular uptake of fatty acids (Ma et al., 1994; Rinninger et al., 1998; Mead et al., 2002) and hydrolyzing triglycerides in lipoproteins (Oku et al., 2006) while HL is another important lipase with a similar function (Dichek et al., 2006). In the present study, the activity of LPL increased with the increase of the CDCA supplementation but the HL activity was not affected significantly. Similarly, the mRNA levels of LPL were significantly higher in the SOL group compared to other groups while the CDCA supplementation decreased the mRNA levels of HL even though the differences were not significant. Many studies have demonstrated that the LPL activity was increased by the activation of FXR via upregulating the expression of cofactors (Kast et al., 2001) and suppressing the expression of inhibitors of LPL (Kalaany and Mangelsdorf, 2006). Indeed, CDCA had been demonstrated to be the most potent natural ligands for FXR (Parks et al., 1999) and the mRNA levels of FXR were increased with the increase of the supplementation of CDCA in the present study. Thus, the present results suggest that the CDCA supplementation may increase the hepatic LPL activity by the activation of FXR in large yellow croaker.

Previous studies in mammals described SREBP-1 and PPARα as key regulators of metabolism of lipid which could be affected by the n-6/n-3 PUFA ratio (Videla et al., 2004). A diet with a high content of n-6 PUFA could significantly increase the SREBP-1 expression in mice (Kim et al., 1999) and the fatty acid synthase expression in turbot (Peng et al., 2014) which is a known SREBP-1 target (Fiévet and Staels, 2009). Meanwhile the diet with a high content of n-6 PUFA significantly decreased the PPARα expression in Atlantic salmon (Morais et al., 2011). In addition, Ning et al. (2016) demonstrated that the activation of PPARα in Nile tilapia increased lipid degradation and decreased adipogenesis which was similar to mammals. In the present study, significantly lower PPARα expression and higher SREBP-1 expression were observed in fish fed the SO diet compared with fish fed the FO diet. These results suggested that a high n-6 PUFA diet such as soybean oil diet may increase liver lipid deposition by altering the expression of SREBP-1 and PPARα.

The CDCA supplementation significantly increased the expression of PPARα and decreased the expression of SREBP-1 compared with the fish fed soybean oil alone in the present study. As discussed above, FXR is an important regulator in lipid metabolism (Kalaany and Mangelsdorf, 2006) and CDCA is the most potent natural ligand. FXR knockout mice showed marked hepatosteatosis and hypertriglyceridemia (Sinal et al., 2000). Also, the activation of FXR prevented hepatic triglyceride accumulation in a mouse with hypertriglyceridemia (Watanabe et al., 2004) and liver steatosis in an obese mice (Zhang et al., 2006). Further studies showed that the activation of FXR could significantly reduce the hepatic de novo lipogenesis by suppressing the expression of SREBP-1 (Watanabe et al., 2004; Zhang, 2004) and improve fatty acid oxidation by increasing the expression of PPARα in human (Torra et al., 2013). However, the effects of FXR activation on lipid metabolism are complicated. The effects of FXR on PPARα were not observed in mice because of the absence of response elements in the murine PPARα promoter (Torra et al., 2013). Overall, the present

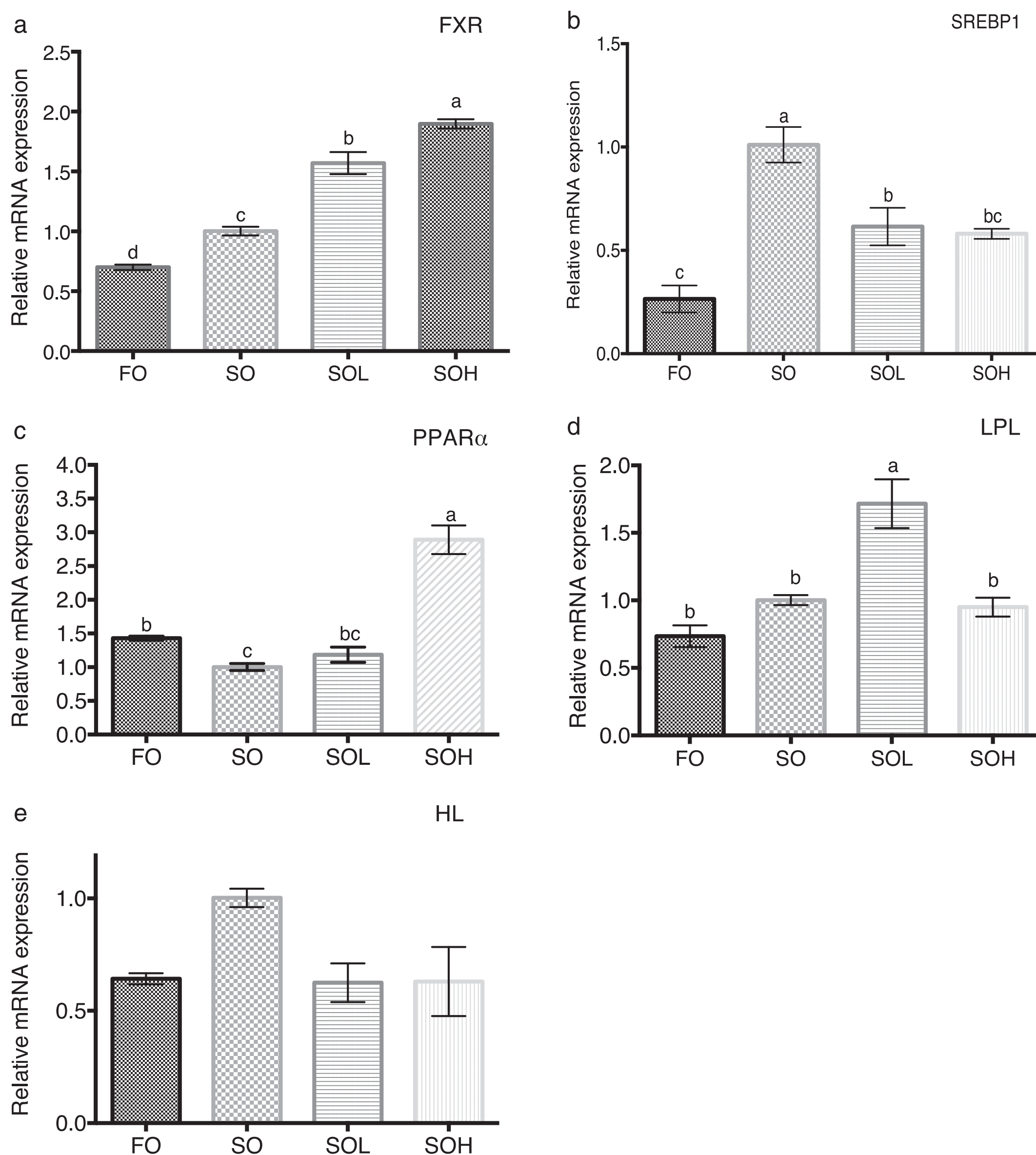

Fig. 2. Effects of dietary soybean oil and CDCA on relative expression of (a) farnesoid X receptor (FXR), (b) sterol regulatory element binding protein-1 (SREBP-1), (c) peroxisome proliferators-activated receptor α (PPARα), (d) lipoprotein lipase (LPL) and (e) hepatic lipase (HL) in larval large yellow croaker. Data were presented as means ± S.E.M. (n = 3). Bars bearing the same letters are not significantly different ($P > 0.05$).

results suggest that the CDCA supplementation may improve lipid metabolism in large yellow croaker by the activation of FXR via inducing the PPARα expression and suppressing the SREBP-1 expression but detailed mechanics need further researches.

Furthermore, growth performance is related to immunity and health. The lack of DHA and EPA and the high content of linoleic acid (18:2n-6) in soybean oil were suggested to negatively affect the immunity and disease resistance in some marine fish such as gilthead sea bream (Montero et al., 2003; Zuo et al., 2014) and large yellow croaker (Tan et al., 2016). Fortunately, some studies in mammals have shown that CDCA treatment had anti-inflammatory effects which are related to the downregulated expression of pro-inflammatory genes (Fiorucci et al., 2004; Wagner and Eferl, 2005). With the above analysis, it could be speculated that dietary CDCA may improve the growth performance of large yellow croaker by reducing inflammatory and improving health. Related research is on going and further studies are needed to explore the mechanisms underlying the effects of dietary soybean oil and CDCA supplementation on the hepatic lipid metabolism and health of large yellow croaker.

In conclusion, high dietary content of soybean oil negatively affected the growth performance and increased lipid accumulation in liver of large yellow croaker while CDCA supplementation could partly reverse the effects. The CDCA supplementation increased hepatic LPL activity and upregulated mRNA levels of LPL and PPARα and downregulated mRNA levels of SREBP-1. Thus, dietary supplementation of CDCA may improve the growth and lipid metabolism in large yellow croaker fed a soybean oil diet.

Conflicts of interest

There are no conflicts of interest to report.

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