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Dietary Role of Omega - 3 Polyunsaturated Fatty Acid (PUFA): A Study with Growing Chicks, *Gallus domesticus*

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Abstract: The 7 days old chicks, *Gallus domesticus* were fed with a diet supplemented with 2.5%, 5% and 10% of ω-3 enriched PUFA (containing 180mg of eciosapentaenoic acid and 120mg docosahexaenoic acid per gram oil) for a period of 30 days. Dietary supplementation of PUFA promotes the growth of the birds that was reflected in the elevation of tissue protein, cholesterol and phospholipid along with a reduction in tissue triglycerides concentrations. Accumulation of ω-3 PUFA along with the depletion of ω-6 PUFA, oleic acid, myristic acid and stearic acid in the tissues was detected. Supplementations of 10% ω-3 enriched PUFA promote the health status of the bird as evident from 20% increase in the haemoglobin concentration of blood, 60% decrease in the serum LDH activity and with no change in the serum cholesterol profiles. 75% reduction in HMG CoA reductase activity along with 62% augmentation of the HMG CoA synthase activity in the liver was recorded which suggest the alteration of cholesterol metabolism in the bird.

Key words: Omega-3, cholesterol, dietary supplementation, tissue protein

Introduction

Although linoleic (9, 12-octa decadienoic acid) acid, a precursor of ω-6 PUFA, is accepted as essential fatty acid for the fishes, poultry birds and mammals, α - linolenic acid (9, 12 and 15-octa decatrienoic acid), a precursor of n3 PUFA, is also essential for the normal development and growth of animals (Henderson and Tocher, 1987). It is important to recognize that linoleic acid will not substitute α - linolenic acid in providing ω-3 PUFA to various tissues. The recognized deficiency symptoms of ω-3 fatty acid in mammals include defective vision (Neuringer *et al*., 1988) and impaired learning ability (Bourre *et al*., 1989). Although dietary linolenic acid may protect chicks from nutritional encephalomalcia induced by vitamin E deficiency (Budowski and Crawford, 1986), an absolute requirement for linolenic acid in poultry has not been demonstrated. Both linoleic and linolenic acids are readily absorbed through the intestinal wall where resynthesis of triacylglycerol and packaging of lipid into proto microns occur for transport to the liver. Both linoleic and linolenic acid follow a number of metabolic pathways which include oxidation in mitochondria to generate ATP, desaturation and chain elongation leading to long chain PUFA of omega 6 and omega 3 and incorporation into the glycerides. Several hormones and dietary factors influence the desaturation of PUFA in mammals (Brenner, 1989) and probably the same may also be true for the birds. Both the series of PUFA undergo further cyclo-oxygenation and lipo-oxygenation pathways to produce several types of eicosanoids. The eicosanoids produced from ω-3 PUFA are functionally different from those produced from ω-6 PUFA. Moreover, the two series of eicosanoids act in antagonistic fashion (Lands, 2000).

Essentiality of long chain polyunsaturated fatty acid (PUFA) in the diet for the brain growth and development has been reported (Broadhurst *et al*., 2002). These are important membrane component and precursor of signaling molecules (Watts and Browse, 2002). The requirement of PUFA during infancy has been related to neonatal growth and development (Patricx and Gerard, 2000). The degree of unsaturated fat plays very important role in the growth of chicks, when four weeks old chick were fed with fat varying in saturated and unsaturated fatty acids from different sources, the metabolism in growing chicks was significantly affected (An-Byong *et al*., 1997). Lopez *et al*. (2001) reported that high fish oil concentration in the diet decreases the saturated and monoenoic fatty acid content in the thymus sample. Castillo *et al*. (2001) showed that fish oil produced a significant reversion of the hypercholesterolemia previously induced by coconut oil feeding. Fish oil also produces a clear decrease in plasma triacylglycerol level. The long chain omega 3 PUFA present in fish oil are extremely effective in lowering of total omega 6 PUFA in liver and muscle (Phetteplace and Watkins, 1989), egg yolk (Hargis *et al*., 1991) of poultry birds.

Although the poultry science in India and other countries is well established with regard to improvement of the meat and larger production of eggs through dietary manipulation, feed formulation of the poultry has not been aimed to improve the health of the consumer (human being) as well as of poultry birds itself. It has been observed that most of the available commercial poultry diets contain 35-40% crude protein and 5-10% of crude fat out of which 60-70% is linoleic acid and 2-3 %

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is linolenic acid. Dietary fatty acids influence the composition of fatty acid profile, more particularly PUFA of both ω-6 and ω-3 series, of the carcass of the animal. Possibility exists to enrich poultry meat with specified PUFA by dietary means, which will offer potential benefits to the birds as well to the consumers. In the present work an attempt has been taken to find out whether the long chain ω3 fatty acid can be supplemented to the poultry birds along with the diet for better growth and maintaining them in well being condition.

Materials and Methods

After obtaining the approval of animal ethics committee of Goa University, the day old poultry chicks, *Gallus domesticus* (Broiler - venkobb strain) were obtained from a local hatchery (Mandovi Hatcheries, Ponda, Goa). The birds were acclimatized to laboratory conditions for 7 days. 7 day-old birds were divided into four groups (six birds per group). Group I birds were maintained with the commercial diet (which served as control). Group II, III and IV birds were supplemented with different doses (2.5%, 5% and 10%) of ω - 3 polyunsaturated fatty acids (Maxepa, a product of M/s., Merck, India) along with the commercial diet for a period of 30 days. This Maxepa contains 180mg Eciosapentaenoic and 120mg of Docosahexaenoic acid per gm of oil. The proximate composition of the feed is presented in Table 1.

Table 1: Proximate composition of feed of chicks. A. Gross Composition

Constituents	% composition of commercial feed (g /100 g of feed)	% composition of feed supplemented with Ω 3 PUFA
Dry matter	92	93.70
Crude protein	38.28	35.75
Crude fat	6.50	15.50
Ash	11.28	10.13
Fibre content	2.86	2.68

B. Fatty acid composition of crude fat of the feed

Fatty acid	Relative % composition of commercial feed	Relative % composition of feed supplemented with Ω 3 PUFA
14:00	7.5	6.26
16:00	16	14.26
16:01	2.3	4.32
18:00	8.3	6.16
18:01	2.5	8.26
18:02	55.5	32.16
18:03	0.5	1.86
20:02	3.2	2.50
others	4.2$	24.32#

$ Unidentified fatty acids of C-14 and C-16 series. # n-3 and n-6 poly unsaturated fatty acids of C-20 and C-22 (with more than three double bonds) series along with unidentified C-14 and C-16 series.

Routine haematological analysis (total count of erythrocytes and leukocytes and haemoglobin concentration) was done. Besides, the tissue protein and tissue lipid profiles viz. total triacylglycerol, total cholesterol, total phospholipid and the fatty acid profiles of the total lipid (Roy *et al.*, 1997) were recorded for liver, pectoral muscle, intestine and total blood. Serum lipid profile including total cholesterol, HDL cholesterol, LDL-cholesterol, VLDL, cholesterol was also recorded using the diagnostic kits (M/s., Crest Biosystems, Goa).

The liver and cardiac function tests were performed by measuring the activities of serum Alkaline phosphatase (EC 3.1.3.1), Glutamate oxalate transaminase (EC 2.6.1.1), Glutamate pyruvate transaminase (EC 2.6.1.2), Lactate dehydrogenase (EC 1.1.1.27) according to the methods given by Godkar (1994). Two regulatory enzymes activity of the cholesterol metabolism namely HMG CoA reductase (EC 1.1.1.34) and HMG CoA synthetase (EC 2.3.3.10) were recorded following the methods of Siedel (1983) and Miziorko (1985) respectively.

All the results were expressed as mean value of six observations and their standard error. Besides, the results of different dietary groups of birds were treated with Analysis of Variance and Student's t-test (Bailey, 1994).

Results

It was observed that the dietary supplementation of ω 3 enriched fish oil over a period of one month stimulates growth, in terms of final weight, of the *Gallus domesticus* (Table 2). The highest weight was recorded for group IV birds supplemented with 10% of Maxepa (dose 3) indicating 35% ($P<0.005$) more growth over that of the control group of birds. This net weight gain of the bird upon supplementation of 10% of ω 3 enriched PUFA was followed by about 30% increase in the daily instantaneous growth rate (G_w) and 30% decrease in the FCR value (Hardy 1989). It was observed in our laboratory that the growing chicks cannot digest the food if the fat content is more than 10-15% depending upon the unsaturation index of the fat.

Table 2: Growth chart of chick (*Gallus domesticus*) supplemented with different doses of Ω 3 enriched PUFA along with the commercial feed for 30 days during post hatching development. (Mean values of six samples and their standard error)

Parameters	C	D 1	D 2	D 3
Average net weight gain of the birds, g	1300.00±150.67	1431.30±152.67	1597.84±120.30	1755.40±110.27
Daily instantaneous growth rate, Gw	0.237	0.240	0.29	0.31
Feed conversion ration, FCR	1.064	0.952	0.875	0.762

C - Control, D1 - 2.5% dose, D2 - 5% dose, D3 - 10% dose.

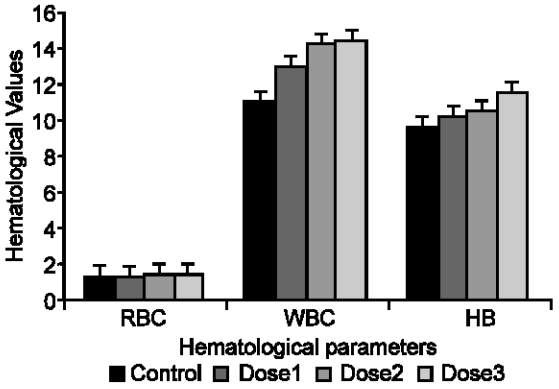

Fig. 1: Changes in the routine haematological parameters of the growing chicks, *Gallus domesticus* supplemented with 2.5%, 5% and 10% of ω 3 enriched PUFA over a period of 30 days. Erythrocytes (RBC) count as no. of cells x 10^6 /ml of blood, leucocytes (WBC) count as no. of cells x 10^4 / ml of blood and haemoglobin (Hb) concentration as g/100 ml are expressed. Mean values of six samples from different chicks and their standard error is plotted.

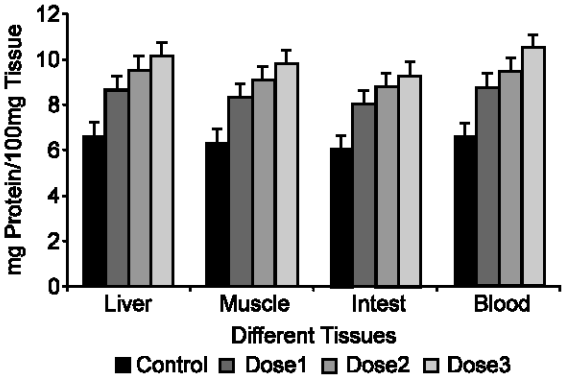

Fig. 2: Effect of dietary supplementation of 2.5%, 5% and 10% of ω 3 enriched PUFA over a period of 30 days, the concentration of total tissue protein of the growing chicks, *Gallus domesticus*. The tissue protein concentration is expressed as mg protein /100 mg of tissue wt. except in blood where the concentration is expressed as mg protein / 100ml of blood. Mean values of six samples from different birds and their standard error is plotted.

Although the total count of erythrocytes remained unaltered due to supplementation of ω - 3 PUFA, almost 30% increase (P<0.001) in the leucocytes count and 20% increase (P<0.001) in the concentration of haemoglobin were noticed over a 30- days period of supplementation of highest dose of PUFA (Fig. 1).

From Fig. 2, it is clear that there was a significant variation (F = 10.68 to 61.80, P<.01) in the tissue protein concentration of the poultry birds supplemented with different doses of PUFA. The dose dependent and tissue dependent increase in the tissue protein concentration was noticed. The maximum level of protein was observed in total blood in comparison to liver, pectoral muscle and intestine. About 50-60% elevation of tissue protein (P<0.001) was noticed in the group IV birds as compared to the control birds.

It is evident that the concentration of total triglycerides, total cholesterol and total phospholipid in different tissues of birds depends on the dose of the supplemented PUFA (F = 25 to 75, P<0.001). Although significant increases (18% to 63%, P<0.01) in the tissue level triglycerides (Fig. 3) and phospholipid (Fig. 5) were noticed with the supplementation of 2.5% PUFA for a period of 30 days, a decreased trend was observed from the second dose onwards. It is interesting to note that with the highest dose of PUFA supplementation tissue triglyceride concentrations are reduced by 13% to 21% (P<0.05) in comparison with the same of control birds. However, a dose dependent increase in the concentration of cholesterol was seen up to the second dose of PUFA supplementation and then a decreasing trend was observed (Fig. 4). This trend is clear in liver, pectoral muscle and intestine. In blood, the triglycerides and cholesterol levels did not show very prominent dose dependent reductions.

With the dietary supplementation of PUFA for a period of 30 days, the tissue level fatty acid profiles of birds were greatly altered (P<00.1). With the decrease in the saturated fatty acid (mainly myristic and stearic acid), monoenoic acid (oleic acid), ω - 6 fatty acids (namely linoleic and arachidonic acid) and augmentation of ω-3 fatty acid (namely linolenic, eciosapentaenoic and docosahexaenoic acid) brought a significant rise in ω -3/ ω- 6 ratio (from 0.25 to 1.53) and lowering (0.69 to 0.38) ratio of total saturated fatty acids and total unsaturated fatty acids (Table 3).

The activities of some liver function enzymes and cardiac function enzymes were presented in Table 4. About 13% increase (statistically equivocal) in the activities of serum GOT and serum GPT and about 60% decrease (P<0 .001) in the activity of serum LDH were recorded due to supplementation of 10% PUFA for a period of 30 days. No significant changes were recorded with respect to the activity of serum ALP.

A dose dependent increase in the level of serum cholesterol and VLDL cholesterol up to the second dose of PUFA supplementation for a period of 30 days was noticed in *Gallus domesticus* (Fig. 6). An initial 90% augmentation in serum LDL cholesterol and 30% declination of serum HDL cholesterol was noticed with

Table 3: Effect of dietary supplementation of Ω 3 enriched PUFA on the fatty acid profiles of total lipid in different tissues of poultry birds, *Gallus doemsticus*. (Mean value of six birds and their standard error)

Fatty acid profiles	Control				Dose 3			
	L	M	I	B	L	M	I	B
14:00	10.20	10.50	10.00	9.10	7.50*	7.80*	8.50*	7.20*
16:00	12.60	11.30	11.50	10.20	10.20*	11.20	10.20*	9.50
16:01	4.20	3.60	4.40	5.20	3.40	2.60*	4.80	4.60
18:00	18.20	16.30	15.20	16.30	14.20*	13.30*	12.20*	10.80*
18:01	17.20	19.30	19.20	18.50	15.60*	16.50*	15.50*	14.50*
18:2 (ω-6)	16.30	16.00	14.50	15.20	10.00*	11.20*	10.60*	9.50*
18:3 (ω-3)	1.20	2.50	2.40	1.60	5.50*	6.20*	2.50	5.80*
20:2 (ω-6)	2.50	1.80	2.60	2.80	1.80	2.50	2.90	2.20
20:4 (ω-6)	10.20	9.30	10.50	11.50	8.30*	7.50*	7.20*	8.50*
20:5 (ω-3)	2.50	2.20	4.50	3.60	8.50*	5.20*	10.80*	8.60*
22:6 (ω-3)	3.50	4.60	3.80	4.20	13.50*	13.90*	13.20*	16.50*
Others	1.40	2.60	1.40	1.80	1.50	2.10	1.60	2.30
TS / TUS	0.69	0.62	0.58	0.55	0.47	0.48	0.45	0.38
(ω-3) / (ω-6)	0.25	0.34	0.39	0.32	1.37	1.19	1.28	1.53

* Changes are significant in compare to the value of control birds. L - liver; M - muscle; I - intestine; B - blood.

Table 4: Effect of dietary supplementation of Ω 3 enriched pufa on liver function and cardiac function enzyme activity in serum of poultry birds, *Gallus doemsticus*. (Mean value of six birds and their standard error).

Name of enzyme	Control	Dose 3
Liver function test		
Alkaline phospahtase (IU/mg of protein)	33.56±1.08	34.32±1.34
Glutamate pyruvate transaminase (IU/mg of protein)	0.15±0.006	0.17±0.005
Cardiac function test	2.76±0.28	1.05±0.25
Lactate dehydrogenase (IU/mg of protein)		
Glutamate oxaloacetate transaminase (IU/mg of protein)	0.14±0.006	0.16±0.007

Table 5: Effect of distary supplementation of Ω 3 enriched pufa on cholesterol metabolism enzyme in serum and liver of poultry birds, gallus doemsticus. (mean value of six birds and their standard error)

Name of the enzyme	Control		Dose 3	
	Liver	Serum	Liver	Serum
HMG CoA reductase	681.21	51.61	165.11	41.47
(IU/mg of protein)	22.57	3.34	12.23	3.40
HMG CoA synthetase	24.60	12.18	39.91	22.94
(IU/mg of protein)	4.35	2.12	3.67	4.50

dietary PUFA supplementation. The serum cholesterol as well as VLDL-, LDL-, HDL-cholesterol levels were brought to the normal level (as in control birds) with 10% of PUFA supplementation.

With the supplementation of 10% of PUFA for a period of 30 days, in liver the HMG CoA reductase activity was declined by 75% ($P<0.001$) with 62% augmentation in HMG CoA synthase activity ($P<0.05$). However the 20% decrease in HMG CoA reductase activity and 88% increase in HMG CoA synthase activity (Table 5) in serum statistically may or may not be significant as the value of the student "t" lies between probability levels 0.1 to 0.05.

Discussion

Both the linoleic and α linolenic acid cannot be synthesized *de novo* by animals (Henderson and Trocher, 1987) but are very essential for the animals for their growth and physiologically well being state. The natural occurrence of α- linolenic acid in the terrestrial and fresh water environment is restricted and that might be reason for lower level of accumulation of ω-3 fatty acids in the body. Arachidonic acid (ω-6 PUFA), eciosapentaenoic acid and docosahexaenoic acid (ω-3 PUFA) are the major long chain PUFA constituents of all subcelluar membranes of liver, gill, erythrocytes, brain of fish and they play very important role to maintain the structural integrity of the membranes (Roy *et al.*, 1997, 1999; Shivkamat and Roy, 2005). These PUFA undergo further metabolism to produce lots of prostaglandin and thromboxane of diene and triene series (Lands, 2000). Much of the expected metabolism and function of eicosanoids in poultry are thought to follow those that have been reported in other animals.

Dietary PUFA and the growth of the birds: Dietary fat spare protein and amino acid from energy yielding processes and direct them towards the growth of the animals. The observed 35% increase in the net weight gain along with about 30% increase in the daily instantaneous growth rate (G_w) and 30% decrease in FCR with 10% supplementation of PUFA (Table 2) indicates the PUFA as growth promoting substance. The rapid growth rate during post hatching developmental period with the supplementation of ω- 3 PUFA meets the nutritional requirement of α-linolenic acid and might instigate the gene transcription for the growth promoting protein as in human infancy (Lapillone and Carlson, 2000). From the Fig. 2 and 3, it is clearly evident that the increase in the net weight gain or the daily spontaneous

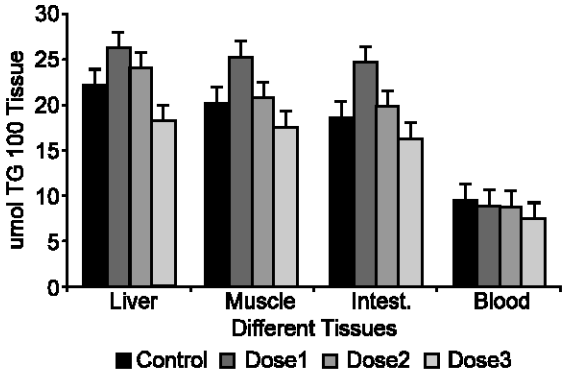

Fig. 3: Effect of dietary supplementation of 2.5%, 5% and 10% of ω 3 enriched PUFA over a period of 30 days, on the concentration of total triglycerides (TG) in different tissues of the growing chicks, *Gallus domesticus*. The triglycerides concentration is expressed as µmol TG /100 mg of tissue wt. except in blood where the concentration is expressed as µmol TG/100ml of blood. Mean values of six samples from different birds and their standard error is plotted.

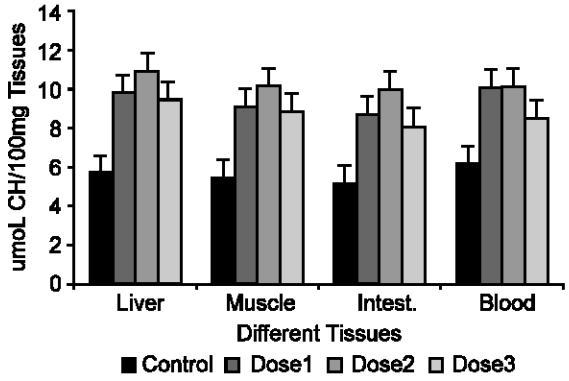

Fig. 4: Effect of dietary supplementation of 2.5%, 5% and 10% of ω 3 enriched PUFA over a period of 30 days, on the concentration of total cholesterol (CH) in different tissues of the growing chicks, *Gallus domesticus*. The cholesterol concentration is expressed as µmol CH /100 mg of tissue wt. except in blood where the concentration is expressed as µmol CH/ 100ml of blood. Mean values of six samples from different birds and their standard error is plotted.

growth rate of the poultry birds due to supplementation of ω-3 PUFA is accompanied with the accumulation of protein and fat in the various tissues. The energy content of the food with the supplementation of PUFA enhances by the caloric factor of 9.5 k calories per gm of added PUFA (Henken *et al.*, 1986).

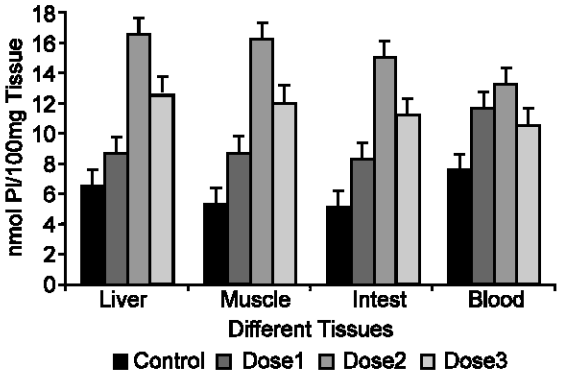

Fig. 5: Effect of dietary supplementation of 2.5%, 5% and 10% of ω 3 enriched PUFA over a period of 30 days, on the concentration of total phospholipid (PL) in different tissues of the growing chicks, *Gallus domesticus*. The phospholipid concentration is expressed as ηmol PL /100 mg of tissue wt. except in blood where the concentration is expressed as ηmol PL/ 100ml of blood. Mean values of six samples from different birds and their standard error is plotted.

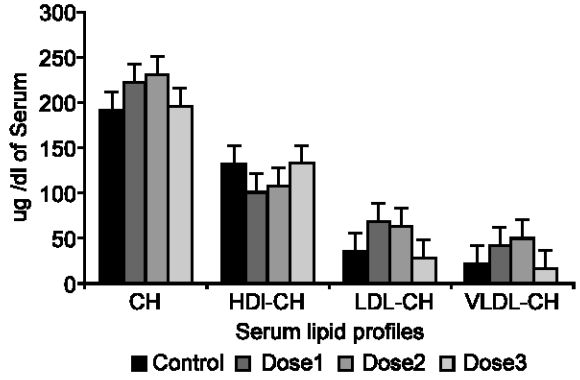

Fig. 6: Effect of dietary supplementation of 2.5%, 5% and 10% of ω 3 enriched PUFA over a period of 30 days, on the serum cholesterol profiles of the growing birds, *Gallus domesticus*. The cholesterol concentration is expressed as µg /100 ml of serum. Mean values of six samples from different birds and their standard error is plotted.

Increase in linolenic, eciosapentaenoic and docosahexaenoic acids along with the decrease in myristic acid, stearic acid, oleic acid, linoleic acid and arachidonic acid in the tissues (Table 3) due to dietary supplementation of 10% ω 3 enriched over a period of 30 days once again confirm the earlier observations of Hargis *et al.*(1991), Cherian and Sim (1991). Dietary supplementation of ω-3 PUFA might modulate the

desaturation systems in birds that need to be confirmed in future. With the increased dose of PUFA the accumulation of cholesterol and phospholipid (Fig. 4 and 5) and depletion of triglycerides (Fig. 3) help to reduce the accumulated fat and channelize it towards the energy yielding process. The reduction in the lipogenesis due to dietary PUFA may be due to the inhibition of different lipogenic pathways (Kersten, 2001).

Dietary PUFA and well being State: PUFA in cell is required for the production of chemical messengers that initiate or control a wide number of physiological function including cell growth and division, control of blood pressure, coagulation of blood, immune reaction tissue inflammation etc. (Lands, 2000). PUFA reduce the incident of narcotizing enterocolitis by modulating platelet activating factor and endotoxin trans location (Caplan and Jilling, 2001). 5 to 20% increase in blood haemoglobin concentration along with 17 to 30% increase in leucocytes count with the supplementation of PUFA confirms the earlier observation of Klinger *et al.* (1996). Enhanced leucocytes count in the blood may be correlated with the increased immuno protective condition upon supplementation of PUFA. Increased haemoglobin concentration is attributed for the better binding of the oxygen molecules for oxidative metabolism of dietary fat which has to be metabolized for energy yielding process to spare protein and amino acid for the growth.

Serum cholesterol profile in the form of HDL, LDL and VLDL cholesterol concentration is a key indicator to understand the health condition of animals. Increasing HDL concentration and lowering of LDL and VLDL cholesterol concentration in serum prevents cardiovascular diseases. From the Fig. 6, it is evident that 10% supplementation ω-3 PUFA did not cause any health hazards to the birds. Daggy *et al.* (1987) already reported that long chain PUFA helps in lowering the production rate of VLDL in roosters. High content of EPA and DHA in certain fish oil prevent the rat from cardiovascular diseases like thrombosis and arteriosclerosis (Banerjee *et al.*, 1992). Dietary PUFA alter inositol phosphate metabolism and protein kinase c activity and thus regulate inter cellular signaling system (Nair *et al.*, 2001). The slight elevation (13%, $p<0.10$, equivocal) of serum GPT, GOT and 60% decrease ($p<0.001$) in serum LDH activity with no change in serum ALP in the PUFA supplemented bird (10% supplementation) as observed in the present study (Table 4) did not find any clue for the necrosis of liver or cardiac tissue. This change may be correlated with shifting of the metabolic pathways, which need to be confirmed in future. Dietary supplementation with palm oil lowers creatine concentration in serum and the activity of GPT in broiler chicken (Olurede and Longe, 2001).

Dietary PUFA and Cholesterol metabolism: The reduction of HMG - Co A in liver and partially in serum (Table 4) once again confirm the lowering of tissue level cholesterol (Fig. 4) by reducing the cholesterol biosynthesis which is also accompanied with the reduction of tissue level triglycerides (Fig. 3) and phospholipid (Fig. 5). Whether these changes in the lipid metabolism, more particularly cholesterol metabolism by exogenous dietary ω 3 PUFA are due to by inducing the transcription of genes encoding protein involved in lipid oxidation or by suppressing the expression of genes encoding protein involved in lipid synthesis (Jump and Clarke, 1999) are yet to be confirmed. Cellular cholesterol in animals is controlled by a family of transcription factors known as sterol regulatory element binding protein (SREBP), which exist in three isomeric forms. PUFA opposes cholesterol-mediated induction of SREBP (Kim *et al.*, 2002). PUFA decrease the hepatic abundance of SREBP 1c, appears to be involved with the regulation of lipogenic gene transcription and 1a, which are able to activate both lipogenic and cholesterol genic gene (Osborne, 2000) by accelerating the rate of mRNA decay (Xu *et al.*, 2001). Supplementation of ω 3 PUFA, over a period of 30 days might reduce the gene expression of SREBP resulting a reduction of HMG CoA reductase, 75% in liver ($p<0.001$) and 20% in serum (equivocal) and lowering LDL m RNA levels. Xu *et al.* (2002) reported that the dietary PUFA increased the nuclear content of the third isomer of SREBP i.e. SREBP 2 and the expression of the cholestrolgenic gene, HMG CoA synthase, whereas they concomitantly suppress the hepatic abundance of SREBP and consequently the expression of lipogeneic genes.

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