Journal of Food Technology 4 (2) : 122-127, 2006

Comparative Study of Lipoprotein Metabolism in Chicken, Turkey and Quail

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Abstract: Lipogenesis plays a role in the maintenances of body composition in the growing animals. Lipogensis was regulated in the liver and adipose tissue by the composition of macronutrients in diet. High carbohydrates and little fat was elevated lipogenesis than rich fat and low carbohydrates. Serum samples were preferred because of cholesterol and triglyceride concentration is about 3-5% higher in serum than in EDTA plasma, although no significant serum plasma difference was observed for HDL. There are a variety of methods to measure the lipoprotein classes. All require separation of the classes before they can be measured and recently analysis was performed using enzymatic kits. Hormonal regulation of lipogenesis has been investigated by measuring hormone levels in the blood as a function of dietary state and by ablating specific endocrine glands in the intact animals, following replacement therapy with specific hormone. Hormone identified in experiments with intact animals was then tested in cell culture systems. Similarly the effect of nutrition on lipoprotein metabolism was reviewed. In the species point of view, the turkey in contrast to chicken seems to be a bird in which muscle growth is more important than adipose tissue growth. This difference in rate of fat deposition in extra-hepatic tissue is due to inherited differences. On the other hands, atherosclerotic plaques were found more severe in the quail selected for high cholesterol than in that selected for low cholesterol.

Key words: Lipoprotein metabolism, chicken, turkey, quail

INTRODUCTION

Lipogenesis is conversion of glucose to triacylglycerol and the liver was the important site of fatty acid synthesis in most animals. Most glycolytic reaction is reversible, allowing the same enzyme to catalyze both catabolism of glucose to pyruvate (glycolysis) and synthesis of glucose from pyruvate (glucogenesis). Glucose derived from the metabolism of dietary carbohydrate is the primary substrate for fatty acid synthesis in most tissues in the body, including liver, adipose tissue and mammary and subcutaneous glands. Fatty acid is important for all cells function, essential for the modification of some proteins and complex carbohydrates. Lipogenesis in liver and adipose tissue convert dietary carbohydrate to triacylglycerol, if the carbohydrate is in access of that required for immediate energy needs; the triyglycerol is stored in adipose tissue and used during periods of food deprivation.

High fat low carbohydrate diets inhibit fatty acid synthesis, fatty acid synthesis also may contribute to elevated levels of plasma tryglycerols and therefore, to development of atherosclerosis[1]. Lipogenesis plays a role in the maintenances of body composition in the growing animals. For instance, chicks of broiler strains weight about 50 g at hatching and grow to 2 kg in 7 weeks, consuming diet contain 10% of its calories. The lipogenic enzyme activates can increase as much as 100 fold during the transition from starved to the feeds state[2]. The composition of macronutrients in diet also regulated lipogensis in the liver and adipose tissue. High carbohydrates and little fat elevated lipogenesis in the lipogenesis than rich fat and low carbohydrates[3]. Our aim was to study lipoprotein metabolism in view of research carried out previously and compare it in different kind of birds in order to investigate genetic polymorphism between breeds.

MATERIALS AND METHODS

Sample collection: Recent food intake exerts little effect on plasma total cholesterol and triglyceride concentration. Therefore, increase in postprandial plasma is related to the fasting triglyceride levels and the amount of fat intake. This is due to the appearance of chylomicrons in the circulation after a fat containing meal and chylomicrons are normally cleared within 9-12 hrs. Transient decreases in HDL and LDL cholesterol were also occur. In general anticoagulants exert osmotic effects in which water leaves the cells and enters the plasma, thus diluting the plasma and lowering the concentrations of non-diffusible components. Serum cholesterol and triglyceride concentration are about 3-5% higher in serum than in EDTA plasma, although no significant serum plasma

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difference was observed for HDL. Sample volume required are as follows 0.2 mL for total cholesterol and triglyceride, 0.5 mL for HDL cholesterol (heparin Mn method) and 0.2 for HDL with direct method[4]. Accordingly, in our laboratory we collected blood samples from fasting chicken and within 15 min, whole blood was centrifuged at 3000g for 15 min at 10°C and then the resultant serum divided into aliquots and immediately stored frozen at -80°C.

Analysis method: The National Cholesterol Education Program (NCEP) working group on lipoprotein measurement was convened in 1989 to develop recommendations for HDL-cholesterol, as well as for LDL-cholesterol and triglyceride measurements and their recommendations was reported in detail by[5]. There are a variety of methods to measure the lipoprotein classes. All require separation of the classes before they can be measured. One way to separate them is by spinning serum for a long time in a high-speed centrifuge (ultracentrifugation). Following centrifugation, the most complete measurement of all the lipoprotein classes is done using electrophoresis[6]. This procedure measures the quantity of each lipoprotein class based on its movement in an electrical field. Other, less extensive procedures are also used. For example, Total cholesterol, free cholesterol, LDL cholesterol, HDL cholesterol and triacylglycerols were analyzed using enzymatic kits. Similarly, if only HDL is to be measured, a chemical is added to the serum that will clump the other classes, leaving HDL free in the serum to be measured by enzymatic kits. LDL and VLDL often are not measured directly but its level is calculated based on the measurements of total cholesterol, HDL and triglycerides using Friedewald formula: LDL = total cholesterol-HDL- (triglycerides/5)[7]. In addition Kouba *et al.*,[8] was used the following formula [(total cholesterol-free cholesterol) X 1.67] to estimate turkey plasma cholesteryl esters. In this calculation it was assumed that the factor for the ratio of average molecular weight of turkey plasma cholesteryl esters to that of the molecular weight of free cholesterol (1.67) were the same in human and turkeys.

RESULTS AND DISCUSSION

The effect of hormones: Hormonal regulation of lipogenesis has been investigated by measuring hormone levels in the blood as a function of dietary state and by ablating specific endocrine glands in the intact animals, following replacement therapy with specific hormone. Hormone identified in experiments with intact animals was then tested in cell culture systems. Insulin and glucagons is a likely signal for the onset of feeding because its response to dietary carbohydrate is large and rapid, a third potential mediator of nutritional status is the thyroid hormone 3,5,3- tri-iodothyronine (T3), circulating T3 levels are increased in the feed state and decreased during starvation in rat[9], chickens[10] and humans[11]. Administration of glucagan to either fed or starved chicks decrease the concentration of circulating Ts[12] and decreased conversion of thyroxin to T3 in liver is probable used[9]. Hyperthyroidism, achieved by administrating thyroid hormone to euthyroid animals, increases rates of hepatic fatty acid synthesis and elevates activates of lipogenic enzyme[13]. The adrenal cortex also is involved in the regulation of lipogensis, although the role glucorticoids in nutritional regulation is less clear then those of insulin, glucagons and T3[14]. In contrast to their action in liver, glucorticoids inhibit lipogensis in adipose tissue[13]. In the adipose tissue insulin increases the number of glucose transporters in the cell membrane[15], in liver glucose transport is independent of insulin[16]. Fat was inhibits the activity of lipogenic enzymes and reduces the rate of fatty acid synthesis[17]. The type of dietary fat influences the composition of triglycerol in chylomicrons and intestinal VLDL. Most of the cholesterol in plasma is transported on 3 major lipoprotein class VLDL-cholesterol, LDL- cholesterol and HDL- cholesterol. The lipoprotein class can be further subdivided using nuclear magnetic resonance spectroscopy. Lipoprotein (a) can not measured by nuclear magnetic resonance spectroscopy, so an immunological assay must be used[18]. Since the liver is the main site of de novo fatty acid synthesis in birds[19] , most fat that accumulates in avian adipose tissue is synthesized in the liver or derived from the diet. Lipids are supplied to adipocytes from triglyceride-rich lipoprotein, i.e., intestinal portomicrons and hepatic very low density lipoproteins VLDL[20].

The effect of nutrition: Conventional reasons to add fat to animal diets include: use as an energy source, to control dust and improve palatability of the diet and in rare cases, as a source of essential fatty acids. Alternative reasons include 1) alteration of the fatty acid profile of a particular product and thus creation of (value-added) products and 2) alteration of adipose development via fatty acid effects on gene expression. The effects of dietary fat on tissue fatty acid profile and on adipocyte development have been investigated in both ruminants and nonruminants[21]. Pettigrew and Moser[22] summarized the effects of dietary fat on performance and carcass characteristics from over 90 studies and concluded that

the increase in carcass fat was independent of whether the calorie: protein ratio in the diet was maintained. Earlier work had suggested that the effects of dietary fat can be offsetif the calorie: protein ratio is maintained[23]. However, the extra-caloric and extra-metabolic effects of dietary fat result in greater efficiency of digestion and energy retention, which most likely account for the increased carcass fat despite this adjustment[21]. Diets deficient in fatty acids will cause metabolic disorders[24].Depressed growth especially in male chickens may be the first sign of an inadequate supply of essential fatty acids. Abnormalities in the structure of membranes, capillaries and skin as well as a general depression of immunity are among the most important consequences of major deficiencies[25]. Usually, fats with a high percentage of unsaturated fatty acids are better absorbed than highly saturated lipids, with the possibility of synergistic effects between fats and different composition[26]. The digestion of animal fat especially improves significantly with increasing age[27]. The fatty acid pattern of the abdominal fat was significantly influenced by the dietary fatty acid[28]. Total cholesterol and triglyceride levels in LDL increased after fat supplementation to the diet. On the other hands[29] suggested that higher insulin levels in broilers fed diets rich in saturated fatty acids could be related to higher fat deposition. Fat deposition in birds fed high fat diets was not correlated with circulating VLDL; the higher correlation with abdominal fat suggests that in these birds, fat deposition is more dependent on hepatic VLDL secretion, despite the high dietary fat level. Total serum cholesterol changed with age among birds. While, serum triglycerides and very low-density lipoprotein cholesterol increased over the 14 to 42 day period in chicks that received dietary lard. Dietary lipids are absorbed and package into chylomicrons in intestinal epithelial cells, secreted into lymph and then enter blood stream, they are remodeled forming cholesterol. Plasma concentration of total cholesterol, VLDL- cholesterol and LDL- cholesterol in chicken fed with 3% cholesterol supplemented diet was increases whereas HDL-cholesterol concentration were not change and VLDL triglycerols concentration were elevated. Cholesterol uptake in mammalian livers is regulated by LDL receptor, which is located on the plasma membrane and is stimulated by intra-nuclear Sterol Regulatory Element Binding Protein (SREBP) level[30].

Lipoprotein synthesis in chicken: VLDL and HDL are synthesized and secreted by liver apolipoprotein B-100 and apo A-I[31] are the major apolipoproteins of chicken VLDL and HDL, respectively. It is unknown why triglycerides are preferentially associated with apo B into VLDL particles, whereas most of phospholipids and cholesterol are associated with apo A-I in HDL. In chicken insulin enhances both denovo lipogenesis and VLDL synthesis, whereas thyroxine and glucogen have apposite effects[32]. Lipoprotein lipase catalyses the hydrolysis of triglycerides to fatty acid and glycerol and then the fatty acid enters the surrounding tissues, in adipose tissues they are re-esterified and stored as triglycerides. In laying hens, the plasma catabolism of VLDL is limited[33], which allows the transport of lipid to oocytes. In bird LPL regulation in adipose tissue seems to be less sensitive to nutritional state[32].

Recently we found high concentration of TG and VLDL in fat chicken line, while the cholesterol and LDL concentration was low, when compared with lean chicken line. High density lipoprotein HDL concentration was similar in both lines. Cholesterol concentration was positively associated with LDL and triglyceride was positively associated with VLDL. HDL was negatively associated with triglyceride and cholesterol. In fat chicken line LDL was positively associated with breast muscle and abdominal fat weight. While, in lean chicken line, cholesterol and LDL was positively associated with all carcass traits.

Lipoprotein synthesis in turkey: Among domestic fowl, the leanness of turkey is well known compared, to chicken in which rapid growth is paralleled with excessive fattening. The physiological mechanisms of this difference are still widely unknown and required elucidation[34]. Therefore, most studies were focused on the synthesis, secretion and adipose uptake of hepatic (VLDL) to understand the specific leanness of the turkey[8,35,36]. While other lipoprotein classes were partly characterized in the adult male turkey by[37]. Indeed the development of adipose tissue is very limited in turkey and meat itself is very poor in lipids (1-2%)[38]. This make the turkey very attractive for human consumer concerned about the nutritional quality of food. Kouba *et al.,* [36] demonstrated that hepatic lipogenesis is much lower in turkeys than in chickens. This suggests that triglyceride output from the liver is much lower in turkeys than in the chicken.

The abdominal fat which is quite representative of fattening in birds, was always much higher in broilers than in turkeys[8]. Similarly, VLDL and total plasma TG concentrations were usually higher in chickens than in turkeys, whatever the physiological state and

experimental conditions[8]. Kouba *et al*., [8] observed that chicken VLDL were significantly poorer in cholesteryl esters than those of turkey ($p<0.05$). For fatty acid composition of the VLDL lipids of chicken and turkeys found that linoleic acid content was always higher in turkeys than in chickens ($p<0.05$), while monounsaturated fatty acid content of chicken VLDL was always higher than that of turkey VLDL ($p<0.01$). The hepatic secretion of VLDL, VLDL-TG and total TG was always higher in chicken than in turkey in both young and old birds, but the rate of secretion was decreased in both species with age[8]. In response to a diet rich in Alph- linolenic acid, the young turkey is more efficient than the young chicken in long chain poly unsaturated fatty acids, this difference in the response to diet may rely on a difference in lipoprotein and especially HDL, metabolism between turkey and chicken[38]. In all lipoprotein classes, including triglyceride-rich lipoprotein (VLDL, IDL and LDL) the proporation of triglycerides is lower than in other avian species (43% of total lipids only in VLDL vs. 55% and 50% in the chicken and goose, respectively) [39]. This low triglyceride content of the turkey lipoproteins is consistent with the low lipogenic capacity of this bird[36].

Lipoprotein synthesis in quail: Quail lipoprotein was composed of three fractions: VLDL, $d<1.020$; LDL, $1.020<d<1.081$; HDL, $1.081<d<1.210$. When animals were fed the cholesterol-free diet, HDL was the predominant form, LDL intermediate; VLDL and chylomicron were smallest in amount[40]. Feeding of cholesterol induced a marked change in the lipoprotein profile. Japanese quail lines were divergently selected over 32 generations for laying hen plasma yolk precursor, as measured by Total Plasma Phosphorus (TPP) and then the High Phosphorus (HP) and Low Phosphorus (LP) lines were developed compared with controlled (R1)[41]. The changes in TPP was found due to genetic selection in the Japanese quail lines which were associated with large alterations in plasma VLDLy concentration decrease gradually from (HP, R1 and LP), respectively. Basal plasma levels of hormones associated with reproduction and lipid metabolism were also different among lines. The results suggest possible increased rates of hepatic lipogenesis, hepatic VLDLy assembly and secretion and plasma VLDLy concentration in association with increases in concentrations of plasma LH, T3, T4 and 17 beta-estradiol[41] .

In the nutritional point of view[42] investigate the effects of various levels of dietary chromium supplementation on performance, carcass traits, blood chemistry and tissue distribution of chromium (Cr3+) in quails. He found that Chromium supplementation decreased carcass fat percentage, serum low-density lipoprotein (LDL) and glucose and increased serum magnesium (Mg) and Cr content of kidney, liver and muscle. Dietary onion and garlic caused an increase in the level of liver and plasma total lipids in Japanese quails. This was due to the effect of an increased feed intake, bile production, digestion and absorption. However, there was no increase in the muscle lipid content. This effect in the muscle could be due to inhibitory effect of onion or garlic on lipoprotein lipase activity[43].

The proportions of plasma high and low density lipoprotein cholesterol have been linked to inherited tendency for atherosclerosis in humans. In Japanese quail males from lines genetically selected for high and low total cholesterol (TC) and (unselected) control line were fed 0.0 or 0.5% cholesterol for 12 weeks. Atherosclerotic plaques were found more severe in the high than in the low line quail and in those fed cholesterol compared to non-cholesterol-fed quail. Serum TG, TC, VLDLC, LDLC and HDLC were also higher in the high than in the low line quail and in cholesterol-fed vs. non-cholesterol-fed quail. Significant interactions indicated that TC and LDLC concentrations were more affected by dietary cholesterol in the high line than in the low line. The low line quail maintained higher HDLC and lower LDLC than the high line[44]

REFERENCES

1. Bradley, F., H. Gartner, T. Charron and K.A. Chesnut, 1996. Alterations in nutritional status regulate acetyl-CoA carboxylase expression in avian liver by a transcriptional mechanism. Biochem. J., 319: 263-268.
2. Cladaras, C. and G.L. Cottam, 1980. Turnover of liver pyruvate kinase. Arch. Biochem. Biophys., 200: 426-433.
3. Volpe, J.J. and P.R. Vagelos, 1974. Regulation of mammalian fatty acid synthetase. The roles of carbohydrate and insulin. Proc. Natl. Acad. Sci. USA, 71: 889-893.
4. Virgil, D., 2000. Laboratory procedure manual: Total cholesterol, HDL-cholesterol, triglycerides and LDL-cholesterol. Johns Hopkins University, School of Medicine, pp: 1-28.
5. Warnick, G.R. and P.D. Wood, 1995. National cholesterol education program recommendations for measurements of high density lipoprotein cholesterol: Executive summary. Clin. Chem., 41: 1427-1433.

6. Chapman, M.J., S. Goldstein, D. Lagrange and P.M. Laplaud, 1981. A density gradient ultracentrifugal procedure for the isolation of the major lipoprotein classes from human serum. J. Lipid Res., 22: 339-358.
7. Friedewald, W.T., R.I. Levy and D.S. Fredrickson, 1972. Estimation of the concentration of low-density lipoprotein cholesterol without the use of the preparative ultracentrifuge. Clin. Chem., 18: 499-502.
8. Kouba, M., D. Hermier and M.A. Bernard-Griffiths, 1995. Comparative study of hepatic VLDL secretion *in vivo* in the growing turkey (Meleagris gallopavo) and the chicken (Gallus domesticus). Comp. Biochem. Physiol., 110B: 47-55.
9. Harris, A.R.C., S.L. Fang, L. Inerfeld, L.E. Braverman and A.G. Vagenakis, 1979. The role of sulfhydryl groups on the impaired hepatic 3,3,5-triiodothyronine generation from thyroxine in the hypothroid, starved, fetal and neonatal rodent. J. Clin. Invest, 63: 516-524.
10. Klandorf, H. and S. Harvey, 1985. Food intake regulation of circulating thyroid hormones in domestic fowl. Gen. Comp. Endocrinol, 60: 162-170.
11. Davidson, M.B. and I.J. Chopra, 1979. Effect of carbohydrate and noncarbohydrate sources of calories on plasma 3,6,3'-triiodothyronine concentrations in man. J. Clin. Endocrinol. Metab, 48: 577-581.
12. Mitchell, M.A. and A. Raza, 1986. The effects of glucagon and insulinon plasma thyroid hormone levels in fed and fasted domestic fowl. Comp. Biochem. Physiol. A Comp. Physiol., 85: 217-223.
13. Volpe, J.J. and P.C. Marasa, 1975. Hormonal regulation of fatty acid Synthetase, acetyl-CoA carboxylase and fatty acid Synthetase in mammalian adipose tissue and liver. Biochim. Biophys. Acta, 380: 454-472.
14. Berdanier, C.D. and D. Shubeck, 1979. Interaction of glucocorticoid and insulin in the responses of rats to starvation-refeeding. J. Nutr., 109: 1766-1771.
15. Sivitz, W.I., S.L. Desautel, T. Kayano, G.I. Bell and J.E. Pessin, 1989. Regulation of glucose transporter messenger RNA in insulin-deficient states. Nature Lond, 340: 72-74.
16. Mueckler, M., 1990. Family of glucose-transporter genes. Implications for glucose homeostasis and diabetes. Diabetes, 39: 6-11.
17. Clarke, S.D., D.R. Romsos and G.A. Leveille, 1976. Specific inhibition of hepatic fatty acid synthesis exerted by dietary linoleate and linolenate in essential fatty acid adequate rats. Lipids, 11: 485-490.
18. Dallongeville, J., A. Leroy and J. Fruchart, 1996. Immunological approach to lipoparticle metabolism. J. Clin. Ligand Assay, 19: 163-167.
19. Griffin, H.D., K. Guo, D.Windsor and S.C. Butterwith, 1992. Adipose tissue lipogenesis and fat deposition in leaner broiler chickens. J. Nutr., 122: 363-368.
20. Bensadoun, A. and A. Rothfield, 1972. The form of absorption of lipids in the chicken, Gallus domesticus. Proc. Soc. Exp. Biol. Med., 41: 814-817.
21. Azain, M.J., 2004. Role of fatty acids in adipocyte growth and development. J. Anim. Sci., 82: 916-924.
22. Pettigrew, J.E. and R.L. Moser, 1991. Fat in Swine Nutrition. In Swine Nutrition. E.R., Miller, D.E. Ullrey and A.J. Lewis, Ed. Butterworth-Heineman, Stoneham, M.A., pp:133-145 .
23. Allee, G.L., 1985. The interaction of dietary protein and energy on swine performance. In proc. of the georgia nutr. Conf. for the feed industry, atlanta, pp: 136-141.
24. Gurr, M.J., 1992. Role of fats in food and nutrition. Elsevier applied Science. London and New York.
25. Wiseman, J., 1984. Assessment of the Digestible and Metabolizable Energy of Fats for Non-ruminants. In: J. Wiseman. (Editor), Fats in Animal Nutrition, Buttworths. London.
26. Bee, G., A. Zimmermann, R. Messikommer and C. Wenk, 1994. EinfluB unterchiedlicher Fettzulagen aufdas Fettsaurenmuster im Chymus von Schweinen. Tagung Schweine und Geflugelemahrung. Halle/Saale, pp: 72-75.
27. Krogdahl, A., 1985. Digestion and absorption of lipids in poultry. J. Nutr., 115: 675-685.
28. Zollitsch, W., W. Knaus, F. Aichinger and F. Lettner, 1997. Effects of different dietary fat sources on performance and carcass characteristics of broilers. Anim. Feed Sci. Tech., 66: 63-73.
29. Crespo, N. and E. Esteve-Garcia, 2002. Dietary polyunsaturated fatty acids decrease fat deposition in separable fat depots but not in the remainder carcass. Poult. Sci., 81: 512-518.
30. Brown, M.S. and J.L. Goldstein, 1999. Aproteolytic pathway that controls the cholesterol content of membranes, cells and blood. Proc. Natl. Acad. Sci. U.S.A, 96: 11041-11048.
31. Banerjee, D., C.M. Redman, 1984. Biosynthesis of high density lipoprotein by chicken liver: conjugation of nascent lipids with apoprotein A1. J. Cell Biol., 99: 1917-1926.
32. Hermier, D., 1997. Lipoprotein metabolism and fattening in poultry. J. Nutr., 127: 805-808.
33. Griffin, H.D., G. Grant and M. Perry, 1982. Hydrolysis of plasma triacylglycerol-rich lipoproteins from immature and laying hens (Gallus domesticus) by lipoprotein lipase *in vitro*. Biochem. J., 206: 647-654.

34. Leclercq, B., 1989. Possibilites dobtention et interet des genotypes maigres en aviculture . INRA Prod. Anim., 2: 275-286.
35. Griffin, H.D. and C.C. Whitehead, 1985. Identification of lean and fat turkeys by measurement of plasma very low density lipoprotein concentration. Br. Poult. Sci., 26: 51-56.
36. Kouba, M., D. Catheline and B. Leclercq, 1992. Lipogenesis in turkey and checkins : Study of body composition and liver lipogenic enzymes activities. Brit. Poult. Sci., 33: 1003-1014.
37. Kelley, J.L. and P. Alaupovic, 1976. lipid transport in the avian species. Part1. solation and characterization of apolipoproteins and major lipoprotein density classes of male turkey serum. Atherosclerosis, 24: 155-175.
38. Mossab, A., M. Lessire, J.M. Hallouis and D. Hermier, 1999. Using Dietary Fat to Improve Polyunsaturated Fatty Acid Content of Poultry Meat. In: Cavalchini, L.G., Baroli, D. (Eds), the Fourteenth European Symposium of the Quality of Poultry Meat, 09/19-23. WPSA, Bologna, Italy, pp: 159-166.
39. Hermier, D., M.J. Chapman and B. Leclercq, 1984. Plasma lipoprotein profile in fasted and refed chickens of two lines selected for high or low adiposity. J. Nutr., 114: 1112-1121.
40. Oku, H., M. Ishikawa, J. Nagata, T. Toda and I. Chinen, 1993. Lipoprotein and apoprotein profile of Japanese quail Biochim Biophys Acta, 1167: 22-28.
41. Chen, S.E., D.W. Long, K.E. Nestor, R.L. Walzem, V.L. Meuniot, H. Zhu, R.J. Hansen and W.L. Bacon, 1999. Effect of divergent selection for total plasma phosphorus on plasma and yolk very low density lipoproteins and plasma concentrations of selected hormones in laying Japanese quail. Poult Sci., 78: 1241-1251.
42. Uyanik, F., M. Eren, B.K. Guclu and N. Sahin, 2005. Effects of dietary chromium supplementation on performance, carcass traits, serum metabolites and tissue chromium levels of Japanese quails. Biol. Trace Elem Res., 103: 187-197.
43. Girish Kumar, V., K.P. Surendranathan, D.R. Gayathri Devi and M.R. Belwadi, 2005. Effect of alliums on total lipid in plasma and tissues of Japanese quails (Coturnix coturnix japonica) with respect to age and sex. Indian J. Exp. Biol., 43: 187-91.
44. Hammad, S.M., H.S. Siegel and H.L. Marks, 1998. Total cholesterol, total triglycerides and cholesterol distribution among lipoproteins as predictors of atherosclerosis in selected lines of Japanese quail. Comp Biochem Physiol. A Mol. Integr. Physiol., 119: 485-492.