J.King Saud Univ., Vol. 13,*Agric. Sci.* (1), pp. 91-113, Riyadh (A.H. 1421/2001)

Review Article

The Role of Lipoproteins in Lipid Metabolism of Chickens

Tarek M. Shafey
Department of Animal Production, College of Agriculture,
King Saud University, Riyadh, Saudi Arabia

(Received 26/11/1419; accepted for publication 12/8/1420)

Abstract. This review addresses the role of lipoproteins in regulating lipid transport and deposition in chickens. The formation, secretion, classes, structure, function and interrelationship of the various plasma lipoproteins are reviewed. The compiled facts reveal the features of the functional role of lipoprotein in lipid metabolism as: (1) the metabolism of lipoproteins is dependant on their structure and the activities of various enzymes; (2) plasma lipoproteins are important factors not only in the mechanisms that control the development of adipose tissues during growth but also in the distribution of lipid to different adipose tissue depots. (3) in some cases, these lipoproteins are targeted to specific tissues (i.e. growing oocytes) by apoproteins found on their surface that interact with high affinity cell surface receptors; (4) differences in plasma lipoprotein profiles among different classes of chickens may reflect the differences in the physiological states of these birds. An important characteristic of chicken plasma lipoproteins is that their concentration changes under different physiological states. This offers exceptional opportunities for comparison studies.

Keywords: chickens, lipoproteins, apoproteins, VLDL, LDL, HDL, lipoprotein receptor.

Introduction

The liver is the major site of lipid synthesis in birds [1,2]. Plasma lipoproteins serve as vehicles for the transport of these lipids to adipose tissues and other extra hepatic tissues. The metabolism of plasma lipoproteins is dependent on their structure and on the activities of various enzymes. The major lipids transported through lipoproteins are triglyceride (TG) and cholesterol (C). Depending on the metabolic state of the chicken, most of the TG is transported either to adipose tissues and ovaries to be stored as lipid or to the cardiac and skeletal muscle to be used as energy.

Considerable information on the nature of the circulatory lipoprotein system of chickens has been accumulated during the last 20 years. Recent knowledge of the physiology of plasma lipoproteins indicates that they are an important factor not only in the mechanisms that control the development of adipose tissues and the distribution

of lipid to different adipose tissues' depots but also they are precursors for yolk lipoprotein that require for the deposition into the developing follicles [3]. Since the form of the lipoprotein is an important part determinant of the transfer of lipid, this review focuses on the transport of lipids in chickens and the mechanisms by which lipoproteins influence the metabolism and deposition of lipids in chickens.

1. Lipoprotein structure

The transport of lipid fractions from sites of absorption and synthesis to sites of utilization requires a system for the production of water-soluble complexes of water insoluble lipids in the aqueous medium of blood plasma. This system was achieved by the formation of lipid and protein complexes that are known as lipoproteins. Lipoproteins are classified and separated according to their density (ultracentrifugation), size (gel filtration), net surface charge (electrophoresis), and other properties (precipitation techniques, affinity columns). The most common classification of plasma lipoproteins is based on their density (Table 1).

Table 1. Chicken plasma lipoproteins

Density range (g/ml)	Lipoprotein fraction	Abbreviation	Ref[1]
d < 1.006	Portomicrons	PM	[3]
d < 1.006	Very low density lipoproteins	VLDL	[4]
1.006 <d< 1.063	Low density lipoproteins	LDL	[4]
1.063 <d< 1.21	High density lipoproteins	HDL	[4]

[1] Reference

Lipoproteins have a common structure with a hydrophobic core of TG and esterified cholesterol (EC) solubilized by a monomolecular film of phospholipid (PL), free cholesterol (FC) and apoproteins [5-7]. This structure of lipoprotein has two important characteristics: Firstly, the size of the lipoprotein particle increases with the relative amount of apolar lipids, since the surface to volume ratio decreases when the particle size increases, and secondly, to meet the requirement of a minimal surface to volume ratio, they are expected to be spherical. The proteins on the surface of a lipoprotein are called apolipoproteins or apoproteins. The main functions of apoproteins are to help solublize EC and TG by interacting with PL; to regulate the reaction of these lipids with enzymes such as lipoprotein lipase (LPL) and hepatic lipase and to bind to cell surface receptors and thus respond to specific sites of uptake and rates of degradation of other lipoprotein constituents. The major classes of plasma lipoproteins are TG-rich lipoproteins (namely, portomicrons (PM); very low density lipoproteins (VLDL), low density lipoproteins (LDL), high density lipoproteins (HDL) and very high density lipoproteins (VHDL) (namely, vitellogenin (VTG)). PM and VLDL size and density distribution overlap, therefore to obtain VLDL, animals or birds must be fasted for sufficient time to allow dietary PM to be cleared from their plasma. These lipoproteins will be discussed below.

1.1 TG-rich lipoproteins

1.1.1 PM

During digestion, lipids are partially hydrolyzed by lipases in the lumen and emulsified with bile acids and are then absorbed into the mucosal cell where resynthesis occurs. The assembly of TG-rich lipoproteins begins with the resynthesis of TG that takes place in the endoplasmic reticulum of the mucosal cell. The TG appears in the form of droplets, which are stabilized by PL, proteins (P) plus free (F) and EC to form PM. In the avian species, the lymphatic system is poorly developed, the villus core being uniformly rather than peripherally occupied with a capillary network and containing no central lacteral. Intestinal lipid is thus absorbed by way of the mesenteric portal system as PM [4,8]. PM carries absorbed dietary lipid from the small intestine to the liver. Avian PM thus corresponds to chylmicron in mammals [4].

The newly formed PM particles of roosters are larger than those of newly formed VLDL and they are marginally higher in TG (79 vs. 76 %) and C (7 vs. 5%) and are lower in P (8 vs. 11%) and PL (6 vs. 8%) [9]. PM particles of immature and laying hens have similar size (~ 155 nm) and lipid composition (TG, 89%; PL, 6.7% and C, 3.8%) [3]. This is probably because they are not involved in yolk deposition. PM particles are larger than VLDL particles (155 vs. 45 nm) and they have a higher content of TG (89 vs. 63 %) and are lower in PL (6.7 vs. 20 %) and C (3.7 vs. 11.3 %). Estimates of the diameter of PM particles vary according to the method employed. By electron microscopy, the diameter of PM particle was found to be about 155 nm for immature and laying hens [3,4]. Using a flotation rate technique, Bensadoun and Rothfeld [4] found that the diameter of PM isolated from functionally hepatectomised roosters injected with Triton WR-1339 was also about 155 nm. In a later study, Bensadoun and Kompiang [10] reported that the diameter of newly secreted PMs from roosters injected with anti-LPL anti-serum was about half that reported previously. Using sucrose density gradient, a newly formed PM particle of roosters has a median diameter of 85 nm [9].

PMs form a small proportion (about 1%) of the circulated lipid in laying hen [11]. They are rapidly metabolized from circulation with a half-life of 3-4 minutes [10]. PMs have a shorter half-life in the plasma than VLDL. However, the accumulation of PM in the plasma can vary according to the composition of the diet and the rates of lipid transport. Feeding diets rich in fat is known to depress hepatic lipogenesis in birds [12]. Thus the plasma of birds fed on a high fat diet contains a greater proportion of circulated PM [4] than does plasma from birds fed on a low fat diet, whereas most of the plasma TG of birds fed the low-fat diet exists as VLDL [3]. Griffin *et al.* [3] suggested that the accumulation of PM in the plasma of laying hen, when compared with that of immature hens, is caused by the lower tissue activity of LPL, although PM from immature and laying hens is similarly susceptible to hydrolysis. Bensadoun and Rothfeld [4] found that 50% of infused fatty acid was recovered in the liver after 30 minutes. It appears that the liver concentrates PM. The catabolism and removal of PM from the circulation will be discussed later.

1.1.2 VLDL

Most lipids deposited in the egg yolk and bodily adipose tissue of chickens is synthesized in the liver and transported in the blood as VLDL [13]. VLDL mainly carry TG from the liver to other organs. VLDL is the major lipoprotein particle produced by the liver of laying hen and oestrogenised roosters. The chemical compositions and major apoproteins of VLDL are shown in Tables 2 and 3. Kompiang *et al.* [9] found that the median particle size of newly formed VLDL of roosters determined by sucrose density gradient is 50% larger than that of circulating LDL. The diameters of newly secreted VLDL particles vary in size from 200 to 1400 Å. Twenty percentages of the particles have diameters ranging between 800 and 1400 Å. At the other extreme of the spectrum, 20% of the particles exhibit diameters between 200 and 500 Å. The newly formed particles of VLDL have a higher content of TG and FC:EC ratio than those of circulated VLDL control (76 vs. 46% and 2.5 vs. 1.2, respectively) and are lower in PL (8 vs. 27%) and C (5 vs. 14%). Using the electron microscopy, Evans *et al.* [30] found that the mean diameter of plasma VLDL of laying hen (d<1.01) particle was about 261 Å with a wide range (215-717 Å) of particle size. Chapman *et al.* [14] showed that chicken VLDL migrated as a single band. VLDL particles were relatively homogeneous in size. Their mean diameter was 340 Å and the range of particle size was 270-540 Å. About 80% of VLDL particles were Sf (floating coefficient) 20-100 and 20% of particles of Sf>100. Griffin *et al.* [3] found that VLDL particles of the laying hen were smaller in size than those of the immature hen (30 vs. 58 nm on average) and they had a higher content of TG (67 vs. 60), PL (25 vs. 17%), and EC (0.8 vs. 0.6), but were lower in C (6.6 vs. 16%) and C:PL ratio (0.3 vs. 1%). Griffin *et al.* [3] suggested that the small size of laying hen VLDL particle facilitates their passage across the oocyte wall into the developing oocyte. The smaller VLDL size of laying hens is an important modification to their function as a yolk precursor [31].

Table 2. Chemical composition of chicken plasma lipoproteins[1]

	VLDL			LDL			HDL			
Chemical composition %[2]	**R[3]**	**LH[3]**	**NLH[3]**	**R[3]**	**LH[3]**	**NLH[3]**	**C[3]**	**R[3]**	**LH[3]**	**NLH[3]**
P	10.9-12.9	12.3-15.5	14	14.5-18	18-22.5	18	54.9	43.9-48.7	44	43
Lipid	87.1-89.1	84.5-87.7	86	82-85.5	77.5-82	82	45.1	51.3-56.1	56	57
C	8.8-13.6	4.6-5.4	11.4	19.9-38.2	-	94	-	13.7-16.2	13.8	18.2
EC	-	-	-	-	28.6-34.9	-	13.7	12-16	-	-
FC	-	-	-	-	8.3-13.4	-	2.4	5-6	-	-
PL	19.7-27.3	16.8-25.2	18.4	25.6-29.7	18.8-30.5	30.5	27.8	22.9-34.5	32.1	27.5
TG	46.1-57.6	55.7-66.3	55.3	21.6-30.5	11.3-40.4	40.4	1.1	3.1-12.6	8.2	11.6

[1] Lipoproteins(VLDL=very low density lipoproteins; LDL=low density lipoproteins; HDL=high density lipoproteins).

2 Chemical composition (P=protein; C=total cholesterol; EC=cholesterol ester; FC=free cholesterol; PL=phospholipid; TG=triglyceride)

[3] Class of chicken (R=roosters; LH=laying hens; NLH=non-laying hens; C=chickens).
Data is summarized from the research of 14-21.

Table 3. Major apoproteins in chickens

Lipoprotein	Density, g/ml	Class of chicken [1]	Apoprotein	M W [2]	% of Tapo[3]	Concentration	Ref [4]
VLDL	d<1006	H	B	250,000	50	26 ug/ml $^{-1}$	[14, 22]
			VLDL-II	9,500	45	1-40 ng/ml $^{-1}$	[23-25]
		IH	B	250,000	62		[26]
		R	VLDL-II	350,000		27 mg/ml	[23]
	d<1006	ER	B	350,000	54		[27]
			VLDL-II	9,500	46	28 mg/ml	[14,23, 25-26]
LDL	d=1006-1063	H	B	250,000	75		[14]
	d=1024-1045	H	B	250,000	80		[14]
	d=1006-1063	IH	B	250,000	90		[26]
			AI	28,000	10		[26]
HDL	d=1063-121	H	AI	28,000			[25]
	d=1063-125	IH	AI	28,000	65		[26]
		R	AI	26,000	90	50-145 mg/ml	[17, 24, 28]
		ER	AI	26,000		48-150 mg/ml	[17, 28]
VTG		R				4-8 ng/ml	[22]
		H				10-25 mg/ml	[29]

[1] H=hen; IH=immature hen; R=rooster; ER=oestrogenised rooster.
[2] Molecular weight. 3 % of total apoproteins

The nutritional state (e.g. fasted, fed) of birds influences the level, characteristics and composition of VLDL. Tarlow *et al.* [32] found that cultured liver cell derived from starved chicks decreased VLDL production. Hermier *et al.* [33] found those VLDL particles of fasted young birds were more homogeneous, with a narrow range (200-400 Å) of particle size. These VLDL particles represent endogenous hepatic synthesis from free fatty acids that released from adipose tissue. In contrast, VLDL particles of refed young birds were larger and more heterogenous (200-700 Å) and that represents a mixture of hepatic VLDL and PM modified after passing through the liver. VLDL of fasted young birds contained low TG (~ 42-45 % by weight) and high EC (~ 15%) and P (~ 22-27%), whilst VLDL of refed young birds contained substantially more TG (~ 59%) and less EC (~11%) and P (~11%) than those of fasted birds. Whilst overfeeding young chickens increased plasma VLDL level [34]. The liver responds to changes in lipogenic state of birds by varying VLDL secretion in two ways: (1) altering the size of the VLDL particles, and (2) changing the lipid composition of the VLDL particles [3].

In broiler chickens, VLDL plus LDL contain between 56% and 90% of total plasma TG [35]. The possibility of using plasma VLDL concentration for selecting leaner chickens has been demonstrated by Griffin et al. [35] and Griffin and Whitehead [36] who found a positive correlation between body fat content and plasma VLDL concentration in broiler chickens. They used plasma VLDL concentration as an indicator for estimating fatness in birds. Divergent selection of a commercial strain for plasma VLDL concentration for seven generations has produced lean and fat lines of chickens with a greater than six fold differences in plasma VLDL concentration

[37-38]. Griffin *et al.* [39] found that the differences in plasma VLDL concentrations between the lean and fat lines of broiler chickens were primarily due to markedly different rates of hepatic VLDL production and the selection for these lines of chickens has made a major effect on partitioning of VLDL-TG between adipose and other tissues. They proposed that a direction of fatty acids to oxidation rather than VLDL synthesis in the liver of birds of the low-VLDL line (lean chickens) is a major cause for their low rate of VLDL secretion and makes an important contribution to improved efficiency of protein utilization. Also, they found that the divergent genetic selection has produced lean and fat chickens with a consistent difference in the activities of lipoprotein lipase (LPL) and B-hydroxybutyrate in leg muscle and heart. These activities were greater in the low-VLDL line than in the high-VLDL line (fat chickens). Differences in LPL activity between the two lines may be important in determining the fate of circulated VLDL. The increase in LPL activity in the muscle of the lean-line may increase the utilization of VLDL-TG at the expense of the deposition of TG in adipose tissue. It appears that the VLDL concentration of plasma and the LPL activity of the adipose tissue are important factors that influence body fatness.

Chapman *et al.* [14] established the presence of a high molecular weight (MW) component(s) as a major component of chicken's apo-VLDL. They reported that serum VLDL of chickens contains a high MW component (>250 000) like an apo-B and up to seven lower MW components of 27 000, 18 500-21 400, 13 500-14000, 8500 and 4000. These lower MW components constitute about 50% of VLDL. Data on the high MW component (apo-B) agrees with Chan et al. [40]. However, Perry *et al.* [41] reported that apo-B formed about 35 and 12% of the total protein of VLDL of laying and immature hens, respectively. Kudzma *et al.* [26] suggested that VLDL from immature hen plasma contains at least six major apoproteins, while those from the plasma of laying hens or oestrogenised roosters contain two apoproteins [27,40]. One of these, the high MWs apo-B, occurs in both immature and laying hen VLDL. The other, apo-VLDL-II (VLDL-II) is synthesized only in response to oestrogens [40-43].

The mechanism of oestrogen action in the liver consists of an increase in the synthesis of VLDL-TG [44-45] and protein [46] through an increase in the biosynthetic rate of VLDL in cells previously committed to its production and a recruitment of new hepatocytes previously uninvolved in its production [23-24, 47-48] and a decrease in the fractional removal rate of TG [23-24,45,49]. Using oestrogen-treated chick liver cells, Miller and Lane [50] observed that the newly formed VLDL contained apo-B, VLDL-II and four other apoproteins. Whereas VLDL isolated from the plasma of oestrogen-treated chicks contained only apo-B and VLDL-II. They suggested that apoproteins that transferred off VLDL following secretion had relatively low affinity for VLDL compared to apo-B and VLDL-II.

They concluded that the apoprotein composition of assembled VLDL is influenced by the relative rate of apoprotein synthesis and possibly by the competition of apoprotein for binding to VLDL due to their inherent differences in affinity for VLDL particles.

The apo-B is similar to the apo-B100 of mammalian's VLDL [14,26]. Apo-B is synthesized mainly in the liver and to a lesser extent in the intestine and the kidney [51]. Using oestrogen-induced liver cell culture, Siuta-Mangano *et al.* [52] found that apo-B is assembled entirely on the polysome as a contiguous 350,000-dalton polypeptide rather than by post translational cross-linking of small peptide precursors. The secretion of this protein is not completely under the influence of oestrogen secretion, but the amount of apo-B secreted may be increased in the presence of oestrogen [53]. Capony and Williams [54] found that the basal level of apo VLDL-B synthesis is 2-2.5% of total liver protein synthesis. However this level can be raised up to 15% by the oestrogen treatment. In embryonic liver, apo-B synthesis was increased by about three folds in the presence of oestrogen.

VLDL-II is synthesized on the rough endoplasmic reticulum of the liver of laying hens and oestrogenised roosters [24,55]. Using 4-weeks old cockerels, Lin and Cham [23,47] found that about 1-2% of hepatocytes contain VLDL-II. Perry *et al.* [41] found that about 12% of the apoprotein of immature-hen VLDL was present in a component with a similar MW to VLDL-II. However, the mobility of this protein was unchanged by omitting reducing agent and therefore its identity as VLDL-II is doubtful.

Apo-B and VLDL-II make up over 95% of the protein content of plasma VLDL of laying hen [24]. Perry *et al.* [41] found that VLDL-II form about 65% of the total protein of VLDL of laying hens. However, Chan [24] reported that VLDL-II formed 40-50% of the VLDL of laying hens and had an 82-amino acid peptide with an apparent MW of 9,500 [24-25, 41]. The function of VLDL-II is transporting lipid and protein across the oocyte into the developing egg. The plasma levels of apo-B and VLDL-II in laying hens are variable (Table 3). Laying hen serum contains an average of 1.5 mg/ml of VLDL protein [15,56], approximately 50% of which consists of VLDL-II [40] consequently the serum concentration of VLDL-II in a laying hen ranges from 600-900 ug/ml. Laying hen plasma VLDL contains about 20 times more VLDL-II molecules per particle than does immature hen VLDL [42,48]. The presence of VLDL-II on laying hen VLDL is very important in protecting VLDL particles from the lipolytic action of LPL [57]. Kudzma *et al.* [26] found that VLDL of immature hens contains a small amount of apo-AI.

Under the condition of C feeding, Kruski and Narayan [58] suggested that C metabolism is controlled mainly by the liver total C concentration. EC rich-VLDL are secreted from hepatocytes. The enrichment of VLDL C in response to diet can occur at the level of synthesis of VLDL by the liver [59] and/ or by retardation of the clearance of nascent TG-rich lipoproteins from C rich blood [60-61]. French *et al.* [62] suggested

that the accumulation of VLDL in hypercholesterolemic rabbits was caused by the inability of LPL to hydrolyse the EC present in the serum of chylomicrons, resulting in a remnant enriched with EC. The effect of the hypercholesterolemic condition on VLDL was investigated by Davis *et al.* [63] who found that the molar ratio of EC:TG varies as a linear function of the same molar ratio in the cell, indicating that the cellular availability determines the VLDL core composition. In these VLDL, the EC:TG weight ratio significantly exceeded those of normally fed chickens. This abnormal VLDL exhibit altered electrophoretic mobility. The addition of 1% C to the diet of growing chickens increased serum C by about 10 fold. Almost all this increase was accounted for by the increase in VLDL fraction. There were smaller changes in LDL and HDL. The TC:TG ratio of VLDL of chickens fed a normal diet was 46.4:32.1, whereas the addition of 1% C to their diet significantly increases this ratio to 76.5:6.0. The EC:TG ratio in hypercholesterolemic chickens was 57.2:6. EC was not measured in the control fed chickens. Increasing the amount of EC in the core results in a compensatory decrease in TG, suggesting there is a competition between these two hydrophobic lipids for a finite core volume. One reason why C-rich diets increase plasma C concentration is that EC produced in the liver from ingested C replaces some of the TG in the VLDL core.

1.1.3 The catabolism of TG-rich lipoprotein

The catabolism of TG-rich lipoprotein is a prerequisite for the subsequent removal of these particles from the circulation [9,10,16]. The removal of PM and VLDL from the circulation begins with hydrolysis of the core TG by LPL. These results in the liberation of large amounts of free fatty acids that are either taken up in adjacent tissues or recirculated by albumin. As TG in the core of PM is removed, the particle shrinks in size and some of its excess surface materials (PL and apoproteins) are shedded as discs or vesicles and transferred to higher density lipoproteins, particularly HDL [16]. Finally PM and VLDL particles (which are now called PM and VLDL remnants, respectively) are rapidly cleared and degraded by the liver [4]. PM remnant is reprocessed and included in the formation of VLDL that is secreted into the circulation. VLDL remnant may be taken up by the liver or metabolized through an intermediate density lipoprotein (IDL) to low density lipoprotein (LDL) [16]. The catabolism of VLDL in immature chickens generated particles of greater density and altered lipid content. Hermier *et al.* [33] suggested that the proportion of VLDL molecules transferred to LDL is small and that probably because VLDL remnants has apo-B that are efficiently taken up and catabolised by the liver, as in the rat. It appears that the main function of plasma lipoproteins is TG transport. PM and VLDL particles are synthesized and secreted by the body organs. LDL and HDL are products of the process of TG transport. LDL is the core remnant and HDL is the surface remnant of the TG-rich lipoproteins.

The clearance rate of radiolabelled VLDL in immature and laying hens was studied by Bacon *et al.* [11]. In laying hens, they found that the amount of VLDL deposited in

egg yolks was about 30%, whilst metabolic oxidation, body fat and liver deposition were accounted for 35, 24 and 11%, respectively, from circulated plasma VLDL. However, in immature hens the clearance rate was higher than that of laying hens. The slower clearance rate of VLDL in laying hens was attributed to their properties. As mentioned earlier that VLDL of laying hens contains apoprotein VLDL-II that protects the VLDL from the action of LPL. The role of LPL in the metabolism of lipoproteins will be discussed later.

1.2 LDL

LDL is isolated between d 1.006 and 1.063. Most of plasma LDL fraction is a catabolic product of VLDL, therefore should be primarily of liver origin [16]. The chemical composition of chicken plasma LDL is shown in Table 2. LDL is the main TG-carrying lipoprotein class in chickens and immature hens. The main function of LDL is transporting C to peripheral tissue [64]. The nutritional state of young bird did not affect the level of LDL in the plasma [33].

Electrophoresis in agarose gel showed that chicken plasma LDL migrated as a single narrow band [14]. The mean diameter of LDL particles isolated in a density of 1.006-1.063 g/ml was 237 Å with a range in particle size between 160 and 380 Å . About 3% of particles in this fraction were of VLDL size (>350 Å). However, the diameter of LDL particles isolated in a density of 1.024- 1.045 g/ml was 234 Å with a range (180- 280 Å) of particle size. About 75% of these particles were between 200 and 260 Å. Using electron microscopy, Evans et al. [30] found that the mean diameter for LDL (d=1.01-1.06) particle is about 264 Å with a wide range (60-496 Å) of particle. The major proportion (>70%) of the LDL tended to be distributed within Sf range 12-20. The concentration of low density substances in Sf range 0-12 was <50 mg/100 ml serum. Chapman *et al.* [14] found that serum LDL of chickens contains a high MW component (>250 000, as a major component) like an apo-B and up to seven lower MW components of 27 000, 18 500-21 400, 13 500-14000, 8500 and 4000. These lower MW components constitute about 25% of LDL. Data on the high MW component (apo-B) agree with Hearn and Bensadoun [65] and Lusky *et al.* [46]. Perry *et al.* [41] found that apo-B formed about 80% of immature-hen LDL.

Plasma LDL from oestrogenised roosters contains apo-B and apo VLDL-II [66], whilst plasma LDL from roosters and immature-hens contains only apo-B as the major apoprotein (Table 3). Perry *et al.* [41] found that about 3% of immature-hen LDL was present in a component with a similar MW to VLDL-II. However, the mobility of this protein was unchanged by omitting reducing agent, therefore its identity as VLDL-II is doubtful. Kudzma *et al.* [26] found that apo- AI comprises about 10 % of total apoproteins LDL of immature hens. This finding may suggest that apo-AI is not completely transferred to HDL following the hydrolysis of VLDL particles by LPL.

1.3 HDL

The transport of plasma lipid in immature hens and male chickens appears to be similar to that in mammals where HDL is the major class of lipoproteins in the plasma [13]. Plasma HDL function is transporting C from peripheral tissues to the liver for catabolism [67]. The composition of chicken plasma HDL is shown in Table 3. HDL contain high proportions of C, EC and PL. TG forms a small proportion of total lipid of HDL; however, the high proportion of HDL in plasma of immature birds indicates that HDL-TG represents a major contributor to the circulating TG. The physical properties of rooster HDL were studied by Kruski and Scanu [17] who found that HDL has a MW, sedimentation coefficient and diffusion coefficient of about 173 000, 3.99 S and 4.36 10^{-7} cm^2, respectively. Serum HDL appears to be homogeneous in size with a mean diameter of 95 Å. However, Behr *et al.* [16] reported that HDL consisted of round particles with a mean radius of 54 Å. However these particles are reduced in size by about 20% by anti LPL treatment. Using electron microscopy, Evans et al. [30] found that the mean diameter for HDL (d=1.06-1.20) particle is about 120 Å, with a range (60-179 Å) of particle size.

The nutritional state (e.g. fasted or fed) of young chickens did not affect the level of HDL in the plasma [33]. In meat chickens and immature hens, HDL is about 92% of the total plasma lipoproteins [26] and that 90-93% of the protein content of HDL is composed of one apoprotein (apo-AI) with a MW of 28,000 [17,68]. However, human HDL contains two apoproteins, apo-AI and apo-AII. Chicken HDL apo-AI is similar to human HDL apo-AI except that it has isoleucine, higher levels of stearic acid and about 20% more α-helix content than human apo-AI [17,26,68]. Rooster HDL contains another apoprotein, represents about 10% of total protein in HDL, with a MW of 15,000 and migrates in urea-polyacrylamide gel to a similar position as human A-II [17,23]. Apo-AI has four isoforms [69] and contains 248 amino acids [70]. Each HDL particle contains three molecules of apoA-1 calculated from Scanu [18]. The major apoproteins of HDL are shown in Table 3.

Almost all HDL disappears upon maturation of the hen when the VLDL becomes the prevalent lipid carrier of plasma protein. The administration of oestrogen to birds reduces the level and alters the apoprotein compositions of HDL. The amount of apoA-1 is reduced to about 5%, while VLDL-II becomes a major component of apo HDL [26]. Using White leghorn hens, Jackson et al. [71] found that apo HDL contains apoA-1 and an increased amount of VLDL-II. However, Chan et al. [28] and Wiskocil et al. [42] found that neither the rate of synthesis of apoA-I nor the number of hepatocytes that synthesising this protein was affected by oestrogen treatment. These findings may suggest that the levels of apoproteins in HDL are determined by the rate of its removal from the circulation than by its synthesis.

Studies have shown that apoprotein synthesis takes place on the cell rough endoplasmic reticulum fraction and the synthesis of that lipid occurs on the cell smooth endoplasmic reticulum fraction, but only achieves the buoyant density of HDL when it reaches the Golgi apparatus [70]. However, these authors found biochemical and

morphological differences between Golgi complexes HDL and serum HDL suggested that the nascent HDL might not be fully assembled in the Golgi apparatus. The Golgi HDL was more heterogeneous and larger and contained less P and TG and more PL than those of HDL serum were. The heterogeneity in size of lipoprotein particles isolated from Golgi apparatus may reflect different stages of lipoprotein maturation. Nascent HDL are secreted in disc-like lipid bilayers, higher in relative PL and C content than the typical spherical particles found in plasma. The transformation to spherical particles apparently takes place within circulation, perhaps at cells' membranes [72]. Apo-AI is known to be synthesized also in chicken liver, kidney, aorta, peripheral arteries, veins and adipose tissue [69] and breast muscle [73]. Because many peripheral tissues synthesize apo-AI, Blue *et al.* [69] postulated that the apo-AI synthesized in peripheral tissues may play a role in the movement of lipid from those tissues to the liver for metabolism and elimination. They suggested the following potential roles for apo-AI; (1) apo-AI may serve to transport C from the cell in the form of an apo-AI containing lipoprotein; (2) peripheral apo-AI may serve as an extracellular acceptor of C released from cell membrane; (3) Apo-AI may serve as an activator of Lecithin:cholesterol acyltransferase (LCAT). LCAT is an enzyme required for net transfer of C from cultured cells to plasma lipoproteins' receptors. In this way HDL may facilitate the transfer of C from peripheral tissues back to the liver, a process referred to as reverse C transport, where C is converted to bile acids and excreted [74]. In mammals, HDL contains apo E which plays a major role in C transport. The presence of apo-AI in all density classes of chickens may suggest that apo-AI may have additional functions similar to those of mammalian apo-E that is not found in chicken lipoproteins [75]. In rooster HDL, Kruski and Scanu [17] found a component of MW 15 000 and a group of fast migrating peptides, resembling the human apo-C peptides.

1.4 VHDL (VTG)

VTG is a lipophosphoglycoprotein found mainly in the blood of laying hens [76] and oestrogenised roosters [77]. The synthesis of VTG is an oestrogen dependent process. However, using a radioimmunoassay, Blue and Williams [22] found a wide range of concentration (4-100 ng/ml) of VTG in the serum of nonoestrogenised roosters. This is may due to the non-specific radioimmunoassay used by the investigators. VTG contains relatively low content of lipid (15-20%) and that corresponds to VHDL with hydrated density more than 1.21 g/ml [13]. Like VLDL, VTG carries lipid synthesized in the liver to the growing oocytes. The concentration of VTG in the blood of laying hen is about 10-25 mg/ml [78].

VTG is synthesized in the liver as a polypeptide with a MW of approximately 240,000 [77,79]. This polypeptide is phosphorylated and glycosylated then associated with lipid and secreted into the blood as a lipoprotein complex, lipovitellinogenin, having the MW of approximately 480,000. This lipovitellinogenin is composed of two identical subunits with a MW of approximately 240,000 and each unit contains two different lipovitellins, α and β lipovitellins [77,80]. The lipovitellin is composed of 20%

(by weight) lipid and 80% protein, 10% of which is serine. Most of the lipid present on the VTG molecule is found in lipovitellin. This lipid consists of approximately 60% PL and 40% TG plus C [80].

VTG exists in the blood in three forms, VTGI, VTDII and VTGIII with a weight ratio of 0.33: 1.0: 0.08 [81]. However, these forms of VTG differ in their responses to hormone induction [81-82]. VTG is the precursor for the yolk granules. The granules account for about 20 % of the yolk solids. This lipoprotein is transported to the growing oocytes and cleaved into two major yolk lipoproteins, lipovitellin and phosvitin [29,77].

2. Lipoprotein receptors, enzymes and related protein

2.1 VLDL and LDL receptors

Lipoprotein receptors and yolk precursors: VTG and VLDL, the major yolk precursors are synthesized in the liver of laying hens and transported to the growing oocytes. VLDL and VTG particles are transferred by endoytosis at the same time. They have to pass through different layers in the follicle wall before they are taken up into the yolk. The layers are thecal layer, basal lamina and granulosa cells. The thecal layer has the ability to exclude any large lipoprotein particles from entering. Following the release of lipoprotein particles from thecal capillaries, the lipoprotein particles firsts cross the basal lamina, then pass through intracellular gap in the granulosa cell monolayer, and finally reach the oocyte plasma membrane where they bind to an a specific surface receptor. The binding of VTG and VLDL to the oocyte plasma membrane is mediated by the same receptor [83] and via the apo-B apoprotein [66,84]. Apo-B plays a key role in oocyte receptor recognition. The receptors are localized in discrete region of the membrane called coated pits. The coated pits are basketlike structures and contain the protein clathrin, which consists of heavy chains (molecular weight 192,000 and light chains (molecular weight 32,000-38,000). These proteins form a fibrous network on the surface of the coated vesicle [85].

Polymerization of clathrin into a lattice along the cytoplasmic inner surface of the plasma membrane is believed to cause the pit to expand and pinch off from the membrane after the VLDL and VTG have bound to the receptors. The coated pit becomes a coated vesicle. The internalization of VLDL and VTG occurs after binding. Coated vesicles lose clathrin after internalization, which persumably recycled to the oolema [86]. These uncoated vesicles are called endosmes, fused together to form very large yolk bodies or globules or spheres and eventually fill the yolk. However, the affinity of immature and laying hen's plasma lipoproteins to the oocyte plasma membrane is pH dependent [41,87]. George et al. [88] demonstrated that chicken oocytes possess membrane receptor sites for the interaction with plasma LDL and VLDL. In laying hen, two specific receptors bind apo-B lipoprotein. One is an oocyte receptor, and the other is synthesized by somatic cells. The oocyte receptor, a 95-kDa protein, mediates the massive uptake of yolk precursors, VLDL and VTG from plasma into the oocyte [83,88]. Studies on a mutant non laying strain of birds (termed restricted ovulator (RO), suggested that the lack of deposition of VLDL and VTG into the oocytes

of RO birds can be described by the absence of the transport function mediated by the 95-kDa membrane protein [56, 89]. Barber *et al.* [90] suggested that the 95-kDa protein might be located in the plasma membrane of the growing oocyte and function as the transport receptor for lipoproteins that form 95% of the dry weight of yolk. The protein would therefore be functioning as a regulatory factor in the deposition of lipid in the oocyte. The somatic cell receptor, a 130-kDa, is involved in the regulation of cellular cholesterol homeostasis [91]. These two chicken receptors are expressed in cell-specific fashion. The oocyte 95-kDa receptor is absent from somatic cells, whilst the somatic cell 130-kDa receptor is absent from the oocyte.

Perry *et al.* [41] found that the kDa values for oocyte plasma membrane binding of lipoproteins form immature hen (mainly LDL) and laying hen (mainly VLDL) were 30 and 45 ug protein/ml, respectively and suggested that the plasma membrane receptors bind to apo-B rather than VLDL-II. Since particles of VLDL of laying hen contain at least 20 times the numbers of apoprotein VLDL-II molecules per particle than do immature hen LDL [42,48] these authors suggested that apo-B mediates the binding of both LDL and VLDL to a single receptor. George et al. [88] found that a single binding site with a kDa of 10.1 ug/ml and 13.0 ug/ml for LDL and VLDL, respectively, isolated from laying hen plasma.

2.2 Enzymes

2.2.1 Lipoprotein lipase (LPL)

LPL is an enzyme synthesized in parenchymal cells and bound to endothelial surface of extrahepatic capillaries by the attachment of heparin or heparin-like compounds [92-93]. The enzyme can be released into the general circulation by heparin injection and its activity can be assayed (postheparin) in plasma [94]. LPL is functional at the capillary endothelial surfaces where circulating lipoprotein particles are bound briefly and the TG hydrolyzed into fatty acids, which can be incorporated into fat cells. The initial interaction between LPL and TG is facilitated by the presence of a specific LPL-activator apoprotein, corresponds to apo-C-II in mammals [95]. VLDL may contain LPL-activator when secreted from the liver, or plasma HDL may transfer the protein cofactor to VLDL nascent [96]. Miller and Lane [50] speculated that the MW of LPL-activator of chickens to be about 6 kDa. Plasma VLDL of immature-hen and PM from immature and laying hens contain a substantial amount of LPL-activator, but VLDL from laying hen plasma contain very little or none [35]. They suggested that the activity of LPL-activator apoprotein might be related to HDL content of the plasma. Although they have found that HDL of immature and laying hen plasma have the same activating ability when expressed per mg of protein.

The uptake of plasma TG into extra-hepatic tissues other than the ovary is mediated by LPL. Kompiang *et al.* [9] and Behr *et al.* [16] found that the administration of anti-LPL serum to roosters caused a linear increase in the concentration of VLDL-TG and a decrease in the level of LDL and a change in the chemical composition of LDL. Whilst the level of HDL was decreased and the HDL particles became smaller and more dense

and accumulated more TG. They suggested that lipid and lipoprotein constituents leave the surfaces of VLDL during their catabolic degradation and that contribute to the mass HDL subfractions.

2.2.2 The activity of LPL and fat deposition in chickens

The activity of LPL is a major factor in the growth of adipose tissue in meat chickens [97]. It determines the amount of TG hydrolyzed and consequently regulating fatty acid uptake into adipose tissues; all TG deposited into adipose tissues must result from the action of LPL. LPL is the rate-limiting enzyme in the hydrolysis of plasma TG-VLDL [98]. A number of factors can influence the activity of LPL. These include age, environment, etc. [98]. The activity of LPL is less sensitive to nutritional conditions of the chickens [33,97]. The concentration of plasma VLDL was decreased in starved chickens, whist the activity of LPL in adipose tissue was little changed by starvation of chickens. However, the secretion rate of VLDL influenced the rates of fat deposition in tissue [33]. Griffin *et al.* [99] suggested that the rapid growth in abdominal fat pad in broilers as compared to layers is attributed to the high activity of LPL and that is very important in directing circulating TG to adipose rather than to other tissues. The higher activity of LPL in adipose tissues of fat animals is attributed to higher VLDL and insulin concentrations [100-101].

2.2.3 LPL and yolk formation

Husbands [102] found that LPL activity in the adipose tissue and heart of laying hens was lower than that in immature hens. This suggests that the lower activity of LPL in adipose tissues of laying hens depresses tissues uptake of TG and this contributed to the direction of VLDL-TG to the ovary rather than to adipose tissues. Akiba *et al.* [103] found a negative relationship between oestrogen concentration and LPL activity in laying hens. This relationship may be account for to the low clearance rate of VLDL from the circulation in laying hens when compared with that in immature hens [11]. LPL is synthesized in avian granulosa cells [104] which possesses 12 times the LPL activity of thecal cells [105] and is about one-tenth that in rooster adipose tissues [10]. LPL in granulosa cells' hydrolysis small TG-lipoproteins particles of hepatic origin (VLDL) that are transported to the yolk and cannot hydrolyzed in vivo large TG-lipoproteins particles (PM) [31]. In vitro, Griffin *et al.* [3] found that hydrolysis of VLDL from immature hens and PM from immature and laying hens proceeded rapidly until at least 40% of lipoprotein TG had been hydrolyzed. In contrast, only a part (1-15 %) of the TG of laying hen plasma VLDL was readily hydrolyzed and further hydrolysis occurred slowly. They suggested that the limited susceptibility of laying hen VLDL to hydrolysis by LPL is a major factor in ensuring transport of lipid to yolk rather to other tissues. Recently, Schneider *et al.* [57] found that the presence of VLDL-II on laying hen's VLDL protected VLDL particles from the lipolytic action of LPL. This finding agrees with Gornall and Kuksis [106] and Griffin *et al* [3] who suggested that lipoproteins are transported across the oocyte wall approximately intact.

2. 3 Lecithin:cholesterol acyltransferase (LCAT)

LCAT is an enzyme, originating in the liver. It is responsible for transesterifying C with fatty acids derived from the 2-position of lecithin [107]. Very little is known about the role of LCAT in avian lipoprotein metabolism. However, it is thought that during lipolysis, lipid transfer protein probably transfers PL from TG-rich lipoproteins to HDL. The production of PL-rich HDL particles may be followed by movement of free C from other lipoproteins into HDL providing substrate (PL and C) for LCAT [74].

3. Practical consideration

3.1 Altering C content of eggs

Despite extensive research, little progress has been made in reducing C content of eggs by dietary manipulation, genetic selection or management means [108-113]. Hargis [113] reviewed about 50 references examining the effects of manipulation of the hen's diet on egg yolk C and concluded that the inability to reduce markedly yolk C concentration is possibly due to natural selection pressures to maintain a certain level of C in the egg for use by the developing embryo. In addition, there is little evidence to suggest that a commercially significant reduction of egg yolk C content is going to occur by means of conventional nutritional manipulation or genetic selection programs. The hen has the capacity to synthesize considerably more C than it normally produces. The inhibition of C synthesis using lipid-lowering drugs such as 7-ketocholesterol did not influence egg yolk C [114]. They inhibited 43% of the activity of HMG-CoA reductase, an early rate-limiting step in C synthesize. Although C is largely synthesized in the liver and transported to the ovum via VLDL, there appears to be little relationship between blood and egg C [113]. VLDL can binds to the whole length of the oocyte plasma membrane but uptake of VLDL into yolk is determined by the availability of receptors or the rate of receptor-recycling for endocytosis. It seems more likely that the uptake of VLDL is not determined by plasma VLDL concentration.

3.2 Altering fatty acids content of eggs

It is well known that the composition of egg yolk fatty acids is a reflection of the fatty acids synthesized by the liver of a laying hen on a standard diet since the amount of egg yolk fatty acids provided by adipose tissue is about 20% [115]. Several reviews have emphasized the role of dietary factors in modifying egg yolk lipid [108,113,116]. The supplementation of vegetable oil to the diet of laying hens has modified the fatty acid composition of egg yolk [110-112,117-119]. Sunflower oil supplementation resulted in a significant reduction in the deposition of palmitic and oleic acids in egg yolk lipids, whilst stearic, linoleic and arachidonic acids contents were increased. Increasing dietary linoleic acid content may enhance the synthesis of linolenic and arachidonic acids in the liver and subsequently increase the deposition of these fatty acids in the yolk. The reduction in egg yolk palmitic and oleic acids was compensated for by the increase in stearic, linoleic and arachidonic acids [111]. Similar results were obtained from feeding other vegetable oils as well as sunflower oil to layers [110,118-119]. The increase in the unsaturated fatty acids and decrease in saturated fatty acids, in eggs from hens fed vegetable or fish oils may be of interest to consumers. Whilst, information related to the comparative influences of dietary components on lipid

transport is limited. Sunflower supplementation did not affect the composition of plasma lipoproteins of laying hens [111].

The fatty acid composition of plasma VLDL is similar to that of egg yolk [30,105]. However, the relative proportion of fatty acids in plasma and egg yolk is slightly different [105]. The concentration of linoleic acid in plasma VLDL and fatty acid of older hens were higher than that of younger age (51 vs. 27 weeks of age). The increase in linoleic acid was accompanied by proportional reductions in stearic and oleic acids. It appears that the transport of fatty acids to the yolk changes with age. Shafey *et al.* [120] suggested that the increase in linoleic acid concentration in plasma VLDL of older hens might have a stimulatory effect on albumin synthesis in the oviduct.

3.3 Lipoprotein and fat deposition in chickens

Considerable research effort is being put into reducing fat deposition in chickens. At present there is at least one approach, which might be used to manipulate carcass fat content in chickens. This approach is the selection of birds on the basis of fat content using methods such as plasma VLDL levels or adipose tissue LPL activity [37]. However, in meat chicken production, slaughter yield and the proportion of valuable parts determine the returns, whilst feed conversion determines production costs. Thus meat chicken selection program should take into account all these factors together.

References

[1] Goodridge, A.G. and Ball, E.G. "Lipogenesis in the Pigeon: in vivo Studies." *American Journal of Physiology*, 213 (1967), 245-249.

[2] Saadoun, A. and LeClercq, B. "Comparison of in vivo Fatty Acid Synthesis of the Genetically Lean and Fat Chickens." *Comparative Biochemistry and Physiology*, 75B (1983), 641-644.

[3] Griffin, H.D., Grant, C. and Perry, M.M. "Hydrolysis of Plasma Triacylglycerol-rich Lipoproteins from Immature and Laying Hens by Lipoprotein Lipase in vitro." *Biochemical Journal*, 206 (1982), 47-654.

[4] Bensadoun, A., and Rothfeld, A. "The Form of Absorption of Lipids in the Chicken, *Gallus domesticus*." *Proceedings of the Society for Experimental Biology and Medicine*, 141 (1972), 814-817.

[5] Havel, R.J., Eder, H.A. and Bragdon, J.H. "The Distribution and Chemical Composition of Ultracentrifugally Separated Lipoprotein in Human Serum." *Journal of Clinical Investigation*, 34 (1955), 1345-1353.

[6] Finer, E.G., Henry, R., Lesllie, R.B., Robertson, R.N. "NRMA Studies of Pig Low and High-Density Serum Lipoproteins. Molecular Motions and Morphology." *Biochemica et Biophysica Acta*, 380 (1975), 320-337.

[7] Edelstein, C., Kezdy, F.J., Scanu, A.M. and Shen, B.W. "Apolipoproteins and the Structural Organization of Plasma Lipoproteins: Human Plasma High Density Lipoproteins -3." *Journal of Lipid Research*, 20 (1979),143-153.

[8] Noyan, A., Lossoe, W.J., Brot, W.J. and Chaikoff, I.L. "Pathways and Form of Absorption of Palmitic Acid in the Chicken." *Journal of Lipid Research*, 5 (1964),538-541.

[9] Kompiang, I.P., Bensadoun, A. and Yang, M.W.W. "Effect of an Anti-lipoprotein Lipase Serum on Plasma Triglyceride Removal." *Journal of Lipid Research*, 17 (1976), 498-505.

[10] Bensadoun, A. and Kompiang, I.P. "Role of Lipoprotein Lipase in Plasma Triglyceride Removal." *Federation Proceedings*, 38 (1979), 2622-2626.

[11] Bacon, W.L., Le Clercq, B. and Blum, J.C. "Difference in Metabolism of Very Low Density Lipoprotein from Laying Chicken Hens in Comparison to Immature Chicken Hens." *Poultry Science*, 57 (1978), 1675-1686.

[12] Yeh, Y.-Y., Leveille, G.A. and Wiley, J.H. "Influence of Dietary Lipid on Lipogenesis and on the Activity of Malic Enzyme and Citrate Cleavage Enzyme in Liver of the Growing Chick." *Journal of Nutrition*, 100 (1970), 917-924

[13] Chapman, M.J. "Animal Lipoproteins: Chemistry, Structure and Comparative Aspects." *Journal of Lipid Research*, 21 (1980), 789-853.

[14] Chapman, J.M., Goldstein, S. and Laudat, M.L. "Characterization and Comparative Aspects of the Serum Very Low and Low Density Lipoproteins and Their Apoproteins in the Chicken (*Gallus domesticus*)." *Biochemistry*, 16 (1977), 30006-30015.

[15] Nimpf, J., Radosavljevic, J. and Schneider, W.J. "Oocytes from the Mutant Restricted Ovulator Hen Lack Receptor for Very Low Density Lipoprotein." *Journal of Biological Chemistry*, 264 (1989), 1393-1398.

[16] Behr, S.R., Patsch, J.R., Forte, T. and Bensadoun, A. "Plasma Lipoprotein Changes Resulting from Immunoligically Blocked Lipolysis." *Journal of Lipid Research*, 22 (1981), 443-445.

[17] Kruski, A.W. and Scanu, A.M. "Properties of Rooster Serum High Density Lipoproteins." *Biochemica et Biophysica Acta*, 409 (1975), 26-38.

[18] Scanu, A.M. "Ultrastructure of Serum High Density Lipoproteins: Facts and Models". In: *Symposium on High Density Lipoproteins*. A.O.C.S. Annual Meeting, St. Louis (1978), 7-12.

[19] Yu, J. Y-L., Campbell, L.D. and Marquardt, R.R. "Immunological and Compositional Patterns of Lipoproteins in Chicken (*Gallus domesticus*) Plasma." *Poultry Science*, 55 (1976), 1626-1631.

[20] Burghelle-Mayeur, C., Demarne, Y. and Merat, P. " Influence of the Sex-linked Dwarfing Gene (dw) on the Lipid Composition of Plasma, Egg Yolk and Abdominal Fat Pad in White Leghorn Laying Hens: Effects of Dietary Fat." *Journal of Nutrition*, 119 (1989),1361-1368.

[21] Allen, P.C. "Effect of Eimeria acervulina Infection on Chick (*Gallus domesticus*) High Density Lipoprotein Composition." *Comparative Biochemistry and Physiology*, 87B (1987), 313-319.

[22] Blue, M.L. and Williams, D.L. "Induction of Avian Serum Apolipoprotein II and Vitellogenin by Tamoxifen." *Biochemica Biophysica Research Communication*, 98 (1981), 785-791.

[23] Lin, C.-T. and Chan, L. "Effects of Oestrogen on Specific Protein Synthesis in the Cockerel Liver: An Immunocytochemical Study on Major Apoproteins in Very Low Density and High Density Lipoproteins and Albumin." *Endocrinology*, 107 (1980), 70-75.

[24] Chan, L. "Hormonal Control of Apolipoprotein Synthesis." *Annual Review of Physiology*, 45 (1983), 615-623.

[25] Jackson, R.L., Lin, H.-Y., Chan L. and Means, A.R. "Amino Acid Sequence of a Major Apoprotein from Hen Plasma Very Low Density Lipoproteins." *Journal of Biological Chemistry*, 252 (1977), 250-253.

[26] Kudzma, D.J., Swaney, J.B. and Ellis, E.N. "Effects of Oestrogen Administration on the Lipoproteins and Apoproteins of the Chicken." *Biochemica et Biophysica Acta*, 572 (1979), 257-268.

[27] Williams, D.L. " Apoproteins of Avian Very Low Density Lipoprotein: Demonstration of a Single High MW Apoprotein." *Biochemistry*, 18 (1979), 1056-1063.

[28] Chan, L., Bradley, W.A., Jackson, R.L. and Means, A.R. "Lipoprotein Synthesis in the Cockeral Lver." *Endocrinology*, 106 (1980), 275-283.

[29] Redshaw, M.R. and Follett, B.K. "The Pysiology of Egg Yolk Production in the Hen". In: B.M. Freeman and P.E. Lake (Eds.). *Egg Formation and Production*. British Poultry Science Ltd., Edinburgh, 1972.

[30] Evans, R.J., Flegal, C.J., Foerder, C.A., Bauder, D.H. and La Vigue, M. "The Influence of Crude Cottonseed Oil in the Feed on the Blood and Egg Yolk Lipoproteins of Laying Hens." *Poultry Science*, 56 (1977), 468-479.

[31] Griffin, H.D. and Perry, M.M. "Exclusion of Plasma Lipoproteins of Intestinal Origin from Avian Egg Yolk because of Their Size." *Comparative Biochemistry and Physiology*, 82B (1985), 321-325

[32] Tarlow, D.M., Watkins, P.A., Reed, R.E., Miller, R.S. Zwergel, E.E. and Lane, M.D. "Lipogensis and the Synthesis and Secretion of Very Low Density Lipoprotein by Avian Liver Cells in Nonproliferating Monolayer Culture." *The Journal of Cell Biology*, 73 (1977), 332-353.

[33] Hermier, D., Chapman, J. and LeClercq, B. "Plasma Lipoprotein Profile in Fasted and Refed Chickens of Two Strains Selected for High or Low Adiposity." *Journal of Nutrition*, 114 (1984), 1112-1121.

[34] LeClercq, B., Hassan, I. and Blum, J.C. "The Influence of Force-feeding on the Transport of Plasma Lipids in the Chicken (*Gallus gallus* L.). *Comparative Biochemistry and Physiology*, 47B (1974), 289-296.

[35] Griffin, H.D., Whitehead, C.C. and Broadbent, L.A. "The Relationship between Plasma Triglyceride Concentrations and Body Fat Content in Male and Female broilers - a Basis for Selection". *British Poultry Science*, 23 (1982), 15-23.

[36] Griffin, H.D. and Whitehead, C.C. "Plasma Lipoprotein Concentration as an Indicator of Fatness in Broilers: Development and Use of a Simple Assay for Very Low Density Lipoproteins." *British Poultry Science*, 23 (1982), 307-313.

[37] Whitehead, C.C. and Griffin, H.D. "Development of Divergent Lines of Lean and Fat Broiler Using Plasma Very Low Density Lipoprotein Concentration as Selection Criterion: the First Three Generations." *British Poultry Science*, 25 (1984), 573-582.

[38] Whitehead, C.C. "Selection for Leanness in Broilers using Plasma Lipoprotein Concentration as Selection Criterion." In: *Leanness in Domestic Birds*. Le Clercq, B. and Whitehead, C.C.(Eds.), pp. 41-57. London: Butterworths, 1988.

[39] Griffin, H.D., Camovic, A., Guo, K. and Peddie, J. "Plasma Lipoprotein Metabolism in Lean and in Fat Chickens Produced by Divergent Selection for Plasma Very Low Density Lipoprotein Concentration." *Journal of Lipid Research*, 30 (1989), 1243-1250.

[40] Chan, L., Jackson, R.L., O'Malley, B.W. and Means, A.R. "Synthesis of Very Low Density Lipoproteins in the Cockerel: Effect of Oestrogen." *Journal of Clinical Investigation*, 58 (1976), 368-379.

[41] Perry, M.M., Griffin, H.D. and Gilbert, A.B. "The Binding of Very Low Density and Low Density Lipoproteins to the Plasma Membrane of the Hen's Oocyte. A Morphlogical Study." *Experimental* Cell *Research*, 15 (1984), 433-446.

[42] Wiskocil, R., Bensky, P., Dower, W., Goldberger, R.G., Gordon, J.I. and Deeley, R.G. "Coordinate Regulation of Two Oestrogen-dependent Genes in Avian Liver." *Proceedings of the National Academy of Science,* USA, 77 (1980), 4474-4478.

[43] Shapiro, D. "Steriod Hormone Regulation of Vitellogenic Gene Expressions." *CRC Critical Review in Biochemistry*, 12 (1983), 187-203.

[44] Kudzma, D.J., Hegsted, P.M. and Stall, R.E. "The Chick as a Laboratory Model for the Study of Oestrogen-induced Hyperlipidemia." *Metabolism*, 22 (1973), 423-434.

[45] Kudzma, D.J., Claire, F.S., DeLallo, L. and Friedberg, S.J. "Mechanism of Avian Oestrogen-induced Hypertriglyceridemia: Evidence for Over Production of Triglyceride." *Journal of Lipid Research*, 16 (1975), 123-133.

[46] Luskey, K.L., Brown, M.S. and Goldstein, J.L. "Stimulation of the Synthesis of Very Low Density Lipoprotein in Rooster Liver by Estradiol." *Journal of Biological Chemistry*, 249 (1974), 5939-5947.

[47] Lin, C.-T. and Chan, L. "Oestrogen Regulation of Yolk and Non-yolk Protein Synthesis in the Avian Liver: An Immunocytochemical Study." *Differentiation*, 18 (1981), 105-114.

[48] Lin, C.-T., Palmer, W., Wu, J.-y. and Chan, L. "Oestrogen Induction of Very Low Density Apolipoprotein II Synthesis, a Major Avian Liver Yolk Protein, Involves the Recruitment of Hepatocytes." *Endocrinology*, 118 (1986), 538-544.

[49] Dashti, N., Kelley, J.L., Thayer, R.H. and Ontko, J.A. "Concurrent Inductions of Avian Hepatic Lipogenesis, Plasma Lipids, and Plasma Apolipoprotein B by Oestrogen." *Journal of Lipid Research*, 24 (1983), 368-380.

[50] Miller, K.W. and Lane, M.D. "Estradiol-induced Alteration of Very Low Density Lipoprotein Assembly." *The Journal of Biological Chemistry*, 259 (1984), 15277-15286.

[51] Blue, M.L., Protler, A.A. and Williams, D.L. "Biosynthesis of Apolipoprotein B in Rooster Kidney, Intestine and Liver." *Journal of Biological Chemistry*, 255 (1980), 10048-10051.

[52] Siuta-Mangano, P., Howard, S.C., Lennarrz, W.Z. and Lane, M.D. "Synthesis, Processing, and Secretion of Apolipoprotein B by the Chick Liver Cell." *The Journal of Biological Chemistry*, 257 (1982), 4292-4300.

[53] Nadin-Davis, S.A., Lazier, C.B., Capony, F. and Williams, D.L. "Synthesis of Apoprotein B of Very Low Density Lipoprotein in Embryonic-chick Liver." *Biochemistry Journal*, 192 (1980), 733-740.

[54] Capony, F. and Williams, D.L. "Apolipoprotein B of Avian Very Low Density Lipoprotein: Charateristics of Its Regulation in Nonstimulated and Oestrogen-stimulated rooster." *Biochemistry*, 19 (1980), 2219-2226.

[55] Lin, C.-T. and Chan, L. "Localization of Apo VLDL-II, a Major Apoprotein in Very Low Density Lipoproteins in the Oestrogen-treated Cockerel Liver by Immunoelectron Microscopy." *Histochemistry*, 76 (1982), 237-246.

[56] Ho, K.-J., Lawrence, W.D., Lewis, L.A., Liu, L.B. and Taylor, C.B. "Hereditary Hyperlipidemia in Nonlaying Chickens." *Archives of Pathology*, 98 (1974), 161-172.

[57] Schneider, W.J., Carroll, R., Severson, D.L. and Nimpf, J. "Apolipoprotein VLDL-II inhibits Lipolysis of Triglyceride-rich Lipoproteins in the Laying Hen." *Journal of Lipid Research*, 31 (1990), 507-513.

[58] Kruski, A.W. and Narayan, K.A. "The Effect of Dietary Supplementation of Cholesterol and Its Subsequent Withdrawal on Liver Lipids and Serum Lipoproteins of Chickens." *Lipids*, 7 (1972), 742-749.

[59] Swift, L.L., Manowitz, N.R., Dunn, G. and Le Quire, V. "Isolation and Characterization of Hepatic Golgi Lipoproteins from Hypercholesterolemic Rats." *Journal of Clinical Investigation*, 66 (1980), 415-425.

[60] Havel, R.J. and Kane, J.P. "Primary Dysbetalipoproteinemia: Predominance of a Specific Approtein Species in Triglyceride-rich Lipoproteins." *Proceedings of the National Academy of Sciences*, 70 (1973), 2015-2019.

[61] Ross, A.C. and Zilversmit, D.B. "Chylomicron Remnant Cholesteryl Esters as the Major Constituent of Very Low Density Lipoproteins in Plasma of Cholesterol-fed Rabbits." *Journal of Lipid Research*, 18 (1977), 169-181.

[62] French, J.E., Robinson, D.S. and Harris, P.M. "The Role of Cholesterol in the Interaction of Chyle and Plasma." *Quarterly Journal of Experimental Physiology*, 40 (1955), 320-330.

[63] Davis, R.A., Malone-McNeal, M. and Moses, R.L. "Interhepatic Assembly of Very Low Density Lipoprotein: Competition by Cholesterol Esters for the Hydrophobic Core." *Journal of Biological Chemistry*, 257 (1982), 2634-2690.

[64] Goldstein, J.L. and Brown, M.S. "Familial Hypercholesterolemia: A Genetic Regulatory Defect in Cholesterol Metabolism." *American Journal of Medicine*, 58 (1975), 147-150.

[65] Hearn, V. and Bensadoun, A. "Plasma Lipoproteins of the Chicken, *Gallus domesticus*." *International Journal of Biochemistry*, 6 (1975), 295-301.

[66] Nimpf, J., George, R. and Schneider, W.J. "Apolipoprotein Specificity of the Chicken Oocyte Receptor for Low and Very Low Density Lipoproteins: Lack of Recognition of Apolipoprotein VLDL-II." *Journal of Lipid Research*, 29 (1988), 657-667.

[67] Schaefer, E.J., Eisenberg, S. and Levy, R.I. "Lipoprotien apoprotein metabolism." *Journal of Lipid Research*, 19 (1978), : 667-687.

[68] Jackson, R.L., Lin, H.-Y., Chan, L. and Means, A.R. "Isolation and Characterization of the Major Apolipoprotein from Chicken High Density Lipoproteins." *Biochemica et Biophysica Acta*, 420 (1976), 342-349.

[69] Blue, M.-L., Ostapchuk, P., Gordon, J.S. and Williams, D.L. "Synthesis of Apolipoprotein AI by Peripheral Tissues of the Rooster. A Possible Mechanism of Cellular Cholesterol Efflux." *Journal of Biological Chemistry*, 257 (1982), 11151-11159.

[70] Banerjee, D. and Redman, C.M. "Biosynthesis of High Density Lipoprotein by Chicken Liver: Nature of Nascent Intracellular High Density Lipoprotein." *The Journal of Cell Biology*, 96 (1983), 651-660.

[71] Jackson, R.L., Gotto, A.M., Stein, O. and Stein, T. "A Comparative Study on the Removal of Cellular Lipids from Landschutz Ascites Cells by Human Plasma Apolipoproteins." *Journal of Biological Chemistry*, 250 (1975), 7204-7209.

[72] Bowden, J.A. "Biosynthesis and Properties of High-density Lipoproteins." Day, C.E. (Ed.). In: *High-Density Lipoproteins*. Pp. 149-175. New York: Marcel Dekker, 1981.

[73] Shackelford, J.E. and Lebherz, H.G. "Synthesis and Secretion of Apolipoprotein A-I by Chick Breast Muscle." *Journal of Biological Chemistry*, 258 (1983), 7175-7180.

[74] Glomset, J.A. "The Plasma Lecithin: Cholesterol Acyltransferase Reaction." *Journal of Lipid Research*, 9 (1968), 155-167.

[75] Hermier, D., Forgez, P. and Chapman, M.J. "A Density Gradient Study of the Lipoprotein and Apolipoprotein Distribution in the Chickens, *Gallus domesticus*." *Biochemica Biophysica Acta*, 836 (1985), 105-118.

[76] Redshaw, M.R. and Follett, B.K. "Physiology of the Egg Yolk Production by the Fowl: The Measurement of Circulating Levels of Vitellogenin Employing a Specific Radioimmunoassay." *Comparative Biochemistry and Physiology*, 55A (1973), 399-405.

[77] Deeley, R.G., Mullinix, K.P., Wetekam, W., Kronenberg, H.M., Meyers, M., Elridge, J.D. and Goldberger, R.F. "Vitellogenin Synthesis in Avian Liver." *Journal of Biological Chemistry*, 250 (1975), 9060-9066.

[78] Redshaw, M.R. and Follett, B.K. "The Crystalline Yolk Platelet Proteins and Their Soluble Plasma Precursor in an Amphibian, *Xenopus laevis*." *Biochemistry Journal*, 124 (1976), 759-766.

[79] Bergink, E.W., Wallace, R.A., Van de Berg, J.A., Bos, E.S., Gruber, M. and Ab., G. "Oestrogen-Induced Synthesis of Yolk Proteins in Roosters." *American Zoology*, 14 (1974), 1177-1193.

[80] Bernardi, G. and Cook, W.H. "Separation and Characterization of the Two High Density Lipoproteins of Egg Yolk α & β-lipovitellin." *Biochemica et Biophysica Acta*, 44 (1960), 96-105.

[81] Wang, S.-Y. and Williams, D.L. "Differential Responsiveness of Avian Vitellogenin I and Vitellogenin II During Primary and Secondary Stimulation with Oestrogen." *Biochemica Biophysica Research Communication*, 112 (1983), 1049-1055.

[82] Evans, M.I., Silva, R. and Burch, J.B.E. "Isolation of Chicken Vitellogenin I and III CDNAs and the Developmental Regulation of Five Oestrogen-responsive Genes in the Embryonic Liver." *Genes & Development*, 2 (1988), 116-124.

[83] Stifani, S., Barber, D.L., Nimpf, J. and Schneider, W.J. "A Single Chicken Oocyte Plasma Membrane Protein Mediates Uptake of Very Low Density Lipoprotein and Vitellogenin." *Proceedings of the National Academy of Science*, USA, 87 (1990), 1955-1959.

[84] Griffin, H.D., Perry, M.M. and Gilbert, A.B. "Yolk Formation." Freeman, B. (Ed.). In: *Physiology and Biochemistry of the Domestic Fowl,*" pp. 345-380. London: Academic Press, 1985.

[85] Pearse, B. M.F. "Coated Vesicles from Pig Blood." *Journal of Molecular Biology*, 97 (1975), 93-98.

[86] Burley, R.W. and Vadehra, D.V. *Egg Yolk: Structure and Properties and Biosynthesis and Assembly In the Avian Egg*, pp. 171-268. Burley, R.W. and Vadehra, D.V. (Eds.). New York-Singapore: John Wiley & Sons, 1989.

[87] Yusko, S., Roth, T.F. and Smith, T. "Receptor-mediated Vitellogenin Binding to Chicken Oocytes." *The Biochemical Journal*, 200 (1981), 43-50.

[88] George, R., Barber, D.L. and Schneider, W.J. "Characterization of the Chicken Oocyte Receptor for Low and Very Low Density Lipoproteins." *Journal of Biological Chemistry*, 262 (1987), 16838-16847.

[89] Jones, D.G., Briles, W.E. and Schyeide, O.A. "A Mutation Restricting Ovulation in Chickens." *Poultry Science*, 54 (1975), 1780 (abs.)

[90] Barber, D.L., Sanders, E.J., Aebersold, R. and Schneider, W.J. "The Receptor for Yolk Lipoprotein Deposition in the Chicken oocyte." *Journal of Biochemistry*, 266 (1991), 18761-18770.

[91] Hayashi, K., Nimpf, J. and Schneider, W.J. "Chicken Oocytes and Fibroblasts Express Different Apolipoprotein-B Specific Receptors." *Journal of Biological Chemistry*, 264 (1989), 3131-3139.

[92] Scow, R.O., Blanchette-Mackie, E.J. and Smith, L.C. "Role of Capillary Endothelium in the Clearance of Chylomicrons. A Model for Lipid Transport from Blood by Lateral Diffusion in Cell Membranes." *Circulation Research*, 39 (1976), 149-162.

[93] Nilsson-Ehle, P., Garfinkel, A.S. and Shotz, M.S. "Lipolytic Enzymes and Plasma Lipoprotein Metabolism." *Annual Review of Biochemistry*, 49 (1980), 667.

[94] Cryer, A. "Tissue Lipoprotein Lipase Activity and Its Action in Metabolism." *International Journal of Biochemistry*, 13 (1981), 525-541.

[95] Fitzharris, J.J., Quinn, D.M., Goh, E.H., Johnson, J.D., Kashyap, M.L., Srivastava, L., Jackson, R.L. and Harmony, J.A.K. "Hydrolysis of Guinea Pig Nascent Very Low Density Lipoproteins Catalysed by Liprotein Lipase: Activation by Human Apolipoprotein C-II." *Journal of Lipid Research*, 22 (1981), 921-933.

[96] Eisenberg, S. and Levy, R.I. "Lipoprotein Metabolism." *Advances in Lipid Research*, 13 (1975), 1-89.

[97] Benson, J.D. and Bensadoun, A. "Response of Adipose Tissue Lipoprotein Lipase to Fasting in the Chicken and the Rat. A Species Difference." *Journal of Nutrition*, 107 (1977), 990-997.

[98] Cryer, A. "Tissue Lipoprotein Lipase Activity and Its Action in Lipoprotein Metabolism." *International Journal of Biochemistry*, 13 (1981), 525-541.

[99] Griffin, H.D., Butterwith, S.C. and Goddard, C. "Contribution of Lipoprotein Lipase to Differences in Fatness between Broiler and Layer-strain Chickens." *British Poultry Science*, 28 (1987), 197-206.

[100] Gruen, R., Hietanen, E. and Greenwood, M.R.C. "Increased Adipose Tissue Lipoprotein Lipase Activity During Development of the Genetically Obese Rat." *Metabolism*, 27 (1978), 1955-1966.

[101] McNamara, J.P. and Martin, R.J. " Muscle and Adipose Tissue Lipoprotein Lipase in Fetal and Neonatal Swine as Affected by Genetic Selection for High or Low Back Fat." *Journal of Animal Science*, 55 (1982), 1057-1061.

[102] Husbands, D.R. "The Distribution of Lipoprotein Lipase in Tissues of the Domestic Fowl and the Effects of Feeding and Starving." *British Poultry Science*, 13 (1972), 85-90.

[103] Akiba, Y., Jensen, L.S., Barb, C.R. and Kraeling, R. "Plasma Estradiol, Thyroid Hormones and Liver Lipid Content in Laying Hens Fed Different Isocalorific Diets." *Journal of Nutrition*, 112 (1982), 299-308.

[104] Brannon, P.M., Cheung, A.H. and Bensadoun, A. "Synthesis of Lipoprotein Lipase in Cultured Avian Granulosa Cells." *Biochemica et Biophysica Acta*, 531 (1978), 96-108.

[105] Benson, J.D., Bensadoun, A. and Cohen, D. "Lipoprotein Lipase of Ovarian Follicles in the Domestic Chicken (*Gallus domesticus*)." *Proceedings of the Society for Experimental Biology and Medicine*, 148 (1975), 347-350.

[106] Gornall, D.A. and Kuksis, A. "Alterations in Lipid Composition of Plasma Lipoproteins During Deposition of Egg Yolk." *Journal of Lipid Research*, 14 (1973), 197-205.

[107] Glomset, J.A. "Plasma Lecithin Cholesterol Acyltransferase." In: *Blood Lipids and Lipoproteins: Quantitation, Composition and Metabolism*, pp. 745-787. Nelson G.J. (Ed.). New York: Wiley Interscience, 1972.

[108] McDonald, M.W. and Shafey, T.M. "Nutrition of the Hen and Egg Cholesterol." In: *Cholesterol in* Eggs Seminar" pp. 33-39. Egg Industries Research Council, Sydney, Australia, 1989.

[109] Shafey, T.M. "The Relationship between Age and Egg Production, Egg Components and Lipoprotein, Lipids and Fatty Acids of the Plasma and Eggs of Laying Hens." *Journal of Applied Animal Research*,10 (1996), 155-162.

[110] Shafey T.M., Dingle, J.G. and McDonald, M.W. "Comparison between Wheat, Triticale, Rye, Soyabean Oil and Strain of Laying Bird on the Production, and Cholesterol and Fatty Acid Contents of Eggs." *British Poultry Science*, 33 (1992), 339-346.

[111] Shafey, T.M. "The Effects of Dietary Retinol and Sunflower Oil on the Performance, and on the Lipoproteins, Lipids, Cholesterol and Retinol Concentrations of Plasma and Eggs of Laying Hens." *Arab Gulf Journal of Scientific Research*, 16 (1998), 331-345.

[112] Shafey, T.M., Dingle, J.G. and McDonald, M.W. "The Effect of Oat Fractions and Strain of Laying Bird on Production and on the Cholesterol and Fatty Acid Content of Eggs." *Journal of King Saud University, Agric. Sci.* Accepted for publication.

[113] Hargis, P.S. "Modifying Egg Yolk Cholesterol in Domestic Fowl, a Review." *World's Poultry Science Journal*, 44 (1988), 17:29.

[114] Vargas R.E., Allred, J.B., Biggert, M.D. and Naber, E.C. "Effect of Dietary 7-ketocholesterol, Pure, or Oxidized Cholesterol on Hepatic 3-hydroxy-3-methylglutaryl-coenzyme A Reductase Activity, Energy Balance, Egg Cholesterol Concentration, and ^{14}c-acetate Incorporation into Yolk Lipids of Laying Hens." *Poultry Science*, 65 (1986),1333-1342.

[115] Leclercq, B. "Utilisation metabolique des acides gras du tissu adipeux par la poule en ponte." *Annales de Biologie Animale, Biochimie et Biophyique*, 13 (1973), 131-139.

[116] Stadelman, W.J. and Pratt, D.E. "Factors Influencing Composition of the Hen's Egg." *World's Poultry Science Journal*, 45 (1989), 247-266.

[117] Shafey, T.M. and Dingle, J.G. "Factors Affecting Egg Fatty Acid and Cholesterol Content." In: *Australian Poultry Science Symposium*, Poultry Research Foundation (Ed.), University of Sydney and World Poultry Science Association, NSW, Australia, 1992, 79-83.

[118] Fisher, H. and Leveille, G.A. " Fatty Acid Composition of Eggs as Influenced by Dietary Fats." *Poultry Science*, 30 (1957), 1116. (abstract)

[119] Sim, J.S., Bragg, D.B. and Hodgson, G.C. "Effect of Dietary Animal Tallow and Vegetable Oil on Fatty Acid Composition of Egg Yolk, Adipose Tissue and Liver of Laying Hens." *Poultry Science*, 52 (1973), 51-57.

[120] Shafey. T.M., Dingle, J.G. and McDonald, M.W. "The Relationship between Egg Yolk Cholesterol, Egg Production and Age of the Hen in Three Australian Layer Strains." *Journal of King Saud University, Agric. Sci.*, 10. No. 1 (1998), 33-41.

دور الليبوبروتينات في أيض الدهون في الدواجن

طـــارق محمـــد شـــافعي

قـــسم الإنتـــاج الحيـــواني، كلية الزراعة، جامعة الملك سعود، الرياض، المملكة العربية السعودية

(قدم للنشر في ١١/٢٦/ ١٤١٩م وقبل للنشر في ١٢ / ٨/ ١٤٢٠ هـــ)

ملخص البحث. يلقي هذا الإطلاع المرجعي الضوء على تكوين وإفراز طوائف الليبوبروتينات المختلفة والعلاقة بينها ودورها في تنظيم نقل الدهون في دم الدواجن وتخزينها في أنسجة الجسم .

ويتضح من هذه المراجعة الآتي :

١ – يعتمد أيض الليبوبروتينات على تركيبها ونشاط عدة إنزيمات .

٢ – تعتبر الليبوبروتينات من العوامل المهمة ليس فقط في تطور الأنسجة الدهنية أثناء النمو بل أيضاً في توزيع الدهون في هذه الأنسجة المختلفة .

٣ – تحمل الليبوبروتينات موجهات بروتينية تستهدف أنسجة خاصة (مثل البويضات في الدجاجة البياضة) نتيجة تفاعل هذه الموجهات مع مستقبلات خاصة لها على سطح خلايا الأنسجة .

٤ – تعكس الاختلافات الموجودة في مكونات ليبوبروتينات الدم بين أقسام الدواجن إلى الفروق في الحالات الفسيولوجية لهذه الدواجن .

وتعتبر التغيرات في تركيز ليبوبروتينات الدم تحت الحالات الفسيولوجية المختلفة من أهم الخواص الـــتي تتميز بها الدواجن ، والتي تتيح فرصاً مميزة لدراسات المقارنة البحثية .