Lipid Absorption and Transport in Ruminants

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ABSTRACT

The objective of this paper is to review new insights on the biological mechanisms of absorption and transport of lipid in ruminants, especially the modern concepts and analytical methods used in studies on structural properties and intravascular and tissue metabolism of lipoproteins and their factors of variation. The intestinal absorption of lipids (including long-chain fatty acids) is detailed, and variations in the qualitative and the quantitative aspects of absorption with diet composition, especially for high fat diets, are presented. Also, structural properties and distribution characteristics of lipoprotein classes in different lymphatic and blood vessels are compared across several animal species. Physicochemical and hydrodynamic properties of the lipoprotein particles and their apolipoprotein moieties are given for the main classes of lipoproteins. Finally, lipoprotein metabolism is discussed in relation to development and physiological, nutritional, and hormonal status. Intravascular metabolism of lipoproteins, including the role of lipolytic enzymes and lipid transfer proteins, is presented. Characteristics of the intestinal and hepatic synthesis of lipoproteins and apolipoprotein fractions are compared, especially through experiments stimulating the hepatic secretion of triglyceride-rich lipoproteins. Different methods of measurement of lipoprotein tissue uptake or secretion in ruminants are discussed.

(**Key words**: lipids, intestinal absorption, lipoprotein metabolism, nutritional and physiological status)

Abbreviation key: **ACAT** = acyl:cholesterol acyltransferase, **apo** = apolipoprotein (used with A, B, C, D, and E), **CE** = cholesteryl esters, **FA** = fatty acids, **HDL** = high density lipoproteins, **IDL** = intermediate density lipoproteins, **LCAT** = lecithin:cholesterol acyltransferase, **LDL** = low density lipoproteins, **LPL** = lipoprotein lipase, M_r = molecular weight, **PC** = phosphatidylcholine, **PL** = phospholipids, **TG** = triglycerides, **VLDL** = very low density lipoproteins.

DIGESTION AND ABSORPTION OF LIPIDS IN THE SMALL INTESTINE

Composition of Lipids Entering the Duodenum

In adult ruminants, lipid digestion begins in the enlarged forestomach (reticulorumen), but, in nonruminants, little digestion occurs before lipids enter the small intestine. The initial stages of ruminant digestion are characterized by intense lipolysis, fatty acid (**FA**) hydrogenation, and de novo lipid cellular synthesis by microorganisms [see the review by Jenkins (63)]. Very little absorption of the water-insoluble long-chain FA takes place in the forestomach and the abomasum (89).

Thus, lipids of postruminal digesta are mainly composed of saturated NEFA (from dietary and microbial origin; 70%) and small and variable amounts of microbial phospholipids (**PL**) (10 to 20%); all are adsorbed predominantly on particulate matter (75, 87, 89). Similarly, triglycerides (**TG**) of protected fat diets also may be present in the postruminal digesta, associated with solid material. In the particularly acid conditions of abomasal and duodenal digesta (pH 2.0 to 2.5), NEFA are fully protonated (no ionic form is present), which maintains lipid absorption onto the surface of particulate matter (89).

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Lipid Digestion in the Small Intestine

Lipid digestion occurs in a biphasic medium that consists of an insoluble particulate phase to which free FA (and PL) are attached and a soluble micellar phase containing dissolved FA. Transfer of free FA to the micellar phase occurs gradually as digesta go through the intestinal tract; 5% of the total transfer occurs in the duodenum, 20% in the upper jejunum, 25% in the mid and lower jejunum, and 50% in the ileum (75). The process by which FA are released from particulate matter involves polar detergency. Bile secretion in the duodenum favors the interaction of FA with bile PL and water which leads to the formation of a liquid crystalline phase. With increasing pH, this phase then is dispersed in the presence of bile salts to form the micellar solution. Conversion of bile PL to lysophospholipids by pancreatic phospholipase A2 stimulated micellar solubilization of FA (as possibly did PL) and thus improved the FA passage through the unstirred water layer adjacent to the microvilli of the small intestine and the subsequent absorption by the intestinal mucosal cells (78).

With conventional diets, 15 to 25% of total FA are absorbed readily in the upper jejunum (pH 2.8 to 4.2); 55 to 65% of digesta FA are absorbed in the middle and the lower jejunum (pH 4.2 to 7.6) (75, 87, 89).

With diets containing protected lipids, lipid digestion by ruminants occurs, as it does in monogastric animals, in a biphasic system consisting of an oil phase and a micellar phase. Pancreatic lipase and colipase systems convert TG into free FA and 2-monoacylglycerols, which constitute an important factor in the micellar solubilization of free FA (87). Under those dietary conditions and because of the optimal pH (pH 7.5) of lipase activity, TG hydrolysis and, thus, FA absorption do not take place before the midjejunum (75).

Quantitative Aspects of Intestinal Lipid Absorption

Using a wide variety of techniques, such as FA flow measurements in the digestive tract of duodenally cannulated ruminants, numerous authors demonstrated the higher efficiency of ruminants to absorb FA compared with nonruminants (8, 24, 49, 58, 59, 75, 87, 88, 89, 98, 120).

Generally, the intestinal absorption coefficient of individual FA ranges from 80% (for saturated FA) to 92% (for polyunsaturated FA) in conventional diets with low fat content (2 to 3% DM). The particularly high efficiency of ruminants to absorb saturated FA can be explained by two factors: 1) the great capacity of the bile salt and lysophospholipid micellar system to solubilize FA and 2) the acid conditions (pH 3.0 to 6.0) of duodenal and jejunal contents. This low pH is due to a low concentration of pancreatic bicarbonate (75), which probably limits strongly the formation of insoluble Ca soaps with saturated FA.

Use of fat-supplemented diets has been developed during the past decade to meet the energy needs of high yielding dairy cows in negative energy balance (98). Fat addition increases energy density of diets and, thus, allows maximal fat intake but may alter rumen microbial metabolism. Consequently, numerous products have been tested and compared for rumen inertness and digestibility (8, 49, 64, 88, 97, 120, 131).

Increasing the dietary fats generally results in higher apparent intestinal FA digestibility because of dilution of bile FA and of bacterial FA produced in the large intestine. True FA digestibility of dairy cows seems to decrease progressively from 95 to 78% when FA intake increases from 200 g/d (1% DM) to 1400 g/d (8% DM) (97). This decrease suggests limited secretion and activity of pancreatic lipase and biliary lipids (bile salts, PL), which may affect lipid absorption in ruminants at high fat intakes. The role of PL in micelle formation is greater in ruminants than in monogastrics because of the absence of 2-monoglycerides. However, in the case of oil infusion in the proximal duodenum of dairy cows in early lactation, similar FA digestibilities (75 to 78%) were calculated with high amounts of FA infused (1250 to 1700 g/d) with or without addition of lecithin in the diet (24).

The intestinal digestibility of FA may be affected by the source and type of presentation of dietary fats. The FA digestibility was lower in dairy cows fed diets containing 1 to 2 kg of full fat crushed rapeseed (76.4 and 84.2 vs 91.4%) (88). Among other hypotheses, a limited capacity of FA digestion relative to FA intake has been suggested (88). Moreover, incomplete availability of FA when associated with the cellular structure of feeds for micelle formation also could be considered.

Digestibility of total or individual FA was higher in dairy cows fed diets containing Ca soaps of palm FA compared with diets containing animal-vegetable blend fat (3 and 6% of DM) (131). These results could be explained by the higher proportion of unsaturated FA in duodenal digesta with the Ca soap diets. Indeed, Ca soaps are very stable in rumen contents and could provide more effective protection against rumen biohydrogenation. However, compared with a low fat control diet, these high fat diets lowered intestinal digestibility, which emphasizes difficulties in micellar solubilization and absorption processes of FA in these cows. By contrast, intestinal digestibility of total FA in cows fed a diet containing emulsified fat (from milk; 10% DM) was higher (86 vs 80%) than with the low fat control diet (8).

LIPID TRANSPORT SYSTEM IN RUMINANTS

The main function of plasma lipoproteins is to transport lipids from secreting organs (intestine and liver) to peripheral tissues. In farm animals, especially ruminants, the chemical composition and the rate of secretion of lipoproteins are among the main factors that control lipid utilization by tissues and, thereby, the qualitative and quantitative characteristics of meat production and milk yield. Research in this area in the last two decades has improved knowledge of the influence of lipoproteins in the partition of lipids among skeletal muscles, liver, and adipose tissues in meat animals [see the review by Kris-Etherton and Etherton (69)] and in the contribution of lipids to milk fat synthesis [see the review by Palmquist (96)] and steroidogenesis by sexual tissues [see the review by Grummer and Carroll (51)] in cows. More recently, significant progress has been made concerning the mechanisms involved in the intravascular or tissue metabolism of lipoproteins, partly because of the acquisition of more specific analytical methods and new concepts developed in humans and rodents. Several reviews (21, 22, 23, 106, 107) were published in this area during the past decade.

Purification and Characterization of Ruminant Lipoproteins

Lipoproteins are macromolecular complexes containing different lipids and specialized proteins (called apolipoproteins) that are soluble in the fluids of the vascular systems (plasma and lymph) and in the intestinal or follicular fluids. These lipoprotein particles can be compared with pseudomicelles because they are all composed of hydrophilic components [PL, free cholesterol (**FC**), and apo] located at the surface of the particles in a unilamellar membrane and of hydrophobic lipids [TG and cholesteryl esters (**CE**)] located in the inner core of the particles.

Differences in buoyant density, size, and apo moieties allow lipoproteins of ruminants to be isolated and characterized. These steps are carried out either by sequential ultracentrifugal flotation between solvent density limits proposed for human plasma (11, 19, 35, 41, 44, 62, 65, 108, 113, 123, 125, 129)—completed or not by selective precipitation using sulfated dextran (19, 84) or other reagents (13, 18, 46, 60, 100)—or by electrophoresis (19, 86, 114, 122) and immunoelectrophoresis (124). By these methods, plasma lipoproteins of ruminants have been divided into five major density classes that reflect their relative lipid-protein content (Table 1): chylomicrons, very low density lipoproteins (**VLDL**), intermediate density lipoproteins (**IDL**), low density lipoproteins (**LDL**), and high density lipoproteins (**HDL**).

Large variations in the metabolism of ruminant lipoproteins and in the complexity of distribution of their subfractions, especially in the interval of LDL density (1.006 to 1.063 g/ml), have justified the development of high resolution methodologies.

Isopycnic density gradient ultracentrifugation, which allowed subfractionation of >20 lipoprotein fractions in one step according to their hydrated density, has been used for determination of lipoprotein profiles in plasma and lymph in the preruminant calf (9, 72) and for the separation of TG-rich lipoproteins (99, 110) and of HDL (109, 127) in the dairy cow.

Filtration on agarose gel column was also used to isolate and to characterize plasma lipoprotein subfractions in the dairy cow by molecular size (2, 42, 43, 47, 52, 53, 54, 83). However, immunological and electrophoretical techniques clearly showed that both methods, density gradient ultracentrifugation and gel filtration, failed to separate LDL completely from very light HDL particles of bovine

TABLE 1. Percentage of composition of the plasma and lymph lipoproteins in the bovine.[1]

Component	Chylomicrons		VLDL[2]		LDL[3]		Light HDL[4]		Heavy HDL	
	Lymph	Plasma	Lymph	Plasma	Lymph	Plasma	Lymph	Plasma	Lymph	Plasma
Free cholesterol	1–2	4–6	1–2	3–9	3–7	6–8	1–2	4–6	2–8	1–4
Cholesterol ester	1–8	1–4	1–2	5–15	16–32	31–36	28–32	29–33	1–26	13–29
Triglyceride	67–88	72–87	75–80	45–63	5–35	4–21	3–7	1–3	4–9	1–6
Phospholipid	8–20	4–5	10–12	12–17	22–33	18–22	27–30	25–27	9–27	12–27
Protein	2–3	2–3	6–8	8–16	20–27	22–32	30–39	33–39	41–74	39–68
Diameter, Å	650–2400		340–860	310–650	210–260	190–250	120–140	120–150	90–105	93–120
Density limits, g/ml	<.95		<1.006		1.026–1.076		1.060–1.091		1.091–1.180	
	$S_f > 400$[5]		$S_f < 400$							

[1]Data summarized from references (9, 11, 21, 42, 44, 56, 70, 72, 96, 113, 114).
[2]Very low density lipoproteins.
[3]Low density lipoproteins.
[4]High density lipoproteins.
[5]Flotation coefficient in Svedberg units.

plasma and lymph in the density range of 1.039 to 1.076 g/ml (4, 9, 43, 53, 72, 107).

Occurrence of some HDL particles of size and buoyant density similar to that of LDL particles is specific to the bovine and appears during early postnatal development (10). Complete resolution of homogenous LDL and HDL was finally achieved by heparin-sepharose chromatography from calf and cow plasma (29, 31, 70) and from calf lymph (70). Bovine HDL, devoid in apolipoprotein (**apo**) E, are not retarded by heparin-sepharose gel, but the LDL rich in apo B undergo selective and reversible absorption with the resin.

Moreover, the very high saturation of the core lipid FA of ruminant VLDL (reflecting hydrogenation processes occurring in the rumen) limited the efficiency of recovery of VLDL particles in plasma by ultracentrifugation at temperatures <20°C (55, 109).

STRUCTURE AND FUNCTION OF LIPOPROTEINS AND THEIR APOLIPOPROTEIN MOIETIES

Chylomicrons

Chylomicrons are the largest (500 to 2500 Å) and the least dense (flotation coefficient, S_f, >400) lipoprotein particles (72) that are synthesized and secreted by the intestine after a meal containing fat in preruminant calves (11, 72) and in other ruminants (42, 59). The main role of these TG-rich lipoproteins is to transport dietary FA (as TG) to tissues for fat storage, milk fat production, or for oxidation to produce energy (Figure 1). Chylomicron production by ruminants has long been the subject of a controversy, probably because of the low fat content of ruminant diets; however, chylomicron particles clearly are synthesized by the bovine intestine (72). Chylomicron secretion is stimulated by increasing dietary fat (3) or dietary polyunsaturated FA (59) compared with saturated FA, which, in sheep as in rats and in humans (21), leads to secretion of a majority of VLDL particles by intestinal mucosal cells (59). Thus, in sheep under normal low fat dietary conditions (<10 g/d of TG), 72.6% of the lymph lipids (577 mg/h) were located in the VLDL fraction and 27.4% in the chylomicron fraction. After 24 h of infusion of maize oil (52.6% linoleic acid; 48 g/d of TG) through a duodenal cannula, 38.5% of the lymph lipids (1550 mg/h) were present in the VLDL and 61.5% in the chylomicrons (59).

A comparison of the lipid content of bovine chylomicrons in lymph (72) and in plasma (42) (Table 1) showed that the exposure of lymph chylomicrons to serum led to a gain of free cholesterol and a loss of PL, which are probably recovered in HDL particles (3). Apolipoproteins of chylomicrons, determined by electrophoretical and immunological methods (4, 9, 80, 81), are characterized by the prevalence of apo B48. This unique high molecular weight (M_r) apolipoprotein is present in these particles (M_r 265,000 to 320,000) (72) with minor peptides of the apo C family (M_r 10,000), and with variable amounts of apo AI [M_r 28,000 (70)].

VLDL

Bovine VLDL particles (flotation coefficient <400; density <1.006 g/ml) represent the alternative form of TG transfer in lymphatic and blood vessels from the intestine (72). In dairy cows, VLDL are probably less synthesized by the liver (9, 68) (Figure 1) than VLDL in humans.

Simultaneous measurements of VLDL and chylomicron fluxes in the intestinal lymph duct, portal vein, and mesenteric artery have recently provided evidence that the portal vein is a major export pathway of intestinal TG-rich lipoproteins in the calf (36, 71). Similarly, arteriovenous measurements of TG fluxes in the intestine of lactating dairy cows fed a high fat diet indicated a direct secretion of intestinal TG in the portal vein (25). Slow intestinal lipid absorption in ruminants, as in rats, can lead to preferential transport of dietary FA in the portal system (79). This effect occurs in preruminant calves that are fed milk diets that coagulate in the abomasum and in other

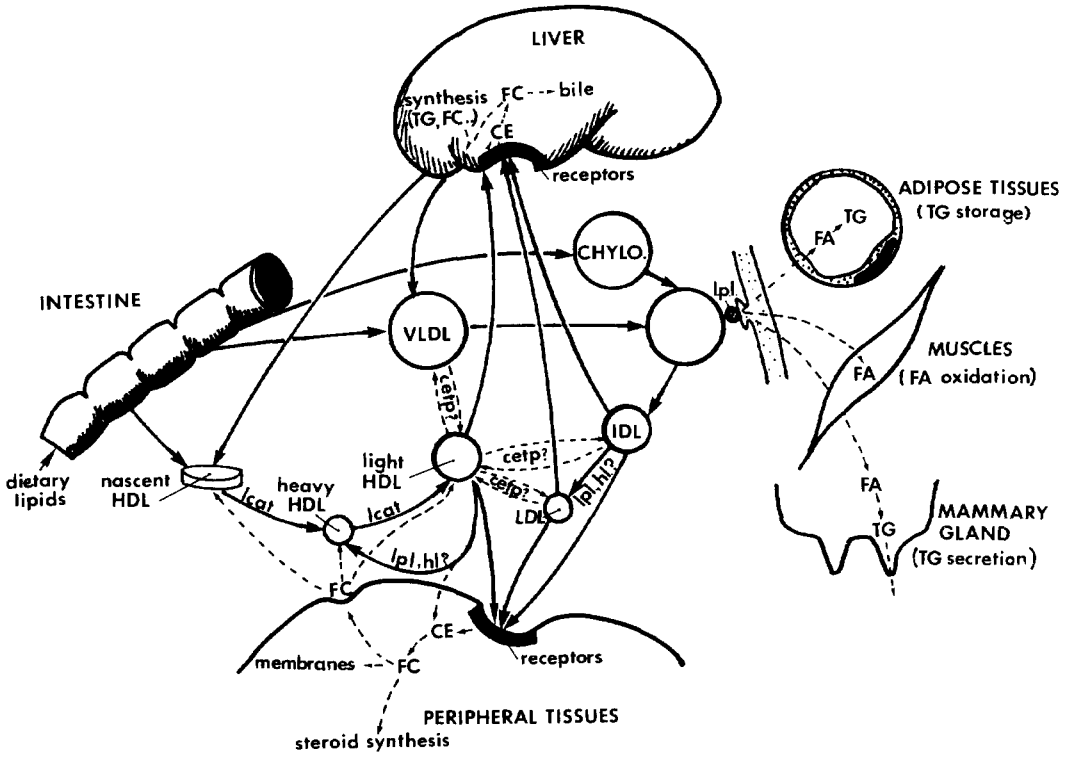

Figure 1. The tissue origins and postsecretory metabolic transformation of lipoproteins within the plasma compartment in ruminants. HDL = High density lipoproteins, LDL = low density lipoproteins, TG = triglyceride, CE = cholesterol ester, cetp = CE transfer protein, lcat = lecithin:cholesterol acyltransferase, CHYLO = chylomicron, VLDL = very low density lipoproteins, lpl = lipoprotein lipase, hl = hepatic lipase, FA = fatty acid, FC = free cholesterol.

ruminants that are fed diets characterized by long rumen retention time.

Plasma VLDL, lacking in fetal calves (10, 44), are generally low in the bovine compared with VLDL in human plasma (21) because VLDL represent 5% of the total lipoproteins in preruminant calves (9, 11, 112) and .5% in dairy cows (46, 52, 112). Although VLDL secretion by the intestine can be enhanced by biliary PL (73) and by high fat diets (125), VLDL concentrations are relatively low in dairy cows, probably because of the rapid turnover of the VLDL pool (46, 99), especially after milking (47).

Lipid Composition. Lipid VLDL are characterized by an abundance of TG in the bovine, as in other mammals, and by elevated PL content (Table 1). But, as in rats, VLDL in bovines are deficient in CE (9, 52, 112, 123), especially those in calf lymph (72). As for chylomicrons, FA composition of VLDL TG is highly influenced by dietary FA at peak lipid absorption in preruminant calves (11) but is not modified by dietary source of FA in dairy cows (52), probably because of the intense hydrogenation of dietary FA by rumen microorganisms.

In preruminant calves, FA composition of plasma VLDL TG (and chylomicron TG) is modified during the postprandial period (11). The intense hydrolysis of plasma TG by LPL during postprandial hyperinsulinemia led to the appearance of more saturated TG in the remaining particles and seemed to indicate selective utilization of dietary unsaturated FA by extrahepatic tissues.

The FA composition of VLDL CE varies in developing calves (62). The low store of essential FA (mainly $C_{18:2n-6}$) in calves and lambs at birth (74, 92) is due to a very low placenta transfer of essential FA from the NEFA in maternal plasma. Because ruminant placenta is impermeable to other plasma lipids, CE synthesized by the intestinal and hepatic acyl: cholesterol acyltransferase (**ACAT**) (90) were naturally poor in essential FA and did not contribute to CE in LDL and HDL. As the calf grows, selective incorporation of absorbed essential FA (mainly $C_{18:2n-6}$) into plasma lecithin in β position and specific transfer to plasma CE by lecithin:cholesterol acyltransferases (**LCAT**) (91) were hypothesized in the TG-rich lipoproteins (62). However, the relatively lower concentrations of $C_{18:2n-6}$ in CE of these particles compared with those of LDL and HDL can be partially explained by ACAT activity, which is nonspecific for the essential FA, as demonstrated in sheep (90).

Apolipoprotein Content. Like other mammals, ruminants synthesize two major molecular mass forms of apo B VLDL: the large form (M_r 520,000) is referred to as apo B100, and a smaller form (M_r 265,000) is designed as apo B48 (9) (Table 2). These forms play a major role in the formation and receptor-binding of the particles. Details of the molecular mechanisms of apo B biosynthesis in mammals have been recently reviewed by Gibbons (45). A single gene is coded for both forms of apo B in mammals. In humans and rats, apo B48 is produced by the intestine via a postranscriptional modification of the apo B mRNA. A single base substitution, catalyzed by a cytosine deaminase, induced the formation of a stop codon leading to the formation of apo B48. Similar mechanisms of specific biosynthesis and secretion of apo B48 by the ruminant intestine may be involved, because apo B48 was only present in calf lymph VLDL (72). The occurrence of both forms of apo B in plasma VLDL indicates that, in ruminants (9, 56) as in the majority of mammals, apo B100 (9) is only synthesized by the liver. However, no evidence exists for a possible hepatic origin of apo B48, as observed in rats (45).

Apolipoproteins other than apo B (M_r <100,000) were also detected in bovine VLDL (9, 56) (Table 2). The bovine counterpart to human apo A-IV has been identified (M_r 42,000) by size and density distribution in plasma VLDL (Table 2) of calves (9) and cows (56). Studies on humans and rodents (21, 22) indicate that apo A-IV plays a significant role in LCAT activation in ruminants. A major component with M_r of approximately 23,000, which may correspond to an unnamed apolipoprotein of rat VLDL, was detected in VLDL and chylomicron fractions in plasma of cows (31, 56).

Multiple (up to 7) species similar to apo C in plasma VLDL (Table 2) have been detected at low concentrations in newborn (44) and developing calves (9) and in steers (44), but not in lactating cows (56). These lower concentrations of apo C proteins in VLDL compared with those in HDL can be explained by a slow

TABLE 2. The biosynthetic origins, plasma distributions, and ascribed functions of the major apolipoproteins in the ruminants.[1]

Apolipoprotein	Approximate molecular weight	Major biosynthetic sites	Plasma distribution	Ascribed functions
A-I[2]	28,000	Intestine, liver	Chylomicrons, HDL[4]	Particle formation, receptor binding, LCAT[7] activation
A-II (?)	8800	Intestine, liver	HDL	Hepatic lipase activator (?)
A-IV	42,000	Intestine	Chylomicrons, heavy HDL	LCAT Activation
B-100	550,000	Liver	VLDL,[5] LDL[6]	Particle formation, receptor binding
B-48	265,000	Intestine (liver ?)	Chylomicrons, VLDL, remnants	Particle formation
CI	8000	Liver	Chylomicrons, VLDL, HDL	LPL[8] Activator
CII[2]	10,000	Liver	Chylomicrons, VLDL, HDL	Lipid binding
CIII[3]	9000	Liver	Chylomicrons, VLDL, HDL	LPL Inhibitor
CIV	10,000	Liver	Chylomicrons, VLDL, HDL	LPL Inhibitor (?)
E (?)	34,000	Liver	VLDL (?), HDL (?)	Receptor binding (?), LCAT activation (?)

[1]From references (6, 7, 9, 12, 27, 56, 70, 71, 93, 121).

[2]Primary structure has been fully determined [(6, 93, 121) for apo A-I; (12) for apo CII].

[3]In the bovine, the primary structure is only partially determined (12).

[4]High density lipoproteins.

[5]Very low density lipoproteins.

[6]Low density lipoproteins.

[7]Lecithin:cholesterol acyl transferase.

[8]Lipoprotein lipase.

rate of transfer from HDL particles when TG-rich lipoproteins enter plasma. Among the apo C proteins characterized from bovine plasma (12, 27, 66, 77, 101), CI and CII proteins (M_r 8000 and 10,000, respectively) enhance lipid binding and bovine lipoprotein lipase (LPL) activity (27). On the contrary, CIII protein (M_r 9000) and, to a lesser extent, CIV protein (M_r 10,000) inhibit LPL activity (27) and, as in humans, probably compete with apo CII for binding sites at the lipid-water interface (12). Recently, the primary structure of bovine apo CII and CIII and their isoforms has been determined from total plasma lipoproteins by Bengtsson-Olivecrona and Sletten (12). The apo CII contains 73 AA residues and exhibits great homology of AA sequence with other species, mainly with human and dog analogues. However, substitutions involving 3 of the 5 residues that are generally considered to be essential for LPL activation have been mentioned for bovine apo CII. The apo CIII was found as three isoforms, which contained 0, 1, or 2 sialic acid residues per molecule. Their sequence (resolved to residue 69) showed several differences in sequence from those of other species, mainly at the ends of the molecule, which may be due to differences in gene structure or different processing.

The apo E has been tentatively identified by electrophoretic methods as a protein with M_r 34,000 in plasma VLDL (56), LDL, and HDL (7, 56) of lactating cows (Table 2). However, similar studies in calves (9, 70, 72) and adult cows (17, 27, 31, 66, 77) failed to detect apo E in these different lipoprotein families. Cholesterol feeding, which classically stimulated production of apo E by the liver in humans and in rabbits, led to inconsistent effects in preruminant calves. With such a diet, an apo E-like protein (M_r 35,000) was present in plasma VLDL and IDL, but not in HDL, in calves (14, 15); however, in the recent work of Leplaix et al. (76) using both immunological and electrophoretical techniques, apo E in VLDL was totally lacking in the preruminant calves.

IDL

The IDL are particles that span the plasma density fraction range of 1.006 to 1.026 g/ml and are intermediates in the VLDL-LDL cascade. The IDL are generated during lipolysis of VLDL, catalyzed by LPL of extrahepatic tissues, and interact avidly with the cell surface of apo B, E receptors (Figure 1). The intense captation of IDL by the liver and peripheral tissues (118) probably explains the very low concentrations of these particles in the plasma (9, 14, 44, 112, 122) and in the intestinal lymph (72) of calves and cows.

The physicochemical properties of bovine IDL (also designated as LDL 1) have been characterized in lactating cows using large volumes (1 to 5 L) of plasma as starting material (109, 123). The core components of IDL consisted mainly of TG (61 to 70%), because CE represented only 1 to 3%, and surface components (apolipoproteins, PL, and free cholesterol) that represented 30 to 39% of the total particles (109). However, in calves fed cholesterol, preferential accumulation of CE (35 to 43% of total IDL) in substitution for TG in the inner core of IDL particles is associated with a 10-fold increase in plasma IDL concentration (from 4 to 40 mg/dl) (75). Outer surface modification of these particles, resulting from massive incorporation of CE and inhibition of cellular receptor synthesis during hypercholesterolemia, may explain the reduction of IDL binding with cellular receptors.

The presence of saturated TG in bovine IDL led to the appearance of unusual IDL that were flat and asymmetric when isolation temperatures were well below the melting points of the core lipids (109).

Little is known on the apolipoprotein content of bovine IDL. The apo B appeared to be a major apolipoprotein in calf particles (9, 76), but another major apolipoprotein with an electrophoretic mobility of M_r 40,000 (apo A-IV) has also been reported in IDL of lactating cows (109).

LDL

Bovine LDL are end products of the intravascular degradation of VLDL via IDL and are implicated in the cholesterol distribution to tissues. Quantitatively, LDL represent a minor lipoprotein class (<10% of total lipoproteins) in the plasma [see reviews by Palmquist (96); Chapman (21, 22), and Puppione (107)] and in the bovine intestinal lymph (70, 72) except in the plasma of fetal calves, where they predom-

inate (10, 44). In ruminants, this low concentration of plasma LDL is probably related mainly to the low plasma CE transfer activity (as in rats and dogs) relative to humans (14 to 22%) (57) and to the concomitant very low activity of hepatic lipase in bovine postheparin plasma (30) (Figure 1).

Bovine LDL particles, large particles with flotation rate of 4.6 to 5.3 and Stokes diameter from 189 to 260 Å (9, 44, 72), are present in bovine plasma at the density interval from 1.026 to 1.076 g/ml. These particles represent a subspecies that is unique and homogenous in size but only in the range of 1.026 to 1.046 g/ml (9). Overlap in the density distribution of LDL and HDL particles occurs in the density range of 1.046 to 1.076 g/ml during the early postnatal period (2 wk) (10). Maturation processes involve the appearance of denser LDL and lighter HDL. The heterogeneity in the plasma (4, 9, 31, 53, 107) and in the intestinal lymph (3, 70, 72) was resolved by use of heparin-sepharose affinity chromatography.

Lipid Composition. Bovine LDL lipids consist of two main components, CE (48% of total lipids) and PL (27%), and two minor components, free cholesterol (10%) and TG (15%) (31, 70). Lipid distribution differed between plasma and lymph (70) (Table 1). Plasma LDL PL are dominated by phosphatidylcholine (PC) (60%) and, to a lesser extent, by phosphatidylethanolamine (21%) and sphingomyelin (18%), except in LDL of the newborn calves, which mainly contain phosphatidylethanolamine (58%) (62). The FA composition of lipid LDL changes more with postnatal development (62) than with diet in the adult (52). Concentrations of $C_{18:2n-6}$ in PC (from 1 to 13%) and CE (from 10 to 35%) increased in calves between 3 d and 12 wk (weaned) (62). The lower CE $C_{18:2n-6}$ in LDL (35 to 39%) than in HDL (46 to 64%) (11, 62) seemed to indicate a dual origin of CE in LDL particles: 1) intestinal and hepatic VLDL, naturally poor in $C_{18:2n-6}$ synthesized by the action of ACAT (90) and 2) HDL CE, rich in $C_{18:2n-6}$ because of the high specificity of ruminant LCAT for $C_{18:2n-6}$ esterified in β position in PC (91).

Apolipoprotein Content. Protein moiety of bovine LDL (isolated by heparin-sepharose affinity chromatography) is essentially composed of apo B100 in plasma because apo B48 is only detectable as a trace component (70, 72). However, LDL from calf lymph exhibited the presence of apo B100 and apo B48 in similar amounts (70, 72). A probable explanation is the occurrence of a process in which intestinal secretion of apo B48 LDL particles and filtration from plasma of apo B100 LDL (already observed in other species) occurs simultaneously (72). As in many other mammals, the virtual absence of apo B48 LDL from bovine plasma suggests that this type of particle is more efficiently removed by tissues via apo B, E receptors than the apo B100 LDL counterpart (72).

HDL

The HDL are the major plasma lipoproteins (>80% of total lipoproteins) in preruminants (9, 11, 44, 62, 112) and ruminants (44, 53, 65, 66, 83, 86, 110, 112, 114, 123), except in fetal calves (10, 44) and in intestinal lymph (72).

The HDL are synthesized and secreted by the liver and the small intestine as discoidal particles that become spheric during CE formation via the LCAT reaction on and after the fetal stage (10, 44). These particles are the main particles implicated in a reverse cholesterol transport system that returns excess cholesterol from peripheral cells to the liver for bile excretion and resynthesis of new VLDL particles (Figure 1).

Lipid Composition. Bovine HDL are structurally and metabolically heterogeneous. They are generally present in plasma as two main distinct populations: 1) light HDL (density range of 1.060 to 1.091 g/ml), ranging in size from 120 to 140 Å, rich in CE (48% of total lipids), but poor in TG (3%) and 2) heavy HDL (density range of 1.091 to 1.180 g/ml), ranging in size from 93 to 120 Å, enriched in TG (7 to 13% of total lipids) compared with those of the light HDL (9, 110, 127) (Table 1). Moreover, very light HDL (density range 1.039 to 1.060 g/ml) were also identified in the density range of LDL in the bovine plasma and intestinal lymph (9, 31, 70). The low plasma CE transfer (57) and hepatic lipase (31, 40) activities explain the major role played by plasma HDL in CE transport in ruminants (LDL for humans and rabbits) (57). Phospholipids, which are the main surface components in bovine HDL, as in LDL, are dominated by PC (40%) (62). The PL

contained elevated $C_{18:2n-6}$ (17 to 23%), which are preferentially transferred to HDL CE (46 to 83%) (11, 52, 62) via the LCAT system (91).

Apolipoprotein Content. The apo A-I (M_r 28,000) predominates in the bovine HDL protein moiety (80%) (9, 31, 56, 66, 72, 126) in six isoforms (56) (Table 1) and plays a multifunctional role that includes binding to cells through formation of amphipathic helices (43% helical structure) (67) and activating LCAT (66). Moreover, analysis of physicochemical properties of micellar complexes containing bovine apo A-I and synthetic lipids demonstrated the important role of apo A-I in the surface structure of HDL shell. The PC is the sole lipid layer adjacent to apo A-I; the mobility of these complexes is restricted by the presence of cholesterol (67). Determination of amino-terminal sequence (6) and, recently, of the primary structure of bovine HDL apo A-I from the purified protein (121) and from the sequence of apo A-I cDNA (93) indicated that this protein of 241 AA is more closely related to corresponding proteins in dog than to those in primates. Similar positions for all 13 detectable antigenic sites between human and bovine apo A-I were determined by comparative predictions (121).

The apo C are mainly associated with HDL in the bovine (9, 27, 66, 72, 77, 101, 102). Details on isolation processes and characterization of the main C peptides (apo C II and C III) are in the VLDL section.

The apo A-II (M_r 17,500), an hepatic lipase activator, was not detected in ruminant HDL, but presence of a polypeptide (M_r 8800) in plasma HDL might correspond to a monomeric form of apo A-II (72, 77, 101). In contrast, apo A-IV (M_r 42,000) was shown in the densest HDL (density range of >1.131 g/ml) of bovine plasma (7, 9). Initially secreted by intestine TG-rich lipoproteins (9), this LCAT activator would rapidly transfer predominantly to HDL, as demonstrated in rats (127).

As with VLDL, the presence of apo E (M_r 34,000) in bovine HDL is debatable (Table 2). separation of bovine HDL by heparin-Sepharose affinity chromatography confirmed the absence of HDL apo E (9, 17, 31, 70, 72) but revealed the presence of an HDL fraction containing a protein that exhibits, as does human apo E, heparin-binding properties (70). This protein probably corresponds to β2 glycoprotein I according to M_r (51,000), amphiphilicity, and immunological reactivity (70).

Postsecretory Modifications. Density distribution of plasma HDL undergoes large fluctuations according to physiological or nutritional states. Initially present as heavy HDL during the fetal stage (10), bovine HDL is rapidly dominated by light HDL 2 wk after birth (10, 112). This marked increase of plasma light HDL is also observed during lactation (82, 114) and probably is the consequence of the preferential transfer of chylomicron and VLDL surface material (PL and apo A-I and C) to these particles, as in humans and rats (127).

Increasing the secretion of TG-rich lipoproteins by the intestine with high fat diets (3) or by the liver in early lactation when NEFA concentrations are elevated (37) induces, in ruminants, massive intravascular lipolysis of these particles, as assumed from their rapid turnover (99) and a concomitant increase in very light HDL in the density range of LDL. Results were similar for young calves and adult cattle fed diets rich in polyunsaturated FA or cholesterol (2, 76) or for steroid-treated calves (111).

Moreover, in dairy cows, diets containing polyunsaturated FA increased plasma β-carotene, which was mainly associated with large HDL in the hydrophobic core (2). However, the transfer mechanism of β-carotene from plasma HDL to peripheral tissues (corpus luteum and mammary gland) is less efficient with diets rich in polyunsaturated FA, possibly because of a lower affinity of HDL to receptor sites (2, 85); therefore, this lower affinity might affect different physiological functions in cattle, namely, fertility.

Synthesis and Secretion of VLDL by the Liver

As in other mammals, the liver of ruminants esterifies acyl moieties that are taken up mainly from the blood (the hepatic synthesis of FA is very low) and that are subsequently secreted in the blood circulation as TG-rich VLDL (39, 68, 115, 119).

Although dairy cattle can mobilize large amounts of FA from adipose tissue to compensate for negative energy balance, and which are then taken up by the liver in proportion to concentrations in blood, VLDL secretion in

dairy cattle is generally thought to be very low compared with TG synthesis (61). This difference explains the moderate to severe hepatic lipidosis noted in one-third of periparturient, high yielding dairy cows (116). Capacity of ruminant liver to secrete VLDL was impaired in intact animals or in cultured hepatocytes. Upon inhibition of capillary LPL by Triton WR 1339, accumulation of plasma TG was slower in feed-deprived goats than in rats (119). In the same way, stable isotope enrichment (D^3 or [^{13}C]leucine) of plasma VLDL-apo B100 revealed a rate of hepatic VLDL secretion that was five times lower in calves (Leplaix, Durand, and Bauchart, 1992, unpublished data) than in humans (28). In animals with catheters implanted in the portal and hepatic veins, a net uptake of VLDL-TG by the liver has been determined in calves (9) and cows (115) that were deprived of feed, but the net hepatic output was slight but consistent in calves (5) and cows (115) that were fed and in sheep during fatty liver development (61). Slow secretion of hepatic TG in the medium was similar in primary monolayer hepatocyte cultures derived from nonmated, pregnant, or lactating ewes (39) and from goat wethers (68).

The general scheme for hepatic synthesis of VLDL constituents (TG, PL, free cholesterol, and apolipoproteins), VLDL assembly, and secretion in mammals is well established (45, 128). The apo B is synthesized in the rough endoplasmic reticulum and encounters lipids synthesized in the smooth endoplasmic reticulum at the junction of the two compartments. Transport vesicles carry the nascent VLDL to the Golgi apparatus, where terminal glycosylation of VLDL apolipoproteins occurs. Finally, secretion vesicles "bud off" from the Golgi and carry VLDL to the cell surface, where they fuse with the sinusoidal membrane, releasing VLDL into the blood via the space of Disse.

Little is known about the rate-limiting steps in VLDL production by the liver of ruminants. Local reuptake of newly secreted lipoproteins rich in apo B in the unstirred water layer of culture liver cells (Hep G2 cells), which possess LDL receptors (130), was hypothesized for ruminant liver (38). However, the quasi-absence of nascent VLDL in the hepatic Golgi fractions of the fed calves (Bauchart, Durand, and Chapman, 1988, unpublished data) indicates that biosynthesis and availability of VLDL constituents for VLDL packaging are more likely to be affected than the secretory processes.

The origin of the FA esterified into TG in the liver can modulate VLDL secretion. In rats, dietary conditions that favor de novo hepatic FA synthesis stimulate VLDL output, but fat mobilization or high fat diets that provide extracellular performed FA reduce the VLDL secretion [see the review by Gibbons (45)]. Therefore, the high rate of esterification of exogenous FA in ruminants during high fat mobilization may increase the TG cytoplasmic storage pool and reduce the TG secretory pool as previously suggested for rats (33). This hypothesis is supported by observations for dairy cows, for which hepatocyte apo B mRNA concentrations were lower in very early lactation than in the dry period or in midlactation (20). Moreover, FA turnover and the transfer quotient of TG determined in the dairy cows clearly showed that TG secretion was not enhanced in early lactation (104). However, in hepatocyte culture from lactating ewes, VLDL secretion was twice as high as in dry or pregnant ewes (39).

Rates of synthesis and release of apo B from the membrane of the secretory apparatus clearly control the hepatic secretion of VLDL in ruminants, as in other mammals (38). Whether transcription and translation of mRNA for apo B varied greatly according to dietary and hormonal conditions is not clear (105), but apo B production usually exceeded secretion (38). In cultured rat hepatocytes, degradation of apo B in the endoplasmic reticulum may determine the portion of apo B entering the VLDL assembly and secretion pathway (34). Recent studies (39) on sheep hepatocyte cultures showed that insulin, which stimulated intracellular degradation of newly synthesized lipoproteins rich in apo B (role on lipolysis) in rat hepatocytes, strongly reduced apparent hepatic TG export. However, those authors (39) did not observe changes with somatotropin treatment.

Among other PL, PC is specifically required for secretion of VLDL from the liver in mammals (128). Methionine, a methyl donor for PC synthesis and a possible limiting AA (together with lysine) for apo B synthesis, has been used for stimulating VLDL-TG secretion and milk yield. In high yielding dairy cows

equipped with catheters in portal and hepatic veins, perfusion of methionine and lysine in a mesenteric vein stimulated the net hepatic output of VLDL during early lactation (37). However, a methionine hydroxy analog, given in the diet of cows in early lactation, failed to stimulate TG secretion (104), perhaps because of the premature degradation of methionine hydroxy analog by rumen microorganisms (103).

The influence of other nutrients or hormones also has been tested. The addition of VFA (C_2, C_3, and iso-C_5) as an energy supplement or polyunsaturated FA ($C_{18:2n-6}$) as a PC precursor did not modify TG exported by hepatocytes isolated from lactating goats (1). Cholesterol, a potent inducer of apolipoprotein secretion (14), increased VLDL secretion in two calves equipped with catheters implanted in hepatic vessels but had no significant effect in three others (76). Estrogen, which stimulated hepatic production of VLDL in birds, led to complex effects in calves, as in humans. Short-term (1 h) portal infusion of 17β-estradiol stimulated VLDL output, but long-term (11 h) treatment by i.m. injection reduced secretion (5). In lactating and nonlactating cows, 17β-estradiol injection for 2 wk increased plasma VLDL during starvation (50). In other respects, blood infusion or i.m. injection of triiodothyronine strongly reduced VLDL concentration in calf plasma in portal (–75%) and hepatic veins (–80%) compared with those in the mesenteric artery, suggesting a reduction of VLDL production by the intestine and the liver (Durand, Auboiron, Chapman, and Bauchart, 1992, unpublished data).

DEGRADATION AND CATABOLISM OF LIPOPROTEINS

Metabolism of Lipoproteins Rich in TG

Following secretion and activation by apo CII, large chylomicrons and VLDL particles are rapidly metabolized by LPL (26), which is anchored by the membrane-bound heparin sulfate chain to the surface of the capillary endothelium (94). The hydrolysis of TG generates high amounts of FFA that are available to extrahepatic tissues—such as adipose tissue, heart, skeletal muscle, and lactating mammary gland (Figure 1)—and smaller particles termed "remnants" (from chylomicrons), and IDL (from VLDL). The comparative biochemistry and physiology of LPL have been reviewed by Cryer (32).

In lactating cows, the half-life of chylomicrons and VLDL (density range of <1.020 g/ml) is very short, ranging from 1.6 to 4.5 min (46, 99). Curve analysis of FA radioactivity disappearance in milk fat shows that 44 to 47% of milk FA was calculated to be of direct dietary origin via TG-rich lipoproteins (46, 99).

Formation and Catabolism of LDL

Bovine LDL particles are mostly generated by LPL activity from IDL fractions in plasma because hepatic lipase activity is lacking (40) or very low (30). The half-life of LDL is approximately 100 times that of VLDL (99) and depends on the physiological and nutritional status of the cows.

The work of Rudling and Peterson (118) in fetal and adult bovines shows clearly that plasma LDL are closely related to tissue LDL receptors. Quantitatively, LDL receptors are mostly produced by skeletal, intestinal, and hepatic tissues, whereas the tissue concentrations are highest in adrenal glands and in corpus luteum (118). As observed in the liver, during gestation, fetal (10, 44) and maternal LDL (113) in plasma decrease as a result of the increased LDL receptors in a period when estrogen concentrations increase in both maternal and fetal plasma. Conversely, after parturition, estrogen production declines rapidly and is accompanied by reduced LDL receptor proliferation and progressively increased LDL concentrations in plasma in postnatal calves (10, 62, 112) and in cows during early lactation (113, 122).

Nutritional treatments had variable effects on LDL metabolism. In calves fed cholesterol, the increase in the mean proportion of CE in LDL particles (41 vs. 31%) led to a twofold elevation of plasma LDL (76), which indicated, as with IDL, either lower affinity or reduced number of tissue LDL receptors. However, the apparent increase of LDL particles in calves fed high polyunsaturated FA diets (117) was strictly due to the accumulation of very light HDL, which exhibited a density range similar to that of their LDL counterpart (Leplaix,

Durand, and Bauchart, 1992, unpublished results).

Interaction of HDL with Cells

The main functions of HDL are to deliver cholesterol to tissues for primary steroidogenesis (liver, ovary, testis, and adrenal gland) or membrane synthesis (a wide variety of tissues such as fibroblasts) and for the transport of cholesterol away from tissues (fibroblasts and arterial smoother muscles) to the liver (Figure 1). Cholesterol uptake by tissues from HDL particles may take place by different pathways involving either internalization of the particles by receptor-mediated endocytosis (LDL or apo B, E receptor) or by cholesterol delivery independent of receptor-mediated endocytosis (51).

The probable absence of apo E in bovine HDL, as observed in plasma (9, 31, 70), intestinal lymph (70, 72), and follicular fluid (17), considerably limits HDL internalization mediated by the LDL receptor. However, specific, saturable high affinity binding sites for human HDL have been demonstrated in bovine liver membranes (85). The higher affinity of human HDL_2, HDL_3, and HDL_3 that is free of apo E—compared with HDL_3 that is enriched by apo E, pre-β HDL, LDL, or VLDL—for the bovine HDL receptor (85) suggested that all bovine HDL subclasses would specifically interact with HDL-binding sites of liver cells. In the ovarian steroidogenic cell system of the bovine, only small HDL (heavy HDL or HDL_3) were capable of crossing the basement membrane that separates follicular fluid and granulosa cells from blood circulation (16) and, therefore, interacted with HDL-binding sites. Similarly, cortisol secretion by corticotropin-stimulated adrenal zona fasciculata cells was mainly stimulated by bovine HDL cholesterol rather than by LDL cholesterol (29).

The HDL receptors promote reversible binding of HDL to the cell surface without internalization of lipoprotein particles. This binding induces cholesterol delivery to cells and, conversely, may be a mechanism by which HDL receptors facilitate cholesterol transport from cells (95).

The role of HDL apolipoproteins as ligands for the HDL receptor has been discussed by Grummer and Carroll (51). The apo A-I and C (and apo A-II in nonruminants) can bind the HDL receptor by the hydrophobic region of their helicoidal structure rather than by their primary sequence. This ability explains the very low capacity of HDL-binding sites by human pre-β HDL, in which apo A-I conformation is very distinct from that of conventional HDL (85). Interaction of HDL particles with HDL-binding sites, which probably function at nearly complete occupancy, is regulated by the affinity and the number of these sites, not by the concentration of circulating HDL (85).

The bovine HDL receptor, which consists of a single 110-kDa membrane protein, has been identified and characterized from aortic endothelial cells (48). Like its human counterpart, this cell surface protein binds HDL and apo A-I proteoliposomes, but not LDL or apo E particles (48).

CONCLUSIONS

This review has presented and discussed 1) the structure of lipoprotein particles and their role in lipid transport in ruminants and 2) the mechanisms of lipoprotein metabolism that control the partitioning of circulating FA and cholesterol between target tissues and the related cellular pathways of lipid metabolism.

The most promising area for future research in lipid transport in lactating and meat animals seems to cover the following regulatory factors: 1) the activities of lipid transfer between lipoprotein fractions, which modify the chemical composition of the particles and therefore their metabolism; 2) the relative importance of the intravascular lipolysis of TG-rich lipoproteins in adipose, muscle, and hepatic tissues (gene expression, availability, and activity of lipolytic enzymes); 3) the hepatic secretion of lipoproteins rich in apo B and their constituents, which control hepatic lipid metabolism (gene expression and intracellular proteolysis of apo B and the availability of apo B and TG for VLDL packaging); and 4) the tissue binding and uptake of LDL and HDL particles (the nature and role of ligands, such as apo B and apolipoproteins that exhibit apo E properties; identification, tissue distribution, and activity of the cellular receptors).

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