The Effect of Palmitic and Oleic Acids on the Properties and Composition of the Very Low Density Lipoprotein Secreted by the Liver*

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SUMMARY

Livers from normal fed male rats were perfused in vitro with equimolar quantities of either palmitic acid or oleic acids, and, after an experimental period of 4 hours, the very low density lipoprotein (VLDL) was isolated from the perfusate by ultracentrifugation in zonal rotors. The VLDL was characterized by rate-zonal mobility, by lipid composition, and by electrophoretic mobility on paper. Approximately 50% more VLDL triglyceride was secreted by the liver when oleate was the substrate than when palmitate was the substrate. The VLDL produced when palmitate was infused was a more dense particle and had a slower rate-zonal mobility under our conditions of zonal ultracentrifugation than did the VLDL produced from oleate. When palmitate was the substrate, the VLDL secreted by the liver was characterized by a single, apparently homogeneous peak in the density gradient in the zonal rotor, whereas the VLDL produced from oleate appeared to consist of at least two components; the lighter component behaved as a "chylomicron-like" particle in its rate-zonal mobility, whereas the heavier component was similar to the VLDL produced from palmitate. Both fractions derived from oleate and the lipoprotein derived from palmitate migrated on paper as a VLDL ("pre-β-lipoprotein"); the lipid composition of the chylomicron-like VLDL secreted by the liver after infusion of oleate was similar to that of the chylomicrons isolated from the perfusate at the beginning of the experiment although the chylomicrons remained at the origin and did not migrate as a VLDL on paper electrophoresis. The less dense VLDL produced from oleate, furthermore, had a lipid composition which differed from that of the VLDL produced from palmitate; the lighter VLDL produced from oleate contained half as much cholesterol and phospholipid per μmole of triglyceride (TG) than either the VLDL produced from palmitate or the heavier VLDL secreted after infusion of oleate. Even though more triglyceride was secreted per gram of liver when oleate was the substrate than when palmitate was infused, the total quantity of cholesterol and phospholipid secreted, respectively, in response to either fatty acid was the same, e.g. twice as much cholesterol and phospholipid was secreted per g of liver per μmole of VLDL triglyceride derived from palmitate than from the lighter lipoprotein produced from oleate. We can conclude from these data that certain physical properties and the lipid composition of the VLDL secreted by the liver were prescribed by infusion of either palmitate or oleate; it remains to be determined whether these observations can be extended to some general relationship describing effects of saturated and unsaturated free fatty acid (FFA) on the hepatic production and properties of the VLDL. The concentration of TG, phospholipid, and cholesterol in the VLDL of serum in vivo, which is the result, in part, of hepatic secretion of VLDL, must also be regulated by the chemical structure of the FFA from which the triglyceride fatty acids are derived.

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The secretion and accumulation of triglyceride by the isolated perfused liver from the normal fed animal is a function of the concentration of free fatty acid perfusing the liver, the chemical structure of the FFA,1 the extraction ratio for the fatty acid, and the duration of exposure of the liver to the FFA (1). It is of particular interest that the output of TG by the liver was observed to be proportional to the number of carbon atoms in the saturated FFA perfusing the liver, and to decrease as the number of double bonds in the FFA increased (1). It may be presumed that the concentration of TG in the serum of intact animals is determined, in part, by those same factors which were observed to dictate the rate of secretion of triglyceride by the liver in vitro.

1 The abbreviations used are: FFA, free fatty acids; TG, triglyceride; PL, phospholipid; CE, cholesteryl esters; VLDL, very low density lipoprotein ("pre-β-lipoprotein," density <1.006); LDL, low density lipoprotein; HDL, high density lipoprotein.
secreted by the liver in proportion to the amount of TG secreted, it may be postulated that regulation of secretion of cholesterol and phospholipid will be determined by the same factors which affect secretion of TG. The secretion of cholesterol and phospholipid into the VLDL may, indeed, be determined by the necessity of the liver to secrete TG. It can be predicted that more cholesterol and phospholipid would be secreted by the liver into the VLDL when long chain FFA are the substrate than when short or medium chain FFA are provided; it can also be predicted that more cholesterol and phospholipid would be secreted by the liver when saturated FFA are infused than when unsaturated FFA are infused. In order to test these hypotheses, it would be necessary to perfuse the liver with a variety of FFA and to determine the properties of the newly synthesized VLDL. We report here a comparison of various properties and lipid composition of the VLDL when either palmitic acid or oleic acid is the substrate for the formation and secretion of the lipoprotein by the liver.

**METHODS**

Livers from normal male rats (body weight 275 to 325 g) were perfused in vitro using procedures described previously (1). All animals received a balanced laboratory ration and water ad libitum. The liver was removed from the animal, positioned in the apparatus, and perfused for 20 min with a medium consisting of 80 ml of defibrinated rat blood and 60 ml of Krebs-Henseleit bicarbonate buffer, pH 7.4 (5); the medium was gassed continuously with 95% O₂ - 5% CO₂. Immediately following this initial period of equilibration, 70 ml of perfusate were removed for analysis, and an infusion of an albumin-FFA complex was started and was continued for the duration of the experiment. Four hours later the experiment was terminated and additional perfusate was removed for analysis. Equimolar quantities of palmitic acid or oleic acid were bound to albumin (708 moles/50 ml of 10% (w/v) bovine serum albumin in 0.9% (w/v) NaCl) and were infused at the rate of approximately 0.2 ml per min (1).

The perfusate was centrifuged briefly to sediment the erythrocytes, and the supernatant was fractionated into lipoprotein classes by ultracentrifugation in zonal rotors (6, 7). In these experiments, the cell-free perfusate was separated into identifiable lipoprotein fractions containing the lipids of chylomicrons, VLDL, LDL, and HDL plus residual heavier serum proteins by ultracentrifugation in the Spinco Ti-14 zonal rotor for 1 hour at 37,000 rpm in a NaBr gradient in the density range of 1.0 to 1.4. The fractions obtained from the zonal rotor were concentrated and dialyzed by membrane ultrafiltration, and aliquots were extracted with 20 volumes of CHCl₃-CH₃OH, 2:1 (v/v). The extracts were washed with 0.5 volume of aqueous 0.02% MgCl₂, dried in vacuo, dissolved in a small volume of CHCl₃, plated, and separated into lipid classes by thin layer chromatography on silica gel (2). The bands were visualized under ultraviolet light after spraying with 0.01% aqueous rhodamine 6G, scraped from the plates, collected in stoppered tubes, and the lipids were extracted from the bands with 10 ml of CHCl₃. Aliquots of the CHCl₃ extracts were analyzed for triglyceride (8, 9), cholesterol (10), and cholesterol esters (10). For estimation of lipid-soluble phosphorus, the appropriate band, still containing the silica gel, was digested directly without extraction, and inorganic phosphate was measured colorimetrically (11).

The dialyzed and concentrated lipoprotein fractions were characterized also by their electrophoretic mobility on paper (7, 12, 13).

**Fatty acids used in these experiments were purchased from**

Supelco, Inc., Bellefonte, Pa. Chemicals used were of reagent grade and solvents were redistilled before use. Plates for thin layer chromatography, precoated with silica gel (250 μ thick) containing an inert polymer binder, were purchased from Mann. The bovine serum albumin (Fraction V powder), obtained from Pentex, was extracted with isooctane glacial acetic acid, dialyzed, and lyophilized before being used (14). All animals were obtained from the Holtzmann Company, Madison, Wisconsin.

**RESULTS**

A description of the lipoprotein fractions isolated by zonal ultracentrifugation from the cell-free perfusate is presented in Fig. 1. The pattern observed when palmitic acid is infused is seen in Panel A; the pattern observed when oleic acid is infused is presented in Panel B. These lipoprotein profiles are reproducible for the specific substrate and are in no way atypical. Clearly, two dissimilar patterns may be distinguished. The pattern observed in the sample of cell-free perfusate removed prior to infusion of the fatty acids (e.g. zero hours) reveals chylomicrons (tubes 1 to 2, Fraction I), VLDL (tubes 3 to 11, Fraction II), LDL (tubes 12 to 18), and a residual fraction containing HDL and heavier serum proteins.2 Attention in this manuscript is directed toward Fractions I and II only. The fractions isolated by zonal ultracentrifugation were identified further by their electrophoretic mobility on paper (Fig. 2). Fraction I from the sample obtained at zero time remained at the origin as expected; Fraction II behaved as a VLDL ("pre-β-lipoprotein"), and the contents of tubes 12 to 18 behaved as an LDL ("β-lipoprotein"). After perfusion of the liver with oleic acid, both Fractions I and II were characterized by pre-β mobility, as was also Fraction II when palmitic acid was the substrate. Fraction I, the chylomicron-containing fraction, diminished after infusion of palmitic

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2 H. G. Wilcox and M. Heimberg, unpublished information. See also References 6 and 7.
acid, and an increase in the quantity of Fraction II, the VLDL was observed (Fig. 1). The peak of Fraction II was located at a density of 1.02 in the gradient; this density is not the isopycnic density of the fraction since equilibrium was not achieved under the conditions of rate-zonal isolation used in these experiments. After oleic acid was infused, an increase in the absorption in Fraction I was observed; a prominent shoulder on the absorption curve extended into Fraction II. The pattern observed after infusion of oleic acid was suggestive of the possibility that more than one species of triglyceride-containing particle was being secreted by the liver; these subfractions, however, were not separated under these conditions of centrifugation. When the liver was perfused with palmitic acid, a single species of VLDL appeared to be secreted.

The percentage composition of the lipids in Fractions I and II is presented in Table I. The chylomicrons and VLDL isolated from the cell-free perfusate prior to infusion of the fatty acids, as expected, differed in apparent density, in rate of migration in the zonal rotor, and in lipid composition; the VLDL contained relatively less triglyceride and relatively more cholesterol and phospholipid than did the chylomicrons (A versus C). During perfusion, there appeared to be a decrease in the percentage of cholesteryl esters in both Fraction I and Fraction II (p < 0.05). Both VLDL fractions (I and II) secreted when oleate was infused differed in lipid composition (B versus D), in that the lighter particle contained more TG and less phospholipid and cholesterol than did the heavier particle. Similarly, the lighter particle contained more TG and less phospholipid and cholesterol than did the heavier VLDL secreted by the liver when palmitic acid was the substrate (B versus E). The heavier particle secreted after oleate and the VLDL secreted after infusion of palmitate appeared to be identical in percentage composition of lipids (D versus E). Of particular interest is the observation that the liver secreted a chylomicron-like particle when oleic acid was infused (A versus B). The lipid composition of the chylomicrons (A) was similar to that of the VLDL (B); however, after infusion of the fatty acid, the lipoprotein had electrophoretic and immunological properties typical of a VLDL, whereas the chylomicron particle did not. In experiments with palmitic acid, the chylomicrons disappeared from the medium during perfusion. The amount of lipid (about 2 to 4 μmoles of triglyceride, 3 to 5 μmoles of total lipid) in the small quantity of chylomicrons present in the medium could have been removed and metabolized by the liver during 4 hours of perfusion (15).

The total output of triglyceride by the liver when palmitic acid was infused was less than when oleic acid was infused (Table II). When oleic acid was infused, two-thirds of the TG was secreted into the lighter VLDL (Fraction I) and one-third into the

Table I

| Group   | TG       | PL       | C        | CE       |
|---------|----------|----------|----------|----------|
| A versus C | 0.01     | 0.01     | 0.05     | NS       |
| B versus D | 0.025    | 0.02     | 0.01     | NS       |
| B versus E | 0.001    | 0.01     | 0.005    | NS       |

Significance of differences: (p values)
Table II
Output of VLDL lipids by liver

The data are expressed as micromoles of lipid secreted per g of liver per 4 hours, ± standard error. The calculation for the ratio of TG:PL:C (cholesterol) secreted by the liver considers TG to be equal to 1.00. The statistical calculation evaluates the significance of the differences between the ratios for the experimental groups. NS indicates a p value >0.05. Numbers in parentheses indicate the fractions from the zonal rotor which were combined and analyzed.

| Substrate     | Lipid fraction |       |       |       |
|---------------|----------------|-------|-------|-------|
|               | TG             | PL    | C     | CE    |
| Palmitic acid |                |       |       |       |
| A. (1-11)     | 2.06 ± 0.24    | 0.58  | 0.25  | -0.03 |
|               | ± 0.08 ± 0.05  | ± 0.05| ± 0.05| ± 0.03|
| Oleic acid    |                |       |       |       |
| B. (1-11)     | 2.95 ± 0.12    | 0.55  | 0.21  | 0.06  |
|               | ± 0.05 ± 0.05  | ± 0.04| ± 0.02|       |
| C. (1-2)      | 1.99 ± 0.19    | 0.30  | 0.11  | 0.05  |
|               | ± 0.02 ± 0.02  | ± 0.01|       | ± 0.01|
| D. (3-11)     | 0.98 ± 0.08    | 0.23  | 0.11  | 0.02  |
|               | ± 0.03 ± 0.03  | ± 0.03|       | ± 0.03|

Ratio:               |       |       |       |       |
| A. 1.00 : 0.28 ± 0.02 : 0.12 ± 0.02 |
| B. 1.00 : 0.19 ± 0.02 : 0.07 ± 0.02 |
| C. 1.00 : 0.15 ± 0.02 : 0.06 ± 0.01 |
| D. 1.00 : 0.26 ± 0.01 : 0.11 ± 0.03 |

Significance of differences: (p values)

A vs B, 0.05           NS
A vs C, 0.01           0.05
C vs D, 0.005          0.05

heavier VLDL (Fraction II). Despite the difference in output of TG, the absolute output of phospholipid and cholesterol into the VLDL was of the same order of magnitude with both fatty acids. If the output of phospholipid and cholesterol relative to TG is calculated, it can be determined that the VLDL secreted when palmitic acid was infused required more phospholipid and cholesterol for an equivalent quantity of TG than when oleic acid was infused. Furthermore, the VLDL secreted when palmitic acid was the substrate, or the heavier fraction of the VLDL which was secreted when oleic acid was the substrate, contained approximately twice the moles of phospholipid and cholesterol relative to TG than did the lighter VLDL (Tables I and II). Indeed, the ratio of triglyceride to phospholipid to cholesterol secreted in the heavier VLDL when oleic acid was infused was identical with that in the VLDL when palmitic acid was the substrate. It is of additional interest that cholesteryl ester was not secreted when palmitic acid was infused; only a small increment in cholesteryl ester (0.06 ± 0.02 μmoles per g of liver, wet weight per 4 hours) was observed when oleic acid was the substrate, and most of this increment was in the lighter VLDL particle.

DISCUSSION

It is most probable that the output of TG by the liver is a function of the chemical structure and the total uptake of free fatty acids by that organ; the secretion of cholesterol and phospholipid into the VLDL, in turn, probably is determined by the effects of the FFA on the secretion of TG. Since in the post-absorptive state, plasma triglycerides are derived primarily from the liver, the concentration of plasma TG in vivo will, in part, depend on the quantity and properties of the fatty acids which come to the liver either as triglyceride fatty acids from intestinal lymph during absorption, or as FFA released by adipose tissue. The composition of the fatty acids from both sources will be modified by the diet and should determine the quantity and structure of the VLDL and TG fatty acids secreted by the liver; fatty acids derived by biosynthesis from carbohydrate in adipose tissue or liver should also contribute to the secretion of TG by the liver.

It is of interest, therefore, as reported in this manuscript, that important differences in the total output of TG and in the properties of the VLDL secreted by the liver can be observed after perfusion of the liver with either palmitic acid or oleic acids. Although the total uptake of either fatty acid by the liver was not measured in these experiments, it can be presumed that uptake of both was identical (1); furthermore, although the fatty acid composition of the secreted TG was not analyzed by gas-liquid chromatography in these experiments, it can be presumed that the composition favors the infused FFA (1). Clearly, it would be desirable to compare a larger variety of saturated and unsaturated FFA, and long or short chain FFA, to determine whether any general relationships exist between the structure of the infused FFA and the properties of the secreted VLDL. The differences in the properties of the secreted VLDL observed with palmitic or oleic acids are of sufficient magnitude to allow one to postulate such generalities. The VLDL secreted by the liver when palmitate was infused had a lower percentage of TG and a higher percentage of cholesterol and phospholipid than did the less dense lipoprotein produced when oleate was infused. The VLDL secreted when palmitate was the substrate in all probability had a higher mean density (although its isopycnic point was not measured) and had a slower flotation rate in the zonal rotor under our conditions of centrifugation than did the lipoprotein when oleate was the substrate; this apparent difference in density would be expected on the basis of the relative proportions of TG, phospholipid, and cholesterol in the respective lipoprotein fractions (16). The VLDL produced when oleic acid was infused was probably a heterogeneous mixture, consisting of at least two distinct kinds of particles: about two-thirds of the total TG was "chylomicron-like" (Fraction I in the zonal rotor), had a composition of lipids similar to that of chylomicrons isolated from normal rat serum, but was a VLDL (pre-β-lipoprotein) as judged by electrophoretic mobility on paper. The heavier fraction (Fraction II) secreted in response to the infusion of oleic acid appeared identical with the VLDL produced from palmitic acid, and was similar to the "endogenous" VLDL secreted when fatty acid was omitted from the medium. Another observation, which may be of importance, can be derived from these studies. Although more TG was released when oleate was infused than when palmitate was infused, the absolute amounts of cholesterol and phospholipid secreted were, respectively, approximately the same in both treatment groups. Theoretically, then, if equal quantities of TG were produced by the liver from either fatty acid, more phospholipid and cholesterol would be secreted when palmitate was the substrate than when oleate was the substrate. Similar variations have been observed in the properties of the VLDL of intestinal origin during absorption of different long chain fatty acids by the rat in vivo. Ockner et al. investigated the distribution of triglyceride and cholesterol in the
chylomicrons and VLDL of intestinal lymph during the absorption of fatty acid from mixed micelles containing monoolein, taurocholate, and either palmitate, oleate, or linoleate (17, 18). The percentage (w/w) of TG in the chylomicrons was somewhat higher when either oleate or linoleate was administered than when palmitate was given; conversely, the percentage of TG in the VLDL after administration of palmitate was approximately twice that after oleate or linoleate. Of particular interest was the observation that the percentage (w/w) of cholesterol in the chylomicrons was higher when oleate or linoleate was given rather than palmitate, and that the percentage of cholesterol in the VLDL was higher after administration of palmitic acid than after oleate or linoleate. The data of Ockner et al. (17), when recalculated in molar terms (Table III), can be compared directly with the data of Table II. Their data on the absorption of fatty acids (e.g. the secretion of lipoproteins into lymph by intestinal mucosa) may be considered the equivalent of our data on the output of lipoprotein-lipids by the liver into the serum. The exciting aspect about these data is their remarkable similarity. If one calculates the ratio of micromoles of triglyceride to phospholipid to cholesterol secreted into the lipoprotein fractions per unit of time per unit of triglyceride by liver or small bowel, it can be estimated that per 1000 molecules of TG, 50 to 60 molecules of cholesterol were secreted into intestinal chylomicrons or into the chylomicron-like VLDL derived from the liver, regardless of which fatty acid was the substrate. Similarly, about 120 molecules of cholesterol were secreted per 1000 molecules of TG into the intestinal VLDL and into the heavier hepatic VLDL, regardless of which fatty acid was the substrate. Palmitate, however, gave rise to the heavier VLDL exclusively in the liver, whereas oleate resulted in formation of both particles. Palmitate, oleate, and linoleate all gave rise to both chylomicrons and VLDL by the intestinal mucosa, although palmitate increased the proportion of fatty acid transported in the VLDL triglyceride. It is possible that even a greater fraction of the palmitate might have been secreted in the heavier VLDL had monoolein not been a component of the micellar solution. These data emphasize the similarity of the VLDL secreted by liver and intestinal mucosa, both of which can contribute to the total pool of serum VLDL in the intact animal.

One might reasonably inquire why palmitate is incorporated by liver and intestinal mucosa into a lipoprotein particle that contains more phospholipid and cholesterol relative to TG than does the lipoprotein produced from oleate or linoleate. If the primary purpose of the VLDL is to transport TG, and if phospholipid and cholesterol are incorporated into the lipoprotein in some fixed ratio to TG, it is probable that cholesterol and phospholipid are required to effect some specific configuration of the lipoprotein which allows it to carry the TG in the aqueous environment of the blood. The polar hydrophilic groups of the phospholipid, and the sterol hydroxyl, may participate in this function. Palmitic acid (m.p. 63.1) and tripalmitin (m.p. 65.1) are solids at room temperature, having higher melting points than do oleic acid (m.p. 13.4) and triolein (m.p. -4.0). Furthermore, the solubility of oleic acid in polar solvents exceeds that of palmitic acid. These important differences in the physical properties may mean that more of the polar cholesterol and phospholipid are required to stabilize the VLDL which contains a higher proportion of its triglyceride fatty acids as palmitate than as oleate; possibly, a more general relationship may obtain for VLDL triglyceride fatty acids produced from saturated and unsaturated fatty acids, respectively. The protein moiety of the VLDL, which must also be required to stabilize the structure of the lipoprotein and allow transport of TG to occur, may also vary quantitatively, depending on whether the triglyceride fatty acids are derived from saturated or unsaturated fatty acids.

It is probable that the different triglyceride-containing lipoproteins which are secreted by the liver and intestine contribute unequally to the relative quantities of triglyceride, phospholipid, and cholesterol in the serum lipoproteins with density <1.006. It should be considered that blood levels of these lipids depend not only on the disparate particles secreted in response to varying fatty acid substrates, but also on unequal rates of utilization of these lipoproteins by various tissues. The larger, less dense particles, which contain higher proportions of TG, may be utilized more rapidly (19), leaving behind the smaller particles with relatively larger concentrations of cholesterol and phospholipid. If saturated fatty acids preferentially yield the more dense VLDL, which contains a higher ratio of cholesterol and phospholipid to triglyceride, and if these lipoproteins are also utilized at a slower rate, it might account for the lipid-lowering effect of unsaturated fatty acids in comparison to the saturated fatty acids.

**Table III**

Lipids in intestinal lymph during absorption of fat

| Substrate | Lipid fraction |
|-----------|--------------|
|           | Chylomicrons | VLDL |
|          | TG | C | TG | C |
| A. Palmitate | 13.47 | 0.72 | 6.06 | 0.72 |
| B. Oleate | 18.18 | 0.88 | 3.62 | 0.42 |
| C. Linoleate | 16.00 | 0.88 | 3.38 | 0.43 |

**Ratio:**

| Substrate | TG | C |
|-----------|----|----|
| A. Palmitate | 1.00 | 0.053 |
| B. Oleate | 1.00 | 0.049 |
| C. Linoleate | 1.00 | 0.055 |

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