A New Apoprotein of Human Plasma Very Low Density Lipoproteins* 

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SUMMARY

A new apoprotein has been isolated from delipidated human very low density lipoproteins by Sepharose 6B chromatography in 6 M guanidine hydrochloride. This water-insoluble protein was noted to be unique to the plasma very low density and chylomicron fractions. This protein was shown to be homogeneous by urea-DEAE-cellulose chromatography, rechromatography in a guanidine-Sepharose system, sodium dodecyl sulfate polyacrylamide gel electrophoresis, and isoelectric focusing. The molecular weight of this apoprotein obtained by guanidine gel filtration, sedimentation equilibrium in guanidine hydrochloride, and by sodium dodecyl sulfate polyacrylamide gel electrophoresis was 33,000. The amino acid content was significantly different from any previously characterized very low density lipoprotein apoprotein, containing relatively more arginine. By the dansylation and cyanate techniques the NH₂-terminal amino acid was found to be lysine. Digestion with carboxypeptidase revealed the COOH-terminal sequence to be -Leu-Ser-Ala-COOH.

The major apoproteins of human plasma very low density lipoprotein consist of a group of low molecular weight proteins referred to as D proteins (1, 2), and a larger sized apolipoprotein (3). Each of these have been estimated to account for between 40 and 50% of the protein content of human VLDL (3). These previously characterized human VLDL proteins are not unique to this lipoprotein fraction and are isolated with other lipoproteins. The apo-LDL protein appears to be the predominant, if not the only protein of LDL (5), and the D proteins have been recovered in the human high density lipoprotein fraction (6). The isolation and characterization of a new apoprotein from delipidated human VLDL is presented here. This apoprotein appears to be unique to the triglyceride-rich plasma lipoproteins of man.

MATERIALS AND METHODS

Guanidine hydrochloride was purchased from Heico, Inc., Delaware Water Gap, Pennsylvania. Sepharose 4B and 6B were obtained from Pharmacia Fine Chemicals, Inc., Piscataway, N. J., and DEAE-DEAE-cellulose from W. and R. Balston, Ltd., England. Utrapure urea was purchased from Schwarz-Mann, Orangeburg, N. Y. Carboxypeptidases A and B, which had been treated with diisopropyl phosphofluoridate, were purchased from Worthington Biochemicals, Freehold, N. J. Dansyl chloride and kits containing dansyl amino acids were obtained from Pierce Chemical Co., Rockford, Ill. Polyamide sheets were purchased from the Cheng Chin Trading Co., Ltd., Taipei, Taiwan. Reference proteins were the following: serum albumin from Hyland Division of Travenol Laboratories, Inc., Los Angeles, Calif.; ovalbumin and chymotrypsinogen from Worthington Biochemicals; and lysozyme from Boehringer Mannheim Corp., New York, N. Y. Ampholines were obtained from LKB Instruments, Inc., Rockville, Md.

Apoprotein Preparation—Blood was collected from humans with normal and with elevated plasma VLDL concentrations (type IV hyperlipoproteinemia) in disodium EDTA (1 mg per ml). All subjects were sampled after a 14-hour fast. The plasma was separated from the red cells by centrifugation at 2,500 rpm for 20 min in a Lourdes LIA refrigerated centrifuge at 4°C. Lipoproteins were isolated by a modification of the method described by Havel et al. (7). Chylomicrons were removed from the plasma of each subject by ultracentrifugation at the native density of plasma (1.006) for 25 min at 20,000 rpm (1 X 10⁸ X g-min) in a Spinco model L2 ultracentrifuge using a 40 rotor. VLDL (S₁₅, 20-400) were isolated from the chylomicon-free plasma by ultracentrifugation at 40,000 rpm for 18 hours (1 X 10⁸ X g-min) at density 1.006 and 8°C. The VLDL were floated through saline twice by a similar ultracentrifugation and were dialyzed against 200 volumes of 0.1% EDTA buffered to pH 7.4 prior to delipidation. The VLDL were delipidated at 4°C by three extractions with ethanol-diethyl ether (3:1), using 50 volumes of solvent per volume of lipoprotein. The apoproteins were then washed twice with 50 ml of diethyl ether. Less than 0.5% of the delipidated proteins were phospholipid, as determined by the method of Ames et al. (8). No other lipids could be detected.

DEAE-cellulose Chromatography—The chromatography was carried out by a procedure having a similarity to that of Shore and Shore (9) and Brown et al. (1). The DEAE-cellulose was successively equilibrated with 0.05 M Tris, pH 8.2; 0.005 M Tris, pH 8.2; and 0.005 Tris-6 M urea, pH 8.2. A column (1.5 X 20 cm) was poured, a 15-mg sample of apo-VLDL was applied in 3.0 ml of the equilibrating buffer, and the column was run in a descending fashion at a rate of 80 ml per hour. A linear gradient of Tris buffer was used. The starting buffer was 0.005 M Tris-6 M
urea, pH 9.2; and the limiting buffer was 0.1 M Tris-0.04 M NaCl-6 M urea. The eluate was monitored by determining the absorbance at 280 nm.

Polsyamide Electrophoresis—The procedure used was that of Weber and Oeoborn (10). The electrophoresis system, model 4010-1, was purchased from Orteet, Inc., Oak Ridge, Tenn. Gels of 10% acrylamide were prepared in 0.1 m phosphate buffer, pH 7.0, and were cast in polyethylene tubes of diameter slightly greater than about 40 m. The gel was then dried with a mixture containing 7.5% acetic acid and 5% methanol in water at 60° with changes of solution every 12 hours until completion.

Gel Isoelectric Focusing—Gel isolectric focusing (11) was performed on 5% gels prepared by using the following proportions of reagents for 10 ml of gel mix: 30% acrylamide, 1.67 ml; 1.6% bisacrylamide, 1.55 ml; 0.05 M triethanolamine hydrochloride, 0.02 ml; 10% acrylamide, 5.7 ml; 0.05 M pH 7 to 10 ampholines, 0.15 ml; water, 1.0 ml. A 2.9-ml aliquot of the above mix was mixed with 0.1 ml of sample in 10 m urea and 0.05 ml of ammonium persulfate solution (10 mg per ml). The resulting solution was poured into 0.5-cm tubes to a height of 10 cm. Isoelectric focusing was carried out in an apparatus obtained from Hoefer Scientific Instruments. The samples were placed in the gel cradle, and the gel was allowed to equilibrate with 1&10000 voltage of 300 volts at 4° for 4 hours. The lower tank buffer was 1.4% orthophosphoric acid and the upper tank buffer was 1.0% ethylenediamine. The gels were removed from the tubes and stained and destained according to the method of Fish et al. (12).

Sepharose Column Chromatography—Chromatography was performed by the method of Fish et al. (13). Solutions (6 m in guanidine hydrochloride) were prepared using crystals which had been dried in a vacuum desiccator over sodium hydroxide. Approximately 200 ml of gel slurry (Sepharose 4B or 6B) were washed on a sintered glass funnel with 6 M guanidine chloride. The resulting slurry was poured into a 1.5-cm Pharmacia column to a height of 85 cm. The column was equilibrated with the solvent for 48 hours after which it was run in a descending fashion at a flow rate of 2 ml per hour. Void volumes were determined with blue dextran 2000 and total volumes were determined with oxidized mercaptoethanol. Columns were calibrated using 10-ml samples of reference proteins. Usually 25-ml samples of VLDL apoprotein were applied to the column. Proteins were dissolved in 1.0 ml of 6 m guanidine chloride and 1.6 ml of mercaptoethanol and were allowed to react for 16 hours at room temperature. Carbamylation was performed by the addition of 10 ml of iodoacetic acid, keeping the mixture at room temperature for 3 hours. The sample was centrifuged before application to the column to remove trapped air bubbles. Fractions of 1.5 to 2.0 g were collected and the elution of protein was monitored by the absorbance at 280 nm.

Amino Acid Analysis—The analyses were carried out according to the procedure of Speckman et al. (14). The procedure was that of Spackman et al. (14). The analyzer was a Beckman model 121. Protein samples were routinely hydrolyzed for 24 hours with oxygen-free 6 M hydrochloric acid at 108° in a nitrogen atmosphere. The results were corrected for the loss of threonine and serine. The hydrolysis was carried out for 54, 48, 72, 96, and 120 hours, and the results were extrapolated to zero time to determine the true value of these two amino acids. Hydrolysis were dried in a vacuum desiccator over sodium hydroxide and dissolved in 0.005 N HCl for application to the analyzer column.

Tryptophan was determined by the method of Sussle et al. (15). The protein was reacted with o-nitrophenylsulfonyl chloride and the excess reagent was removed with acetone-ether, 1:1. The residue was dissolved in 80% acetic acid and the absorbance of the solution was read at 356 nm.

NH2-Terminal Amino Acid Analysis—A qualitative NH2-terminal amino acid analysis was performed by a procedure similar to that of Woods and Wang (16). Approximately 25 moles of protein were dissolved in 0.5 ml of freshly deionized 10 M urea. To this solution was added 0.5 ml of dansyl chloride solution (10 mg per ml in ace-
tone). The reaction mixture was kept at 37° for 30 min. A column of Sephadex G-50 (1.5 X 25 cm) was prepared in 6 M guanidine hydrochloride. The entire reaction mixture was placed on the column and elution was carried out with 7 M guanidine hydrochloride. The elution of dansyl protein was determined by reading the absorbance of the 1.0-ml fractions at 325 nm. The appropriate fractions were combined and dialyzed through three changes of distilled water. The residue was then taken up in 50 ml of 1% pyridine-water for application to polyamide sheets (7.5 X 7.5 cm). Reference dansyl amino acids were applied to the opposite side of the sheets. The sheets were developed in two dimensions with 1.5% formic acid and with benzene-acetic acid (9:1) as described by Hartley (17). The dansyl amino acids were visualized under ultraviolet light.

Quantitative NH2-terminal analyses were performed by the procedure exactly as described by Blaek (18). A blank was run by omitting both urea and cyanate from the reaction mixture.

COOH-Terminal Amino Acid Analysis—The analyses were performed by procedures similar to those described by Amblor (19). Approximately 7 moles of maleylated protein was dissolved in 1.0 ml of 0.1 M pH 7.0. An enzyme mixture containing 1 ml of 10% LiCl, 8 &10 of carboxypeptidase A (75 mg per ml), and 8 &10 of carboxypeptidase B (10 mg per ml) was prepared, and 40 &10 were added to the substrate. The reaction was allowed to proceed to 37° for 1, 3, and 5 hours, and stopped by the addition of 1 ml of 1 N HCl. The solution was frozen-dried and 1.4 ml of 0.005 N HCl was added for transfer to the analyzer column. Blank reactions containing only the enzymes and not the VLDL protein were run with each determination.

Protein Analysis—Protein determinations were performed according to the method of Lowry et al. (20) using bovine albumin as a standard. Column eluates were dialyzed against 0.05 M phosphate buffer, pH 7.0, to remove guanidine hydrochloride before protein analysis was carried out.

Protein Modifications—Maleylation of protein was performed according to the procedure of Butler et al. (21). A 1.6-ml portion of apoprotein in 4.0 ml of 6 M guanidine hydrochloride was adjusted to pH 8.5 with solid potassium hydrogen phosphate. The solution was chilled to 0° in an ice bath and 15 mg of freshly ground maleic anhydride were added over a period of 15 min. The pH was maintained at 8.5 by the addition of 4 M guanidine hydrochloride. Guanidine hydrochloride and residual maleate were removed by dialysis against 0.005 phosphate buffer, pH 7.5.

Sedimentation Equilibrium—Sedimentation equilibria in 6 M guanidine hydrochloride were measured by the technique of Thomas and Edelstein (22). Apoprotein solutions of 0.2 mg per ml were run at 20,000 and 22,000 rpm at 25° in a Beckman model E analytical ultracentrifuge equipped with a photoelectric scanner. The partial specific volume was obtained from the amino acid composition (23) and the molecular weight calculated by a standard method (24).

RESULTS

Sepharose Column Chromatography—The results of Sepharose 6B-guanidine hydrochloride column separation of reduced and alkylated apo-VLDL are shown in Fig. 1A. Peak I appearing in the void volume of the 6B column was demonstrated by chromatography on the 4B system and by dodecyl sulfate polyacryl-
amide gel electrophoresis to be a protein with a molecular weight of about 300,000, approximately the same as that described by Smith et al. for apo-LDL (5). The protein was immunochemically identical to apo-LDL. Peak II was shown by similar techniques to be of the same molecular size as the D proteins described by Brown et al. (1, 2, 4), and reacted with antisera to apo-Glu and apo-Ala VLDL proteins. Peak II had a molecular size unlike any previously described VLDL apoprotein.
FIG. 1. A, guanidine hydrochloride-Sepharose 6B column separation of delipidated VLDL. Peak I is apo-LDL, Peak III contains the D proteins, and Peak II is a new apoprotein. VLDL was reduced with mercaptoethanol and alkylated with iodoacetic acid. Solvent is 6 M guanidine hydrochloride. B, rechromatography of Peak II on the same guanidine hydrochloride-Sepharose 6B column.

When Peak II fractions were concentrated and rechromatographed on the same system the protein was eluted in identical column fractions (Fig. 1B).

**DEAE-cellulose Chromatography**—When total apo-VLDL was fractionated on the urea-DEAE cellulose system six apoprotein peaks were obtained. The column fractions were monitored at 280 nm and each peak was evaluated in a sodium dodecyl sulfate polyacrylamide gel electrophoresis system. The Peak II protein was eluted as a single peak, just after the second protein peak (apo-Glu) was eluted.

**Polyacrylamide Gel Electrophoresis**—The results of electrophoresis are shown in Fig. 2. The Peak II protein had a migration slightly greater than ovalbumin (mol wt 43,000) and slightly less than chymotrypsinogen (mol wt 27,000). Disulfide reduction did not change the migration of this protein, suggesting that no intra- or intermolecular disulfide bands were present. No carbohydrate could be detected in the Peak II band by periodic acid Schiff staining.

**Gel Isoelectric Focusing**—Isoelectric focusing runs of the Peak II protein indicate a single band with a pI between 9 and 10 (Fig. 3). Occasionally the 23,000 major apo-HDL protein was observed as a minor component on the isoelectric focusing runs. This amounted to a maximum of 5% of the total sample when evaluated by gel scanning densitometry.

**Solubility Properties of Peak II Protein**—Delipidated VLDL proteins were separately incubated at 37° on a mechanical shaker in 0.12 M Tris HCl, 0.03 M NaCl, pH 7.4, and 0.05 M phosphate, pH 7.0, buffers for 3 hours. The water-soluble peptides were determined in the supernatant after centrifuging the mixture at 6000 rpm for 20 min. The VLDL Peak II protein was not found among the aqueous soluble VLDL proteins in other than trace amounts by either the sodium dodecyl sulfate polyacrylamide gel electrophoresis system or by Sepharose-guanidine chromatography. Purified Peak II protein was found to be sparingly soluble in large volumes of aqueous buffer.

**Amino Acid Analysis**—The results of the amino acid analysis of 24-hour hydrolysates of protein from Peak II are shown in Table I. It will be noted that no cysteine is present. This is to be expected as a result of the reduction and alkylation procedure. However, carboxymethylcysteine was also not found, indicating that no cysteine was present in the native protein. The amino acid analyses were compared with those reported for other previously isolated and characterized components of VLDL (1-4). The new Peak II protein was distinctly different, particularly in its higher arginine content. It was similar to the other VLDL apoproteins in its low content of cysteine. The high arginine content of this protein is similar to that of DEAE Fractions 4, 5, and 6 of human delipidated VLDL described by Shore and Shore (26). Although other similarities between Peak II amino acid composition and that of Shore and Shore's proteins were observed, substantial differences were also noted.

**Terminal Amino Acid Analysis**—The only dansyl amino acid found after hydrolysis migrated with didansyl lysine in the two-dimensional solvent systems (Fig. 4). The quantitative results of the Stark procedure also indicates lysine to be the NHz-termi-
Table I: Amino acid composition of Peak II from VLDL

| Amino acid   | Moles/mole of protein | Moles/10^3 moles amino acid |
|--------------|-----------------------|-----------------------------|
| Lysine       | 12.19 ± 0.6           | 49.0                        |
| Histidine    | 1.91 ± 0.3            | 7.7                         |
| Arginine     | 22.68 ± 0.7           | 91.2                        |
| Aspartic acid| 16.93 ± 0.9           | 68.1                        |
| Threonine    | 11.33 ± 1.0           | 45.5                        |
| Serine       | 14.99 ± 0.3           | 60.3                        |
| Glutamic acid| 44.47 ± 2.2           | 178.8                       |
| Proline      | 9.47 ± 0.3            | 38.1                        |
| Glycine      | 15.71 ± 0.9           | 67.2                        |
| Alanine      | 25.94 ± 0.6           | 104.3                       |
| Cysteine     | 18.32 ± 0.4           | 73.6                        |
| Valine       | 1.69 ± 0.4            | 6.8                         |
| Methionine   | 4.38 ± 0.5            | 17.6                        |
| Isoleucine   | 29.85 ± 0.9           | 120.0                       |
| Leucine      | 4.38 ± 0.6            | 17.5                        |
| Tyrosine     | 5.29 ± 0.4            | 21.3                        |
| Phenylalanine| 8.0                   | 32.2                        |

* Based on molecular weight = 33,000. Samples were obtained from two normals and two subjects with increased VLDL and hydrolyzed for 24 hours in 6 M HCl at 110°. The maximal values for valine and isoleucine and the loss of threonine and serine were determined from more prolonged hydrolysis (see text). The data are presented ± standard error of the mean.

† No carboxymethylcysteine was found in the reduced alkylated samples.

‡ This value is a mean of two individuals obtained by a method detailed in Ref. 14.

Table II: Cyanate analysis of Peak II proteins

| Amino acid | Moles/mole of protein |
|------------|-----------------------|
| Serine     | 0.15                  |
| Glycine    | 0.05                  |
| Alanine    | 0.10                  |
| Lysine     | 1.10                  |

* Determined by the method of Stark (17), and calculated on the basis of a molecular weight of 33,000.

Table III: Amino acid analysis of carboxypeptidase digestion of Peak II

| Amino acid | Moles/mole of protein |
|------------|-----------------------|
| Valine     | 0.70                  |
| Leucine    | 0.52                  |
| Serine     | 0.57                  |
| Alanine    | 0.60                  |
| Lysine     | 0.44                  |

* Based on a molecular weight of 33,000 and corrected for blank runs.

† Time of digestion at 37°.

**Most of the apoproteins of human VLDL that have already been isolated and defined have also been observed in other lipoprotein fractions of plasma. The new protein which we have identified appears to be unique to the triglyceride-rich lipoproteins of human plasma. It is found predominantly in VLDL and in chylomicrons, and occasionally in trace amounts in the S 12 to 20 LDL fraction.**

Discussion

Most of the apoproteins of human VLDL that have already been isolated and defined have also been observed in other lipoprotein fractions of plasma. The new protein which we have identified appears to be unique to the triglyceride-rich lipoproteins of human plasma. It is found predominantly in VLDL and in chylomicrons, and occasionally in trace amounts in the S 12 to 20 LDL fraction.
FIG. 5. Sedimentation equilibrium analysis of Peak II from
VLDL in 6 M guanidine hydrochloride at 25°. Plot of In absorb-
ance versus r², where r is the distance from the axis of rotation.
A, 20,000 rpm (●); B, 22,000 rpm (○).

The protein was isolated from VLDL of both hyperlipopro-
teinemic and normal humans. The molecular weight of 33,000
found for this new protein differs from the 250,000 obtained for
the apo-LDL protein (5), 7,000 to 10,000 for the D proteins, and
the 28,000 and 17,000 values obtained for the major HDL pep-
tides (26). The uniqueness in size of this new lipoprotein apo-
protein has aided its isolation and identification. Although it
has the same COOH-terminal amino acid as the apo-Ala protein
of human VLDL, it has a different COOH-terminal sequence, a
different NH₂-terminal amino acid, and a different amino acid
content. It is obviously not an aggregate of the 10,000 apo-Ala
protein. It does have a similarity to the amino acid composition
of the D proteins in that both contain no cysteine or cystine
residues. The large amount of arginine which this protein con-
tains makes its composition unique among human lipoproteins.
The VLDL protein described by Shore and Shore (25) also has
a high content of arginine and some similarity in amino acid con-
tent to the Peak II protein. In the absence of further data char-
acterizing their material it is impossible to decide if the proteins
are in fact identical.

In contrast to the D proteins and the apo-LDL protein of
VLDL, where the relative contents are functions of the size of the
lipoprotein (27), this Peak II protein comprises from 5 to
15% of total VLDL protein for all sized lipoproteins. It has
been observed to be lost from VLDL when these lipoproteins are
incubated with lecithin or lecithin-cholesterol mesophase disper-
sions and is then recovered in the LDL flotation region along
with the mesophase lipid. It is also found in substantial concen-
trations in the LDL fraction of humans with cholestatic liver
disease who have significant amounts of LDL lecithin and
cholesterol in mesophase form (28). The role which this protein
plays in the metabolism of the VLDL lipid is currently under
study.

FIG. 6. Sodium dodecyl sulfate polyacrylamide gel electro-
phoresis of the apoproteins from the major lipoprotein classes.
Solvent was 0.05 M phosphate buffer, pH 7.0, containing 0.1%
sodium dodecyl sulfate. The numbering of VLDL proteins is
the same as in Fig. 2.

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