Phospholipid transfer protein (PLTP) purified from human plasma was found to enhance the transfer of cholesterol from single bilayer vesicles containing phosphatidylcholine and cholesterol to high density lipoprotein-3. The rate of cholesterol transfer was greatly influenced by the cholesterol content of the donor vesicles. The maximal rate was observed with the vesicles containing 20–25 mol % cholesterol. This was in contrast to a progressive decline in the rate of phosphatidylcholine transfer with an increase in the cholesterol content. To determine the binding of cholesterol and phosphatidylcholine to PLTP, the mixtures of PLTP and the vesicles containing 3H-labeled phosphatidylcholine and 14C-labeled cholesterol were incubated and subjected to sucrose density gradient centrifugation. Determination of the label profiles showed that cholesterol as well as phosphatidylcholine were transferred from the vesicles to PLTP. The reversible nature of the binding was shown by the transfer of labeled cholesterol and phosphatidylcholine bound to PLTP to the acceptor vesicles or low density lipoprotein. Isothermal equilibrium binding of PLTP for cholesterol and phosphatidylcholine showed that PLTP possessed a considerably higher affinity and binding capacity for phosphatidylcholine than for cholesterol. The phosphatidylcholine binding affinity and capacity were greater when PLTP was incubated with phosphatidylcholine vesicles without cholesterol. A possible importance of PLTP-mediated cholesterol transfer in the circulation was described.

In human plasma, lipids can be exchanged and transferred between different lipoprotein classes by spontaneous, temperature-dependent transfer or by protein-mediated transfer (1). Unesterified cholesterol transfer and exchange occur by a spontaneous process through collision and aqueous diffusion (2, 3). In the cell, possible mechanisms of cholesterol movement also include aqueous diffusion, vesicle-mediated transport, and soluble carrier-mediated transport (4). Protein-mediated exchange and transfer of free cholesterol in the plasma have not been fully investigated. There are two types of lipid transfer proteins in plasma (1). Cholesteryl ester transfer protein promotes the transfer of neutral lipids such as cholesteryl esters and triglycerides, as well as phospholipids (PLs),1 among plasma lipoproteins (5–8). Phospholipid transfer protein (PLTP) facilitates the transfer and exchange of PL but not neutral lipids (9–12). PLTP was also shown to be responsible for the conversion of HDL₃ to HDL₂ size particles (12, 13).

Lipolysis of triglyceride-rich particles such as chylomicrons and very low density lipoproteins is known to deplete the triglyceride in the lipid core and to produce an excess surface coat of PL, free cholesterol, and apolipoproteins (14–16). Tall et al. (17) showed that PLTP enhanced the transfer and exchange of PL between very low density lipoproteins and HDL during lipolysis. They also showed that phosphatidylcholine (PC) vesicles may serve as a model of the redundant surface coat of lipolyzed particles to study the PL transfer to HDL (18). In this communication, we show that PLTP promoted cholesterol transfer in addition to PL transfer. The magnitude of cholesterol transfer from PC-cholesterol vesicles to HDL₃ by PLTP was much greater than that from LDL to HDL₃. PLTP was shown to possess the binding sites that can accommodate large numbers of both PC and cholesterol.

EXPERIMENTAL PROCEDURES

Materials—Human plasma samples were obtained from the Champaign County Blood Bank. LDL (d 1.019–1.045 g/ml) and HDL₃ (d 1.125–1.21 g/ml) were isolated from fresh, nonturbid plasma by the sequential ultracentrifugation method as described previously (19, 20). Cholesterol (>99%) and crystallized lyophilized bovine serum albumin were obtained from Sigma. 1-Palmitoyl-2-oleoylphosphatidylcholine was obtained from Avanti Polar Lipids Inc. (9,10-3H)Dipalmitoyl phosphatidylcholine (DPPC) (specific activity, 37.5 Ci/mmol), 7-3H) cholesterol (specific activity, 22.0 Ci/mmol), and [4,14C]cholesterol (specific activity, 54.0 mCi/mmol) were obtained from Du Pont NEN. [3H]Acetyl choline (specific activity, 500 mCi/mmol) was purchased from Amersham Corp. All other chemicals were of reagent grade quality and used without further purification. Glass-distilled water was used to prepare all aqueous solutions.

Buffer—Experiments were performed primarily in 39 mM phosphate buffer containing 60 mM NaCl and 0.025% EDTA, ionic strength 0.16, pH 7.4, unless otherwise specified.

Purification of PLTP—PLTP was purified from human plasma according to the method described previously (12). The final purification of the transfer protein was 38,000-fold over the starting plasma. The purified PLTP was stored at 4 °C in 0.4 mM phosphate buffer, pH 7.4, containing 2 mM NaN₃ and 0.025% EDTA.

Determination of PLTP-mediated Cholesterol and PC Transfer—Single bilayer vesicles containing PC and varying amounts of cholesterol were prepared essentially according to the method of Batzri and Korn (21) as described previously (12, 20). PC and cholesterol transfer assays were carried out using labeled donor vesicles containing 90 nmol of egg PC and varying amounts (0–37 nmol) of cholesterol/100 μl and HDL₃ (180 μg as protein) as the acceptor in phosphate buffer containing 60

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1 The abbreviation used are: PL, phospholipid; LDL, low density lipoprotein; HDL, high density lipoprotein; PC, phosphatidylcholine; PLTP, phospholipid transfer protein; DPPC, dipalmitoyl phosphatidylcholine.

Phospholipid Transfer Protein Mediates Transfer of not Only Phosphatidylcholine but Also Cholesterol from Phosphatidylcholines-Cholesterol Vesicles to High Density Lipoproteins*

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the final volume of 280 μl in phosphate buffer containing NaCl. Lipid transfer between the donors and acceptors was terminated by selectively precipitating donor particles with dextran sulfate and MgCl₂ (12). In both assay systems, radioactivities in HDL₃ (supernatant) were determined as described previously (12). Net mass transfer of cholesterol was determined by quantitating amounts of cholesterol present in the HDL₃ supernatant solutions by the method of Gamble et al. (23).

Labeling of HDL₃—HDL₃ was labeled with [³H]acetic anhydride (specific activity, 400 Ci/mmol) at the acetic anhydride/lysine molar ratio of 0.1 as described previously (20, 24).

Density Gradient Centrifugation—A discontinuous sucrose density gradient was formed in 5.5-ml quick seal tubes (Beckman Instruments) by layering 2.4 ml of phosphate buffer, 1.2 and 0.9 ml of phosphate gradient was formed in 5.5-ml quick seal tubes (Beckman Instruments) by layering 2.4 ml of phosphate buffer, 1.2 and 0.9 ml of phosphate buffer to one third of the tube. A 2.4 ml sample of the gradient was placed on top of the sucrose density gradient fractions were collected, and the radioactivity of each fraction was determined. The radioactivity peak coinciding with the HDL₃ protein radioactivity peak was used to calculate the area of labeled cholesterol transferred to HDL₃ after correcting for a trace amount of nonprecipitated vesicle radioactivity in the supernatant that was made by subtracting the zero time values from the observed transfer values in the presence and absence of PLTP. The extent of cholesterol transfer was determined by using the equation given under “Experimental Procedures.” The results presented are representative of three similar experiments carried out with different preparations of purified PLTP. Inset, the amounts of radioactive cholesterol (curve a) and cholesterol mass (curve b) transferred by varying concentrations of PLTP during incubation at 37°C for 10 min were plotted against PLTP concentrations. Curve a was obtained by subtracting nonspecific transfer values in the absence of PLTP from the transfer values obtained in the presence of PLTP. Curve b shows a result representative of three similar experiments.

RESULTS

Cholesterol Transfer from PC-Cholesterol Vesicles to HDL₃—The time dependence of PLTP-mediated cholesterol transfer was studied by using PC-[³H]cholesterol vesicles as the cholesterol donor and HDL₃ as the acceptor in the presence of varying amounts of purified PLTP. The incubation was carried out at 37°C for different periods. The rate of cholesterol transfer increased progressively during the incubation period of 10 min at 37°C (Fig. 1, lines 2–4) in the presence of all concentrations of PLTP. An increase in nonspecific transfer during incubation was also given (Fig. 1, line 1). It is to be noted that the highest concentration of PLTP used in this assay, 560 ng/280 μl (2 μg/ml), roughly corresponds to the PLTP concentration in human plasma (12). The extent of transfer of labeled cholesterol by 560 ng of PLTP (Fig. 1, line 4) was approximately five times that of nonspecific cholesterol transfer (Fig. 1, line 1). Fig. 1, inset, shows magnitudes of PLTP-mediated transfer of labeled cholesterol (curve a) and cholesterol mass (curve b) at a 10-min incubation as a function of PLTP concentration. The radioactive cholesterol transfer as well as net mass transfer of cholesterol increased linearly up to the PLTP concentration of 200 ng. A further increase in the PLTP concentration resulted in deviation from linearity. The net mass transfer of cholesterol amounted to roughly 80% of the transfer of labeled cholesterol.

The transfer of cholesterol from PC-cholesterol vesicles to HDL₃ was also shown by density gradient centrifugation of the PC-cholesterol vesicle/HDL₃ mixtures incubated in the presence and absence of PLTP for 10 min at 37°C. The extent of nonspecific transfer occurring in the absence of PLTP was represented by the area given by the cholesterol radioactivity curve overlapping with the HDL₃ protein radioactivity peak (Fig. 2A). In the presence of 560 ng of PLTP, the extent of cholesterol transfer from the vesicles to HDL₃ was substantially enhanced, as shown by the increase in the area of the cholesterol radioactivity peak coinciding with the HDL₃ protein radioactivity peak (Fig. 2B). The extent of cholesterol transfer from PC-cholesterol vesicles to HDL₃ was approximated from the total peak areas of labeled cholesterol and the relative peak area of labeled cholesterol transferred to HDL₃ after correcting
for the area of nonspecific transfer. Approximately 14% of total cholesterol, or 3 nmol of cholesterol, was transferred. A net mass transfer of approximately 2.4 nmol of cholesterol could be expected on the basis of the relationship between the transfer of labeled cholesterol and net mass transfer (Fig. 1, inset). We observed that PLTP does not facilitate cholesterol transfer at 4°C or in an ice bath. However, nonspecific transfer of cholesterol could occur at these temperatures although at considerably reduced rates.

Effect of Cholesterol Content of PC-Cholesterol Vesicles on the PLTP-mediated PC and Cholesterol Transfer—Although PC transfer from the vesicles to HDL₃ by PLTP was inhibited by an increase in the cholesterol content of the donor vesicles (Fig. 3A; Refs. 12, 29, and 30), cholesterol transfer from the vesicles to HDL₃ was increased by increasing the cholesterol content of the vesicles up to approximately 20 mol %. A further increase in the cholesterol content reduced the extent of cholesterol transfer (Fig. 3B). The relationship between mol % of cholesterol transferred to HDL₃ and mol % of cholesterol in donor PC-cholesterol vesicles showed that the increase in the cholesterol content of vesicles progressively increased transfer of cholesterol given in mol % with respect to the sum of cholesterol and PC transferred to HDL₃ (Fig. 4A). The ratio (C/(C + PC))transf/(C/(C + PC))v in Fig. 4B, ordinate, represents the ratio of mol % of cholesterol transferred to HDL₃ to mol % cholesterol in the vesicles. This ratio was consistently reduced with an increase in the mol % of cholesterol in the donor PC-cholesterol vesicles (Fig. 4B).

Binding of Cholesterol and PC to PLTP—We demonstrated an actual uptake of cholesterol and PC molecules by PLTP from donor vesicles and subsequent donation of these bound lipids to acceptor particles (Fig. 5). Fig. 5A shows the distribution of labeled PC and cholesterol in control vesicles incubated in the absence of PLTP. Fig. 5B shows the profile of control PLTP as determined by the PC transfer activity. The incubation of vesicles with PLTP resulted in the transfer of a substantial amount of cholesterol and PC radioactivities to the PLTP (Fig. 5C). The PLTP fractions with bound lipids were collected and pooled. When one-third of the pooled PLTP fraction resulted in the transfer of most of the PC and cholesterol radioactivities remained associated with the PLTP peak (Fig. 5D). However, the addition of unlabeled PC vesicles to the second one-third of the pooled fraction resulted in the transfer of about 40% each of labeled cholesterol and PC to LDL, whereas approximately 60% of PC and cholesterol radioactivity remained associated with PLTP (Fig. 5E). It appeared that PLTP-mediated cholesterol and PC

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**Fig. 2.** PLTP-mediated cholesterol transfer from PC-cholesterol vesicles to HDL₃ determined by density gradient centrifugation. The vesicle/HDL₃ mixtures were incubated for 10 min at 37 °C in the absence (A) and the presence of 560 ng of PLTP (B) in a final volume of 200 µl of phosphate buffer containing NaCl. After incubation, the samples in a sucrose density gradient were centrifuged in a vertical rotor. The distribution patterns of labeled cholesterol (○) from PC/[3H]cholesterol vesicles and 3H-labeled HDL₃ protein (●) were determined from their radioactivities in collected fractions but obtained from separate experiments in which the sample mixtures contained only one radioactive component. The labeled protein (●) distribution was obtained to indicate the position of HDL₃ in sucrose density gradient centrifugation profiles. The total recoveries of cholesterol and HDL₃ protein radioactivities in the assay mixtures were within the range of 65–75%. The compositions of vesicle/HDL₃ mixtures used for density gradient centrifugation were the same as those of the assay mixtures for cholesterol transfer reaction. Results obtained from representative experiments are shown.

**Fig. 3.** Effect of vesicle cholesterol content on the PLTP-mediated PC and cholesterol transfer from vesicles to HDL₃. Vesicle preparations containing 90 nmol of [3H]-labeled PC and 0–29 mol % of [14C]-labeled cholesterol were used as the donors. The donor vesicles were incubated with acceptor HDL₃ (180 µg as protein) in the presence and absence of 200 ng of PLTP for 10 min at 37 °C. The transfer assay was carried out in a similar manner as given for Fig. 1. The data given are representative of three similar experiments carried out by using different sets of vesicle preparations.
transfer involved the uptake of lipid molecules by PLTP from donor particles and subsequent transfer of these lipids to the acceptor particles.

For the binding study, we assumed that cholesterol partitioning between the outer and inner monolayer is 70:30 and that only cholesterol in the outer monolayer is subjected to exchange (31, 32). The Scatchard plots for cholesterol and PC binding to PLTP were obtained by determining the amounts of PC and cholesterol bound to PLTP obtained by sucrose density gradient centrifugation of the incubation mixtures containing PLTP and vesicles. The plot obtained for the PLTP incubated with the vesicles without cholesterol (Fig. 6A) gave an apparent $K_d$ of 53 nM for PC and an apparent binding capacity of 70, in mol PC/mol PLTP. For the PLTP incubated with the vesicles containing cholesterol, the Scatchard plots were obtained for both PC binding (Fig. 6B) and cholesterol binding (Fig. 6C). The binding parameters obtained were an apparent $K_d$ of 131 nM for PC and 373 nM for cholesterol and an apparent binding capacity, in mol lipid/mol PLTP, of 43 for PC and 13 for cholesterol.

**Transfer of Cholesterol from LDL to HDL$_3$**—Although both PLTP-mediated cholesterol transfer (Fig. 7A) and PC transfer (Fig. 7B) from LDL to HDL$_3$ increased with increases in the PLTP concentration, the extents of cholesterol and PC transfer from LDL to HDL$_3$ were rather limited. After a 30-min incubation in the presence of 600 ng of PLTP in the assay mixtures, about 3.2 nmol of cholesterol was transferred by PLTP, compared with 10 nmol of nonspecific cholesterol transfer. The lower cholesterol transfer rate was not due to the method used for labeling LDL. When LDL was labeled with the filter paper method (33), the degrees of nonspecific and PLTP-mediated cholesterol transfer were similar to those obtained by the ethanol method used (11, 12). The magnitude of PLTP-mediated PC transfer from LDL to HDL$_3$ (Fig. 7B) was greater than the cholesterol transfer (Fig. 7A).

**DISCUSSION**

In the present study, we demonstrated that PLTP mediated a substantial transfer of not only PC, but also cholesterol from PC-cholesterol vesicles to HDL. The rate of cholesterol transfer from the vesicles to HDL was profoundly influenced by the cholesterol content of the donor vesicles (Fig. 3B). A progressive increase in the rate of cholesterol transfer, with an increase in the vesicle cholesterol content to 20 mol %, might have re-
flected increased availability of cholesterol in the vesicles. The decrease in the rate of cholesterol transfer at higher cholesterol contents might have been caused by decreased fluidity of the lipid interface, as was attributed to a decline in PC transfer with an increase in the cholesterol content of vesicles (29, 30).

Incorporation of cholesterol into egg PC vesicles is well known to transform the disordered bilayer into an ordered structure (34, 35). We are currently investigating the binding of PLTP to the vesicles, as influenced by cholesterol, to clarify the mechanism of the cholesterol effect on the PLTP-mediated PC and cholesterol transfer.

We showed that PLTP indeed possessed a large number of binding sites for PC and cholesterol. On the basis of apparent binding capacity values of 43 mol of PC and 13 mol of cholesterol/mol of PLTP obtained from Scatchard plots, the particle mass of PLTP with maximum numbers of bound PC and cholesterol was found to be 117 kDa. PC and cholesterol contributed 29 and 4.3%, respectively, of the total mass on a weight basis. We assumed that the hydrated density of PLTP-lipid complex having maximal number of bound PC and cholesterol was 1.18 g/ml. By using a PL molecular volume of 1270 Å³, a free cholesterol molecular volume of 600 Å³ (36), and a presumed partial specific volume of PLTP-glycoprotein of 0.70 g/ml, the amount of hydrated water became 0.22 g/g PLTP-lipid complex. Then, the PLTP used for lipid binding, having a hydrated density of 1.22 g/ml, would contain 23% PC and 3% cholesterol on a weight basis, if they were the only lipids present at the same PL:cholesterol ratio. The extent of hydration was considered proportional to the total mass of the PLTP-lipid complex. Our preliminary study indicated that purified PLTP indeed contained phospholipids and cholesterol. During the incubation for the binding assay at 37°C for 1 h, radioactive phosphatidylcholine and cholesterol in vesicles could have equilibrated with those originally associated with the purified PLTP. The decrease in the hydrated density of PLTP fractions after incubation with vesicles was apparently due to an increase in the lipids bound to multiple binding sites of PLTP.

PLTP may shuttle PC and cholesterol molecules between donor and acceptor particles. We demonstrated not only the transfer of labeled PC and cholesterol from vesicles to PLTP, but also the transfer of the labeled lipids from the isolated PLTP fraction to unlabeled vesicles. Transfer of the bound lipids to LDL was also observed. Although the carrier mechanism may largely be responsible for the transfer, the involvement of the ternary complex of PLTP with acceptor and donor particles for the transfer cannot be eliminated. We previously observed that the rate of PLTP-mediated PC transfer from vesicles to HDL₃ was considerably higher than that of PLTP-mediated PC transfer from LDL to HDL (12). Vesicles appar-
Phospholipid Transfer Protein

tently served as efficient PC donors for PLTP. This was not the case for cholesteryl ester transfer protein, which transfers phospholipids in addition to neutral lipids among plasma lipoproteins. The rate of PC transfer by cholesteryl ester transfer protein from vesicles to HDL₃ was low and was comparable with the rate of PC transfer from LDL to HDL₄ (12). Furthermore, cholesteryl transfer mediated by PLTP in the vesicle/HDL assay system was about four times greater than that of the LDL/HDL system. The efficient transfer of both labeled PC and cholesterol by PLTP from vesicles to HDL₃ apparently reflects the presence of multiple PC and cholesteryl binding sites on PLTP. The binding of PLTP to vesicles may lead to the transfer of several molecules of labeled PC and cholesterol from vesicles to bound PLTP during a single successful encounter. The number of these lipids transferred from LDL to PLTP on the interaction between LDL and PLTP may be rather limited, due possibly to the surface properties of LDL. Cholesterol and phospholipids may be tightly packed in the LDL surface, with some phospholipids immobilized by association with apoB-100 (37).

It is possible that the transfer of labeled PC and cholesterol from the vesicles to PLTP is not likely to involve the direct transfer of intact vesicle segments. It may rather involve the transfer of vesicle PC and cholesteryl to the multiple binding sites of PLTP governed by the affinities of PLTP for PC and cholesteryl and the binding capacities for these components. This may be followed by the transfer of these bound, labeled lipids to acceptor HDL₃. We observed that the increase in the cholesterol content of vesicles progressively decreased the ratio of mol % cholesterol transferred to HDL to mol % cholesterol in the vesicles, which is given by (C/C₈ + PC/PC₈)trans/C/C₈ (Fig. 4B). This ratio was approximately 0.8 for donor vesicles containing 7.7 mol % cholesterol, and about 0.6 when the vesicles contained 29 mol % cholesterol. Such a decline may reflect, in part, binding of cholesterol and PC to PLTP, as influenced by the cholesteryl content of vesicles.

The present report described our observations on the PLTP-mediated transfer of PC and cholesterol as well as the binding of these lipids to PLTP. PLTP-mediated net mass transfer of phospholipids from very low density lipoproteins to HDL was previously reported (38). We showed PLTP-mediated stimulation of a net mass transfer of cholesterol from vesicles to HDL. PLTP may play an important role in the transfer of cholesteryl and phospholipids from the redundant surface coat of lipolyzed, triglyceride-rich lipoproteins to HDL₃ and possibly to the plasma membrane of the cells, such as hepatocytes, which are actively using the excess phospholipids and cholesteryl. We recently observed that PLTP substantially enhanced the transfer of cholesteryl from cultured fibroblasts to acceptor lipoprotein particles. The nature of PLTP involvement in the first step of reverse cholesterol transport will be described in our subsequent article.

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