Insect Lipid Transfer Particle Catalyzes Bidirectional Vectorial Transfer of Diacylglycerol from Lipophorin to Human Low Density Lipoprotein*

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Insect plasma lipid transfer particle (LTP) catalyzes vectorial net transfer of diacylglycerol (DAG) from Manduca sexta larval high density lipophorin (HDLp-L) to human low density lipoprotein (LDL) producing an LDL of lower density and lipophorin subspecies of higher density. At equilibrium, a stable DAG-depleted very high density lipophorin species (density = 1.25 g/ml) is formed. Electrophoretic analysis of the substrate and product lipoproteins showed that apoprotein exchange or transfer between human LDL and lipophorin did not occur during the lipid transfer reaction. Facilitated net transfer of cholesteryl ester, free cholesterol, and phospholipid occurred to a much lower extent than DAG net transfer, indicating that under these conditions, LDL serves as a sink for lipophorin-associated DAG. This reaction, therefore, provides a method whereby the mass of lipid associated with human LDL can be modified in vitro without alteration of its apo-protein component. The DAG content of LDL increased in a linear manner with respect to LTP concentration and time during the initial phase of the reaction, demonstrating the utility of this system as a quantitative assay method for LTP-mediated net DAG transfer. When \(^{[3}H\)DAG-labeled LDL was prepared and employed in transfer experiments with unlabelled lipophorin, labeled DAG was recovered in the HDLp-L fraction. The amount of labeled DAG recovered in the HDLp-L fraction was dependent on the ratio of LDL to HDLp-L in the reaction. Thus, in this system, LTP-mediated DAG redistribution is bidirectional, suggesting that the final equilibrium distribution of lipid may be dictated by the properties of potential donor/acceptor lipoproteins rather than by inherent particle substrate specificity of LTP.

Lipophorin is the plasma lipoprotein found in insect hemolymph which serves to transport neutral glycerolipid (as diacylglycerol (DAG)\(^1\)) from sites of ingestion or synthesis to sites of utilization (1). Although its structural properties are not understood, it has been postulated that a basic matrix structure comprised of the two integral lipophorin apoproteins, apolipophorin I (Mr ~ 240,000) and apolipophorin II (Mr 80,000), plus some lipid, has the capacity to accept additional lipid to form larger, less dense lipophorin particles. Likewise, lipid may be removed from lipid-rich lipophorin particles with retention of the basic matrix structure (2). Thus, an important feature of DAG transport by lipophorin is that DAG turnover occurs at a faster rate than protein turnover (3), with the result that lipophorin functions as a reusable lipid shuttle. It is thought that the unique ability of lipophorin to exist in several distinct forms that differ in size, density, and DAG content (4) is important in the proposed shuttle mechanism.

The way in which the DAG content of preexisting lipophorin particles can be modified is not understood, but it has been postulated that a lipid transfer particle (LTP) may be involved (2). LTP has been isolated and shown to catalyze net transfer of DAG between isolated lipophorins of different density and lipid composition in vitro, resulting in dramatic changes in lipophorin size and density. Net lipid transfer occurs from the lower density lipophorin to the higher density lipophorin in the presence of LTP, producing, at equilibrium, a single lipoprotein population intermediate in density between the starting lipoproteins (2, 5). LTP has also been shown to catalyze net transfer of phospholipid and triacylglycerol from a human apolipoprotein A-I-stabilized phospholipid/triolein microemulsion to human low density lipoprotein (LDL) (6). On the other hand, when a non-lipophorin very high density lipoprotein was employed as lipid acceptor in incubations with lipophorin and LTP, facilitated DAG exchange but not net transfer occurred (7). Thus, the nature of the LTP-catalyzed reaction seems to depend on the structural properties of potential donor/acceptor substrate particles as well as their inherent lipid binding capacities.

In an attempt to understand further the catalytic properties of LTP we have examined the ability of LTP to catalyze lipid transfer between lipophorin and human LDL. It is demonstrated that LTP catalyzes a bidirectional but vectorial transfer of lipophorin-associated DAG into human LDL without affecting its apoprotein component. The reaction produces a DAG-enriched LDL and a DAG-depleted lipophorin species as stable end products. The potential utility of LTP as a tool

\(^1\) The abbreviations used are: DAG, diacylglycerol; LTP, lipid transfer particle; HDLp-L, high density lipophorin-larval; LDL, low density lipoprotein; SDS, sodium dodecyl sulfate; CE, cholesteryl ester.

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to modify the lipid content and composition of LDL is discussed.

**MATERIALS AND METHODS**

Isolation of Lipophorin, LDL, and LTP—[3H]DAG high density lipophorin-larval (HDLp-L) was isolated from 4-day-old fifth instar larvae of the tobacco hornworm *Manduca sexta* obtained from a continuing laboratory colony reared as described elsewhere (8). [9,10-3H]Oleic acid (Amersham Corp., 10 Ci/mm) was administered, animals were bled, and collected hemolymph was subjected to density gradient ultracentrifugation as described previously (9, 10). LDL-HDLp-L was prepared as described elsewhere (2). LTP was isolated from hemolymph of 7-day-old fifth instar larvae that had entered the prepupal stage according to the procedure of Ryan et al. (5). LDL was isolated from freshly collected plasma between a density of 1.006 and 1.063 g/ml by sequential density gradient ultracentrifugation. [3H]Cholesteryl ester (CE) LDL (specific activity = 6.78 × 10^6 dpm/pmol CE) was prepared according to Nishikawa et al. (11). Lipoproteins and LTP were subsequently stored at 4 °C until use.

**Lipid Transfer Assays**—Given amounts of human LDL and insect ([3H]DAG-HDLp-L (specific activity = 75,000 dpm/mg DAG) (1:1 protein ratio in a typical experiment) were incubated in 0.05 M Tris, pH 7.4, for a specified time at 33 °C in the absence or presence of a given amount of LTP. Following incubation, the samples were transferred to ice, adjusted to a density of 1.23 g/ml with solid KBr (33% (w/v); 20 ml, final volume), transferred to a 39-ml Beckman Quick-Seal tube, and overlaid with 0.9% NaCl. The tubes were centrifuged at 50,000 rpm for 4 h at 4 °C in a VTI-50 rotor. Alternatively a VTI-65.2 (5.1-ml capacity) or a Ti-70.1 (39-ml capacity) rotor was employed. Following centrifugation, the tube contents were fractionated from the top. Radioactivity in each fraction was determined by liquid scintillation spectrometry, densities were determined by refractionometry, and protein was determined by the method of Smith et al. (12). Transfer of DAG from LDL to lipophorin was studied using [3H]DAG-LDL produced using the experimental system described above. Transfer of CE from LDL to HDLp-L was examined using [3H]CE-LDL plus cold LDL and HDLp-L as substrates. Incubations were conducted and lipoproteins reisolated as described above.

**Lipid Analysis**—Lipoprotein samples were extracted according to Bligh and Dyer (13) and the organic soluble phosphorous determined as described by Ames and Dubin (14). The DAG content of lipoprotein fractions was determined from radioactive profiles using the specific activity of DAG in the original lipophorin, as no DAG is present in the starting LDL. Free and esterified cholesterol as well as neutral glycerolipid were determined using enzyme-based assay kits purchased from Wako Pure Chemicals, Dallas, TX. Thin layer chromatography of lipids extracted from lipoprotein fractions was performed on precoated Silica Gel G plates developed in hexanediethyl etheracetic acid (70:30:1; v/v/v). Plates were scanned on a BioScan system 200 radiochromatogram scanner.

**Electrophoresis**—The apoprotein content of lipoprotein fractions was analyzed by sodium dodecyl sulfate (SDS)-polyacrylamide gel electrophoresis according to Laemmli (15). A 4-15% acrylamide gradient slab gel was employed along with a 2.5-cm 3% acrylamide stacking gel. Gels were run at a constant 30 mA for 3.5 h and stained with Coomassie Blue.

**RESULTS**

**LTP-catalyzed Changes in Lipoprotein Density Distribution**—Given the differences in lipid and protein composition between human LDL and insect HDLp-L (8, 16), quantitative separation can be achieved by a 4-h vertical rotor density gradient ultracentrifugation. When LDL or HDLp-L was incubated separately with LTP, there was no change in their respective relative densities. Furthermore, there was no change in lipoprotein density distribution when LDL and HDLp-L were incubated together in the absence of LTP (Fig. 1A), indicating that the lipoprotein substrates are stable particles and are not prone to breakdown or aggregation under the incubation and reisolation conditions employed. In the presence of catalytic amounts of LTP, however, changes in the lipoprotein density distribution were observed. After 15 min, an alteration in lipoprotein density distribution was apparent (Fig. 1D) for the lipophorin fraction, which floated to a slightly higher density than the original HDLp-L. After a 120-min incubation in the presence of LTP, LDL floated to a lower density position, but the HDLp-L exhibited a bimodal density distribution (Fig. 1C). Peak I corresponded to the density of the HDLp recovered after a 15-min incubation, but peak II was of higher density and was observed only in the 120-min incubation. Increased amounts of LTP (Fig. 1D), longer incubation times, or an increased LDL:HDLp-L protein ratio in the incubation resulted in recovery of an even greater proportion of the HDLp at a density corresponding to peak II. This lipoprotein density distribution was unaffected by longer incubation times or further increases in LTP amount, indicating that a thermodynamically stable end point had been reached.

**Characterization of the Protein Components**—The apoprotein distribution of the starting lipoproteins as well as the product species obtained following incubation with LTP were examined by SDS-polyacrylamide gel electrophoresis (Fig. 2). The results show that the lower density fraction obtained following a 2-h incubation of LDL and HDLp-L contained no lipophorin apoproteins whereas the peak I and peak II fractions contained only the two characteristic lipophorin apoproteins. Likewise, all apoB was recovered in the fractions corresponding to a density of LDL. The presence of minor bands in the LDL lanes was attributed to proteolytic degradation of apoB. No evidence for the presence of other human serum apolipoproteins in the LDL preparations employed was obtained. When [3H]HDLp-L was used in incubations with LDL
Facilitated Bidirectional Vectorial Lipid Transfer

ApoB

ApoLp-II

ApoLp-I

FIG. 2. SDS-polyacrylamide gel electrophoresis of lipoproteins. LDL and HDLp-L (2 mg of protein each) were incubated in the presence and absence of LTP (5 μg) for 120 min at 33 °C. Following incubation, the samples were subjected to density gradient ultracentrifugation and the lipoproteins isolated. After dialysis, the lipoprotein fractions were electrophoresed on a 4–15% acrylamide gradient slab gel in the presence of SDS. (A) Lane 1, LDL control; lane 2, LDL following incubation with LTP; lane 3, HDLp-L control; lane 4, peak I lipophorin; and lane 5, peak II lipophorin.

and LTP, all of the aqueous soluble radioactive material was recovered at a density greater than the original HDLp-L (data not shown), lending further support for the conclusion that lipophorin apoproteins do not exchange or transfer to LDL during the reaction.

Net Transfer of DAG from Lipophorin to LDL—Experiments using [3H]DAG-HDLp-L were performed to investigate the effect of LTP on the density distribution of DAG. Following incubation of [3H]DAG-HDLp-L with LDL in the presence and absence of LTP, the density distribution of radioactivity was determined (Fig. 3). In control experiments lacking LTP, nearly all of the radioactive material was recovered in association with HDLp-L. In incubations containing catalytic amounts of LTP, however, there was a major change in the density distribution of radiolabel. At 15 min (panel B), approximately 50% of the labeled DAG was recovered at a density corresponding to LDL while the remainder was associated with the lipophorin fraction of density equal to peak I lipophorin. At 120 min, over 80% of the labeled DAG was recovered in the LDL fraction, with the remainder recovered at a density corresponding to peak I lipophorin. Peak II lipophorin, which at 120 min contained an approximately equal amount of protein as peak I lipophorin, possessed a lower amount of DAG compared with that present in peak I lipophorin. When an increased amount of LTP was included in the incubation, the small amount of radioactivity recovered in association with lipophorin was recovered at a density corresponding to peak II lipophorin. These results indicate that LTP catalyzed substantial net transfer of DAG from HDLp-L to LDL, which is consistent with the observed shifts in lipoprotein density.

In order to confirm that the [3H]DAG remained unchanged during the transfer reaction, the lipids present in LDL, isolated following incubation of LDL, [3H]DAG-HDLp-L, and LTP were extracted and analyzed by thin layer chromatography and radiochromatogram scanning. The results demonstrated that the only radioactive material present corresponded to DAG. LDL has a large pool of CE which conceivably could transfer to lipophorin. We tested this by using [3H] CE-LDL in incubations with unlabeled HDLp-L. The results show (Fig. 4) that under these conditions, LTP induced transfer of less than 3% of the LDL-associated CE. Lipid analysis

FIG. 3. The effect of LTP on the distribution of DAG between lipophorin and LDL. [3H]DAG-HDLp-L (300,000 dpm/mg of protein) and LDL (2 mg of each on the basis of protein) were incubated in the presence and absence of LTP. Following incubation, the samples were subjected to density gradient ultracentrifugation, fractionated (1.1 ml), and the radioactivity (50-μl aliquot) and density in each fraction determined. Panel A, control incubation (120 min, no LTP); panel B, 15-min incubation with 5 μg of LTP; panel C, 120-min incubation with 5 μg of LTP; panel D, 120-min incubation in the presence of 20 μg of LTP.

FIG. 4. The effect of LTP on the distribution of [3H]CE. [3H] CE-LDL (32 μg of protein; specific activity = 6.78 × 10^6 dpm/μmol CE) plus 988 μg of cold LDL protein were incubated with HDLp-L (1 mg of protein) for 60 min at 33 °C in the presence of 2.5 μg of LTP. Following incubation, the samples were subjected to density gradient ultracentrifugation, fractionated (1.1 ml), and the radioactivity (25-μl aliquot) and density of each fraction determined.
performed on isolated peak II lipophorin revealed that it possesses only small amounts of lipid (~15% of the particle mass) comprised mostly of PL (80%), DAG (10%), and lesser amounts of free and esterified cholesterol. These results are consistent with the observed flotation behavior of peak II lipophorin (equilibrium density = 1.26 g/ml) and when compared with the density (1.19 g/ml) and composition (98% lipid, 62% protein) of HDLP-L (8), indicates that a significant loss of lipophorin-associated DAG occurred. Indeed, the percent weight composition of DAG was changed from 18% for HDLP-L to <2% for peak II lipophorin.

The amount of radiolabeled DAG transferred from [3H]DAG-HDLp-L to LDL increased linearly as a function of LTP concentration and time during the initial phase of the reaction (~<50% transfer) when a 1:2 HDLP-L:LDL protein ratio is employed in the incubation. During the initial phase of this reaction, the LTP-catalyzed net transfer of DAG from HDLP-L to LDL occurs at a rate of 2.4 µg of DAG/min/µg of LTP protein under the given experimental conditions. Thus, this reaction provides a useful quantitative lipid transfer assay method analogous to an assay system reported earlier (10) which employed insect low density lipophorin rather than human LDL. In contrast to the previous method, the present assay provides a convenient estimate of facilitated net DAG transfer rather than exchange.

**Vectorial Transfer of DAG Is Bidirectional**—An important question regarding the mechanism of LTP-catalyzed transfer of DAG from HDLP-L to LDL concerns whether the reaction is reversible. It is possible that LTP does not interact with LDL as a lipid donor. Thus, upon transfer of DAG into LDL the DAG may no longer be accessible to LTP. Alternatively, the final equilibrium distribution of DAG between LDL and HDLP-L could be dictated by the respective hydrophobic environments provided by potential donor/acceptor species. This question was examined by preparing [3H]DAG-LDL and incubating it with unlabelled HDLP-L and LTP. The results (Fig. 5) demonstrate that [3H]DAG associated with LDL is transferred to HDLP-L in the presence of LTP. Furthermore, the amount of radiolabeled DAG recovered in association with HDLP-L was dependent on the amount of acceptor HDLP-L in the incubation. Thus, LTP-catalyzed DAG transfer is bidirectional, and the observed redistribution of DAG occurs through an exchange reaction with a final equilibrium distribution that favors association with LDL.

**DISCUSSION**

Using a novel lipid transfer particle from the plasma of *M. sexta*, we have described a reaction system capable of effecting dramatic changes in the density distribution of insect lipophorin and human LDL. Lipophorin serves as lipid donor in this reaction and is ultimately transformed into a particle possessing greatly reduced amounts of DAG. The nature of the reaction between LDL and lipophorin, in which facilitated vectorial net DAG transfer occurs from HDLP-L to LDL, contrasts sharply with results obtained with lipophorins as substrate. The directional flux of LTP-catalyzed net lipid transport was observed when two lipophorin species of different density are employed as donor and acceptor from lipid rich-donor lipophorin to relatively lipid-poor acceptor lipophorin, producing a lipoprotein product intermediate in density between the starting lipoproteins (2, 5). Since these substrates possess the same basic matrix structure, neither substrate particle provides a preferred hydrophobic environment for DAG, and ultimately homogenization of particle size and density occurs. In the case of human LDL and insect HDLP-L, however, HDLP-L serves as lipid donor, producing a DAG-enriched human LDL and DAG-depleted lipophorin particles.

In other studies, insect LTP has been shown to catalyze net transfer of phospholipid and triacylglycerol from apoprotein-stabilized microemulsions to LDL (6). Comparison of these results suggests that the physical properties and hydrophobic environment provided by potential donor or acceptor particles are determinants of the directional flux of facilitated lipid transfer. Other important factors may include the lipid specificity of the transfer catalyst, lipid substrate availability, as well as the lipid-binding capacity of potential donor/acceptor species.

An important concept of the shuttle hypothesis of lipid transport by lipophorin (1) is that lipids may be removed from the particle without destruction of its basic matrix structure. The present transfer reaction, in which LDL serves as a sink for HDLP-L-derived DAG, may be similar to physiological processes in which LTP has been shown to play a role in transfer of lipophorin-associated DAG between cells and lipoprotein (17). Interestingly, the density and lipid composition of peak II lipophorin obtained in this in vitro lipid transfer system are similar in density and lipid content to the lipophorin species isolated and characterized from *M. sexta* eggs (18) or the hemolymph of larvae reared on a fat-free diet (19). Thus, it may be that the lipophorin product obtained in this reaction resembles physiologically relevant lipophorin species which represents a stable arrangement of lipid and apoprotein that conceivably constitutes the postulated basic matrix structure inherent in all lipophorin forms.

Insect LTP appears to be distinct from other plasma lipid transfer catalysts described to date. First, it exists as a very high density lipoprotein of high molecular weight (M, > 670,000). LTP has three apoprotein components and 14% lipid in the native particle (10). Although it has been shown that the lipid component of LTP is in equilibrium with that of potential donor or acceptor lipoproteins (10), the precise role of individual LTP apoproteins is not clear. A second distinguishing feature of LTP is its propensity to catalyze unidirectional net lipid transfer versus homo or hetero exchange of lipid. By contrast, human cholesteryl ester transfer protein catalyzes reciprocal random exchange of CE and triacylglycerol (20, 21). LTP-catalyzed net transfer establishes an altered final equilibrium lipid distribution that results in changes in the total mass of lipid associated with lipoprotein substrates rather than redistribution of lipid classes via a simple exchange process.

![Fig. 6. Bidirectional exchange of DAG between LDL and lipophorin. [3H]DAG-LDL (1 mg of protein; 200,000 dpm/mg of protein), prepared by incubation of LDL with [3H]DAG-HDLP-L and reisolated by density gradient ultracentrifugation, was incubated for 15 min with a given amount of unlabeled HDLP-L protein in the presence or absence of LTP (2.5 µg of protein/mg of LDL protein). Following incubation the samples were subjected to density gradient ultracentrifugation in a Beckman Ti-70.1 rotor, fractionated (1.1 ml), and the radioactivity (50 µl aliquot) and density in each fraction determined. Open squares, 5 mg of HDLP-L, no LTP; closed diamonds, 1 mg of HDLP-L; closed squares, 2 mg of HDLP-L.](http://www.jbc.org/Downloaded from http://www.wiley.com/go/jbc)
The assay system described can provide a quantitative measure of LTP activity. We have previously employed M. sexta low density lipophorin and HDLp-L as substrates in transfer assays (10). Although this method is quite sensitive, it is restricted to measurement of DAG exchange in the initial phase of the reaction. At longer times or at higher LTP concentrations, particle size and density homogenization complicate reisolation of the original donor/acceptor populations. The reaction described here is advantageous in that the starting LDL does not contain DAG. Therefore, DAG recovered in LDL is due to transfer. Second, homogenization of donor/acceptor species does not occur since the directional flux of net lipid transfer is from HDLp-L to LDL. The observed initial rate of facilitated lipid transfer (<50% DAG transfer from lipophorin to LDL) in this system was 2.4 µg/min/µg of LTP. This high rate of net lipid mass transfer suggests that LTP is a highly efficient catalyst and lends support to the proposal that it is involved in net transfer of DAG during energy-demanding DAG-fueled, flight activity (17). After approximately 50% transfer of labeled DAG to LDL, linearity of the reaction progress with respect to LTP concentration is lost. The ensuing slower phase of the reaction appears to involve conversion of peak I to peak II lipophorin, which apparently is more complex kinetically.

Although the present results illustrate the ability of LDL to serve as an acceptor of HDLp-L-associated DAG, they do not reveal its capacity to bind exogenous lipid. In the incubation conditions employed in this study (1:1 LDL:HDLP-L protein ratio), the lipid pool associated with HDLp-L is considerably smaller than that of LDL. By accepting the lipid available from HDLp-L under these conditions, HDLp-L is depleted of available lipid prior to saturation of LDL. Further studies on facilitated lipid transfer between HDLp-L and LDL have revealed that when the HDLp-L lipid pool is increased relative to LDL, saturation of LDL particles with exogenous lipid occurs. Subsequently, LTP promotes equilibration of the HDLp-L and LDL lipid pools in a time-dependent manner. Thus, it appears that although LTP is capable of transferring a variety of lipids associated with lipophorin (22), DAG is a preferred substrate, compared with CF, in the present reaction system. Whether this apparent specificity is a reflection of the structural properties of potential donor/acceptor particles or an inherent property of LTP remains to be determined.

The observed changes in lipid distribution catalyzed by LTP as well as the observed bidirectional nature of DAG transfer could conceivably result from two possible mechanisms: a carrier-mediated mechanism in which LTP alternates between lipid bound and unbound states or one in which net transfer of lipid occurs during formation of a ternary collision complex between LTP and the donor and acceptor particles. Regardless of the mechanism, however, LTP may provide a useful method to alter the lipid content of lipoproteins. Indeed, it is known that the lipid content of human LDL influences the conformation of apoB (23) and therefore may affect its interaction with receptors. By choosing appropriate lipid donors and incubation conditions, selective controlled modification of LDL lipid composition by LTP should be possible.

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