Specific Docking of Apolipoprotein A-I at the Cell Surface Requires a Functional ABCA1 Transporter*

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Olivier Chambenoit†, Yannick Hamon‡, Didier Marguet‡, Hervé Rigneault§, Maryvonne Rosseneu†, and Giovanna Chimini¶

From the ¤Centre d’Immunologie INSERM-CNRS de Marseille Luminy, Parc Scientifique de Luminy 13288 Marseille, France, ¶Ecole Normale Supérieure de Physique de Marseille, Institut Fresnel, Domaine Universitaire Saint Jérôme 13397 Marseille, France, and the ¤Department of Biochemistry, University of Gent, 9000 Gent, Belgium

The identification of defects in ABCA1 as the molecular basis of Tangier disease has highlighted its crucial role in the loading with phospholipids and cholesterol of nascent apolipoprotein particles. Indeed the expression of ABCA1 affects apolipoprotein A-I (apoA-I)-mediated removal of lipids from cell membranes, and the possible role of ABCA1 as an apoA-I surface receptor has been recently suggested. In the present study, we have investigated the role of the ABCA1 transporter as an apoA-I receptor with the analysis of a panel of transfectants expressing functional or mutant forms of ABCA1. We provide experimental evidence that the forced expression of a functional ABCA1 transporter confers surface competence for apoA-I binding. This, however, appears to be dependent on ABCA1 function. Structurally intact but ATPase-deficient forms of the transporter fail to elicit a specific cell association of the ligand. In addition the diffusion parameters of membrane-associated apoA-I indicate an interaction with membrane lipids rather than proteins. These results do not support a direct molecular interaction between ABCA1 and apoA-I, but rather suggest that the ABCA1-induced modification of the lipid distribution in the membrane, evidenced by the phosphatidylserine exofacial flipping, generates a biophysical microenvironment required for the docking of apoA-I at the cell surface.

The removal of cellular lipids is promoted by high density lipoproteins (HDL), the plasma shuttle mediating reverse cholesterol transport from peripheral tissues to the liver for further uptake and metabolism (1). However, whether the interaction of the lipid-poor apoA-I particle, protein core of the nascent HDL, with cell membranes is mediated by a specific receptor and how its loading with phospholipids and cholesterol occurs is still a matter of debate (2). The recent discovery that a defective ABCA1 transporter leads to Tangier disease (3–9) has directly implicated this transmembrane protein in the active release of cellular lipids and prompted an investigation into its role as a candidate apoA-I receptor (10, 11). Indeed a correlation between the cAMP-induced cell surface apoA-I binding and the expression of ABCA1 in macrophage-like cell lines has been reported (12). Very recently, in addition, a direct molecular interaction between ABCA1 and apoA-I at the cell surface has been proposed on the basis of chemical cross-linking experiments (10, 11). To gain further insight into this issue, we developed an apoA-I binding assay based on the use of a fluorochrome-conjugated ligand. The analysis of apoA-I binding to a panel of transfectants expressing either functionally intact or defective ABCA1 proteins (13) led us to exclude that the transporter behaves as a bona fide receptor for apoA-I. Indeed, whereas surface binding increases with the expression of a functional ABCA1, the expression of structurally intact but functionally impaired ABCA1 proteins fails to elicit specific binding. Considering that, as previously demonstrated, ABCA1 promotes the transbilayer redistribution of phospholipids at the plasma membrane (13), we propose that ABCA1 favors the specific docking of apoA-I at the cell surface by providing a distinctive spatial arrangement of phospholipid species in the outer membrane leaflet. This model is supported by: (i) the colinear increase of apoA-I binding and exofacial PS exposure as a function of ABCA1 expression, and (ii) the mobility parameters of membrane-bound apoA-I. Indeed, the values of translational diffusion coefficients, assessed by fluorescence correlation spectroscopy (FCS) are consistent with the molecular interaction of apoA-I with rapidly diffusing lipids rather than membrane-anchored receptors (14–16).

EXPERIMENTAL PROCEDURES

Cell Culture—RAW 264.7 cells (ATCC, Rockville, MD) were routinely maintained in culture in Dulbecco’s modified Eagle’s medium supplemented with 10% fetal bovine serum, 1 mM sodium pyruvate, 1 mM penicillin/streptomycin. ABCA1-EGFP, mutant ABCA1-EGFP, and control transfectants were obtained as described (13) and maintained under hygromycin selection (0.2 mg/ml). cAMP stimulation was performed for 24 h at 37 °C in the presence of 0.3 mM cpt-cAMP (Sigma-Aldrich).

Turbitimetric Assay—Fluorescent-labeled apoA-I was tested versus native apoA-I for its ability to interact with dimyristoylphosphatidylcholine (DMPC) vesicles and analyzed by monitoring the decrease in optical density at 325 nm as a function of temperature as described (17, 18).

Lipids Effluxes—Cells were labeled for 72 h in Dulbecco’s modified Eagle’s medium containing 1% fetal calf serum, 1.5 mM [14C]cholesterol, and 10 μCi/ml [14C]choline chloride (both from Amersham Pharmacia Biotech). Cells were then incubated for 24 h in Dulbecco’s modified Eagle’s medium, 1% fetal calf serum with or without 0.3 mM cpt-cAMP. Cells were then washed in phosphate-buffered saline, 0.5% bovine serum albumin, and effluxes were performed for 16 h in a 0.5%
bovine serum albumin medium with or without 10 μg/ml apoA-I. Medium was separated from cells, and lipids were extracted with chloroform and methanol (19). Radioactivity in the medium and cells was determined by liquid scintillation counting. The percentage of efflux is expressed as the number of counts in the medium divided by the total number of counts. Each value is the average of four points.

Immunoprecipitation—Immunoprecipitation analysis was performed on 107 RAW 264.7 cells labeled overnight with 300 μCi/ml 35S protein labeling mix (PerkinElmer Life Sciences). For all experiments the labeled apoA-I (apoA-I/Cy5) was diluted to 100 μg/ml in binding buffer (1.8 mM CaCl2, 1 mM MgCl2, 5 mM KCl, 150 mM NaCl, 10 mM HEPES, pH 7.4), and aggregates were removed by ultracentrifugation for 30 min at 100,000 × g. Binding was performed in the presence of 10 μg/ml of apoA-I/Cy5 (or as indicated for saturation experiments) at 4 °C on 5 × 106 cells detached by mild trypsinization (0.005% in phosphate-buffered saline). At the end of the incubation period, cells were rapidly washed prior to fixation with 1% paraformaldehyde.

Annexin V Binding—Annexin V (ann-V/Cy5) labeled as described (22), was diluted at 4 μg/ml in binding buffer, and aggregates were removed by ultracentrifugation for 30 min at 100,000 × g. Binding was performed in binding buffer for 10 min at 4 °C, in the presence of a final annexin V/Cy5 concentration of 4 μg/ml. The mean of the relative fluorescence intensity (RFI) for annexin V/Cy5 was calculated for each subset of given EGFP RFI.

**RESULTS**

Fluorescence-based assay for apoA-I binding—We previously reported that the forced expression of an ABCA1-EGFP chimera is able to induce an increased cellular release of choline-containing phospholipids and cholesterol to the specific acceptor apoA-I (13). In the same experimental system, we also observed that a reduction of lipid effluxes tracks the progressive silencing of ABCA1 expression induced by tetracycline (not shown). To better characterize the functional effects of the graded ABCA1 expression in transfected cells, easily monitored by FACS analysis, we set out to develop a fluorescence-based assay of apoA-I binding. The assay was first validated by checking whether the fluorochrome conjugation altered the physiological properties of apoA-I. No significant difference in the behavior of labeled versus unlabeld apoA-I was detectable by a standard turbidimetric assay for phospholipid binding (Fig. IA) nor by a classical phospholipid and cholesterol efflux assay...
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FIG. 3. ApoA-I binding is exclusively induced by a functional ABCA1 transporter. A, dot plots of dual-channel flow cytometric analysis on AG and mock-transfected control cells show that the presence of ABCA1 molecules (EGFP channel) elicits apoA-I binding (Cy5 channel). The histogram panels show the behavior of Cy5 RFI on the gated cell populations. A right shift of mean RFI in ABCA1 positive versus negative or mock-transfected cells is detectable. Gates are shown on the dot panels. B, binding parameters of ABCA1-induced apoA-I cell association as measured in the presence of the indicated increasing amounts of labeled apoA-I. ○, EGFP-negative; ■, EGFP-positive cells; ▲, ABCA1-induced binding. In the representative experiment (of 3) values are: $K_d$ 21.5 × 10⁻⁵ M, $B_{max}$ 65.9 ± 5 AU. C, dual-channel cytometric analysis on transfectants expressing mutants form of ABCA1 shows that the function of ABCA1 is essential to elicit apoA-I binding. Dot plots indicate the lack of increase in Cy5 RFI even at high values of EGFP RFI. Histogram plots recorded on Cy5 channel further demonstrate that the mean Cy5 RFI is indistinguishable from that of mock-transfected or EGFP-negative cells. Gates are shown on the dot plots. D, saturation curves of ApoA-I binding on MM cells, expressing the ATPase-deficient form of ABCA1 shows that the function of ABCA1 is essential to elicit apoA-I binding.

The flow cytometric analysis of apoA-I cellular binding was then performed on unstimulated RAW cells and showed a very low level of cell-associated fluorescence (Fig. 2A). In agreement with previously reported data (12, 25), the cell-associated fluorescence homogeneously increased after cAMP treatment (24 h at 37°, 0.3 mM), as shown by the right shift of the mean RFI (3.4 ± 0.4-fold increase over unstimulated cells, $n = 4$). As expected the cAMP-induced binding could be competed by a 50-fold molar excess of unlabeled apolipoprotein. The increase in apoA-I surface binding was paralleled by an increased synthesis of ABCA1 protein as detected by immunoprecipitation of metabolically labeled RAW cells (Fig. 2B). This is likely to result from a CAMP-mediated transcriptional activation of the ABCA1 gene, as suggested by Refs. 11, 25, and 26. The saturation curves measured in the presence of increasing amounts of labeled ligand on cAMP-stimulated and -unstimulated RAW cells allowed the estimation of the parameters of specific cAMP-induced apoA-I binding in our assay ($K_d$ of 1.44 ± 0.12 μg/ml/5.1 ± 0.4 × 10⁻⁸ M, $n = 3$) (Fig. 2C; Refs. 25, 27).

ApoA-I Surface Binding Requires Functional ABCA1—To elucidate the link between ABCA1 expression and apoA-I binding, we carried out a series of experiments on macrophages derived from ABCA1-null animals (9, 13), which showed a 2.1 ± 0.2-fold decrease in apoA-I surface labeling versus cells from wild-type controls ($n = 2$, not shown) and on AG cells, i.e. HeLa cells expressing a functional chimeric ABCA1 transporter under the control of a tetracycline-sensitive promoter (13). The chimera consists of a C-terminal fusion of EGFP to the 2261 amino acid full-length mouse ABCA1 transporter (GenBankTM/EBI accession number X75926; nucleotides 84–6869, Ref. 28). By means of dual-channel flow cytometric recordings, we analyzed the behavior of apoA-I surface binding in these cells as a function of ABCA1 expression (Fig. 3). The cell population was subdivided into EGFP-negative cells (i.e. cells that have lost the expression of the transporter) and EGFP-positive cells by manually gating below or above the threshold of autofluorescence. As shown in Fig. 3A, EGFP-negative AG cells demonstrated a very low binding, comparable with that of nonstimulated RAW or of control mock-transfected HeLa cells.

Conversely, the whole population of EGFP-positive (RFI > 4 in the experiment shown) AG cells shows a significant increase in apoA-I-specific binding (3.2 ± 1.4-fold increase in mean RFI over negative cells, $n = 10$), similar to that induced by cAMP treatment on RAW cells. The specific binding showed an apparent $K_d$ of 24.3 ± 8 × 10⁻⁸ M, 6.9 ± 2.4 μg/ml, $n = 3$ (Fig. 3B). In contrast to the macrophage cell line, no modification of binding was observed after 24 h of cAMP incubation or cholesterol loading of AG and mock-transfected cells (not shown). This indicates that both stimuli, at least under our experimental conditions, do not promote post-translational activation of the transporter, and this indirectly confirms previous reports locating its action to the transcriptional level. In addition, the lack of induction of apoA-I binding on mock-transfected HeLa cells supports a cell-restricted sensitivity of ABCA1 regulatory sequences to both the CAMP and cholesterol-mediated activation (27). To establish whether the physical presence of ABCA1 at the cell surface was sufficient to elicit apoA-I binding, we measured the interaction of labeled apoA-I to transfectants expressing mutant forms of ABCA1 (KM, MK, and MM). As already described, the mutations harbored by these proteins hamper ATP binding/hydrolysis at either or both the nucleotide-binding cassettes of the transporter without altering its folding or its intracellular routing (13). No specific apoA-I binding was detectable despite the presence of equivalent amounts of transporter at the cell surface, as indicated by similar intensity of EGFP fluorescence in the cell lines tested (Fig. 3, C and D). These data thus clearly indicate that an intact function of
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**Fig. 4. ApoA-I interaction at the cell surface.** ABCA1-induced apoA-I docking at the cell surface is saturable. A, dot plots of dual-channel flow cytometric recordings on AG cells. Gates corresponding to three arbitrarily chosen ABCA1 expression levels (+, ++, and ++++) are shown. In the experiment shown these are ABCA1+ EGFP RFI: >5 and <10; ABCA1++ EGFP RFI: >11 and <50; ABCA1++++ EGFP RFI: >50. B, analysis of the effect of increased ABCA1 expression on apoA-I binding parameters shows that the binding affinity is unchanged, whereas $B_{\text{max}}$ estimates increase as a function of ABCA1 expression. $K_0$, $27 \times 10^{8}$ M and $B_{\text{max}}$, 28 ± 2.6 M for ABCA1++; $21 \times 10^{8}$ M and $B_{\text{max}}$, 78 ± 6.7 M and $B_{\text{max}}$, 114 ± 6 M for ABCA1+++ +++. $B_{\text{max}}$ values are in arbitrary units. C, single channel dot plots showing the correlation curve (CC) of apoA-I and ABCA1-EGFP as indicated by arrows. The cross correlation curve (CC) is also shown.

**DISCUSSION**

In this study, we have described the development of a fluorescence-based assay for apoA-I binding, which we applied to the investigation of the ABCA1 transporter as a candidate apoA-I receptor. Based upon our results, we can conclude that the surface expression of ABCA1 is essential to the generation of specific cellular binding sites, but we ruled out ABCA1 as a molecular receptor for lipid-free apolipoproteins.

We actually observed that the expression of apoA-I cell surface association is mediated via the interaction with a membrane-anchored receptor, we measured the mobility parameters of apoA-I and ABCA1-EGFP by FCS (Fig. 4D). This method allows the quantification of the retardation in diffusion acquired by apoA-I after its interaction with the cell membrane of ABCA1-expressing AG cells. The translational diffusion coefficient $D_t$ for free apoA-I was $2.4 \times 10^{-7}$ cm$^2$/sec$^{-1}$ ± 0.6 on 13 independent measurements and for membrane-bound $D_t = 1.6 \times 10^{-8}$ cm$^2$/sec$^{-1}$ ± 1.0 measured on 10 independent cells. The latter values are close to those measured for membrane lipids in fluid phase (15, 16, 29). The lateral diffusion parameters of ABCA1 at the plasma membrane could not be fitted by assuming a single population with uniform diffusion characteristics but were consistently fitted with the model of anomalous diffusion.

The fitting of 13 different data sets from different cells allowed the estimation of a diffusion coefficient $\Gamma$ of $1.1 \times 10^{-10}$ cm$^2$/sec$^2$ ± 0.3 with an $n$ of 0.47 ± 0.06. The different diffusion behavior of ABCA1-EGFP and apoA-I together with the lack of significance of the cross-correlation curves indicates that the two partners are not interacting in the time scale of the detection.

ABCA1 is essential to generate apoA-I binding competence at the plasma membrane.

**ApoA-I Interaction with the Cell Membrane**—Because the expression of ABCA1 in transfectants is heterogeneous, as indicated by the broad distribution of EGFP fluorescence, we manually gated at discrete EGFP fluorescence intensity intervals to detail the behavior of apoA-I binding as a function of the expression of ABCA1 (Fig. 4A). The correlation between apoA-I (reflecting the density of surface binding sites) and EGFP fluorescent intensity (reflecting total cellular content in ABCA1), shown in Fig. 4B, suggests that the surface binding of apoA-I is sensitive to the density of ABCA1 molecules at the cell surface. The latter cannot be assessed directly in our system but can be extrapolated from the total EGFP fluorescence. Indeed the digital quantification of fluorescence distribution on confocal microscopy recordings in low, medium, and high ABCA1-expressing cells showed that a stable fraction (35 ± 5%, n = 16) of total cell-associated fluorescence can be attributed to molecules at the plasma membrane. The saturation curve for apoA-I (Fig. 4B) allowed to estimate that increasing amounts of ABCA1 at the cell surface affected maximum binding without altering binding affinity (in the experiment shown, values are: $K_0$, $27 \times 10^{8}$ M and $B_{\text{max}}$, 28 ± 2.6 M for ABCA1++; $21 \times 10^{8}$ M and $B_{\text{max}}$, 78 ± 6.7 M and $B_{\text{max}}$, 114 ± 6 M for ABCA1+++ +++. By plotting the values of mean RFI for apoA-I (or the $B_{\text{max}}$ values) as a function of ABCA1 expression (Fig. 4C, ■), we observed that the correlation coefficient between the two parameters decreased sharply at high levels of ABCA1 expression. This apparent saturation indicates that the availability of ABCA1 molecules at the cell surface is not the sole parameter affecting the binding of the apolipoprotein.

We then similarly tested the correlation between ABCA1 expression and the exposure of PS at the outer membrane, another ABCA1-elicited phenotype strictly dependent on the activity of the ABCA1 transporter (13). By plotting annexin V binding versus EGFP fluorescence, we detected again a bimodal behavior with a drop in the slope at high ABCA1 expression values (Fig. 4C, ●). This suggests the possibility of a causal link between the two measured phenomena.

To explore further whether apoA-I cell surface association is mediated via the interaction with a membrane-anchored receptor, we measured the mobility parameters of apoA-I and ABCA1-EGFP by FCS (Fig. 4D). This method allows the quantification of the retardation in diffusion acquired by apoA-I after its interaction with the cell membrane of ABCA1-expressing AG cells. The translational diffusion coefficient $D_t$ for free apoA-I was $2.4 \times 10^{-7}$ cm$^2$/sec$^{-1}$ ± 0.6 on 13 independent measurements and for membrane-bound $D_t = 1.6 \times 10^{-8}$ cm$^2$/sec$^{-1}$ ± 1.0 measured on 10 independent cells. The latter values are close to those measured for membrane lipids in fluid phase (15, 16, 29). The lateral diffusion parameters of ABCA1 at the plasma membrane could not be fitted by assuming a single population with uniform diffusion characteristics but were consistently fitted with the model of anomalous diffusion.

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interact with the receptor, and the availability of the molecule in the recipient HeLa cell may be rate-limiting and thus account for the observed saturation of surface binding at high expression of functional ABCA1. Alternatively, the specific docking of apoA-I may not be mediated by interaction with a unique protein receptor, but rather rely on a meticulous molecular arrangement of lipids at the cell surface (31–33). Only the latter would support the interaction of the apolipoprotein with the membrane and the removal of phospholipids and cholesterol which follows. According to this model, apoA-I binding should be considered a consequence of the already demonstrated ability of the transporter to modulate the transbilayer arrangement of lipids (13). The fact that a saturable behavior is also observed when plotting the expression of ABCA1 versus the exofacial PS exposure supports this hypothesis. In the latter case saturation is not surprising. It is conceivable that a cell tolerates only a limited amount of PS on the outer leaflet and that above this threshold a feedback response counteracts further membrane modifications potentially dramatic for cell viability. From a molecular standpoint, this may correspond to an overactivation of the aminophospholipid translocase, or other enzymatic activities able to flip inward the excess PS residues (34). The safety feedback loop will thus concomitantly buffer further increases of both PS flip and apoA-I binding. This hypothesis, schematized in Fig. 5, is reinforced by the diffusion parameters of both membrane-bound apoA-I and ABCA1-EGFP as assessed by fluorescence correlation spectroscopy. The translational diffusion coefficient (Dv) of apo A-I suggests its molecular interaction with lipids rather than with a protein receptor, which should theoretically retard its mobility to a Dv in the range of 10⁻⁹ to 10⁻¹⁰ cm²/sec⁻¹ (15, 16, 29). On the other hand, ABCA1-EGFP behaves according to an anomalous diffusion model (24). This behavior has been previously reported for membrane proteins, like IgE receptor and the LDL receptor (24) and indicates obstruction in lateral diffusion likely to originate from the interaction with other cellular components. In addition, the cross-correlation analysis of diffusion parameters recorded for the ligand and the candidate receptor excludes their interaction in the time scale of the experimental detection.

Our results are only apparently at odds with those of chemical cross-linking reported in (10, 11), that rather emphasizes the spatial proximity of ABCA1 and membrane-bound apoA-I even in the absence of direct molecular interaction. We propose hence that the role played by ABCA1 in promoting apoA-I binding is, in essence, a consequence of the ABCA1-orchestrated modification of the biophysical properties of the membrane. Whether the modification is homogeneously spread over the cell surface or generates only locally and transiently a favorable apoA-I docking environment remains still to be ascertained. The latter case can be reasonably surmised on the basis of the reported physical proximity of the two partners.

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