The Membrane-type Collectin CL-P1 Is a Scavenger Receptor on Vascular Endothelial Cells*

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Collectins are a family of C-type lectins that have collagen-like sequences and carbohydrate recognition domains (CRD). They are involved in host defense through their ability to bind to carbohydrate antigens of microorganisms. The scavenger receptors type A and MARCO are classical type scavenger receptors that have internal collagen-like domains. Here we describe a new scavenger receptor that is a membrane-type collectin from placenta (collectin placenta 1 (CL-P1)), which has a typical collectin collagen-like domain and a CRD. The cDNA has an insert of about 2.2 kilobases coding for a protein containing 742 amino acid residues. The deduced amino acid sequence shows that CL-P1 is a type II membrane protein, has a coiled-coil region, a collagen-like domain, and a CRD. It resembles type A scavenger receptors because the scavenger receptor cysteine-rich domain is replaced by a CRD. Northern analyses, reverse transcription-polymerase chain reaction, and immunohistochemistry show that CL-P1 is expressed in vascular endothelial cells but not in macrophages. By immunoblotting and flow cytometry CL-P1 appears to be a membrane glycoprotein of about 140 kDa in human umbilical vein or arterial endothelial cells, placental membrane extracts, and CL-P1 transfected Chinese hamster ovary cells. We found that CL-P1 can bind and phagocytose not only bacteria (Escherichia coli and Staphylococcus aureus) but also yeast (Saccharomyces cerevisiae). Furthermore, it reacts with oxidized low density lipoprotein (OxLDL) but not with acetylated LDL (AcLDL). These binding activities are inhibited by polyionic ligands (polynisinocic acid, polguanylic acid, dextran sulfate) and OxLDL but not by polycationic ligands (polyadenylic acid or polycitidylic acid), LDL, or AcLDL. These results indicate that CL-P1 might play important roles in host defenses that are different from those of soluble collectins in innate immunity.

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The nucleotide sequence(s) reported in this paper has been submitted to the GenBank†† or EMBL Data Bank with accession number(s)AB005145.

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Collectins are a family of proteins that contain at least two characteristic structures, a collagen-like region and a carbohydrate recognition domain (CRD)1 (1). These lectins are found in vertebrates from avians to humans (2). There are four groups of collectins: the mannan-binding protein (MBP) group including MBP-A and MBP-C (3), the surfactant protein A (SP-A) group (4), the surfactant protein D (SP-D) group (5), and the newly isolated collectin liver 1 (CL-L1) (6). MBP can destroy bacteria through activation of the complement pathway (7) or opsonization via collectin receptors (8). MBP and conglutinin of the SP-D group are β-inhibitors of influenza A viruses that have hemagglutination inhibition and neutralization activities (9, 10). SP-A amplifies the phagocytosis of bacteria by macrophages (11) and opsonizes herpes simplex virus (HSV) (12). SP-D agglutinates bacteria (13) and has hemagglutination inhibition activity against influenza A virus (14). These activities indicate that collectins play an important role in innate immunity (14). In addition, the type A scavenger receptor (SR-A) also contains a collagen-like domain, which forms an oligomeric structure and binding sites (15) that have a broad specificity for ligands. The primary function of scavenger receptors is the destruction and neutralization of pathogens by endocytosis and phagocytosis. Recent knockout data show that SR-AI-deficient mice are sensitive to Listeria and HSV infections. Thus, it appears that scavenger receptors also have a role in innate immunity (16, 17).

Here we report the molecular cloning of a new membrane-type collectin that functions as a scavenger receptor. The cDNA for this receptor was first synthesized from placenta RNA, and the receptor is called collectin placenta 1 (CL-P1). It is present mainly in endothelial cells but is not present in monocyte-macrophage lineage cells. Surprisingly, this new collectin can bind and phagocytose bacteria and yeast as well as oxidized LDL.

** EXPERIMENTAL PROCEDURES

Buffers and Media—Escherichia coli lysis buffer A for the His-Tag system consisted of 6 m guanidine hydrochloride, 0.1 m sodium phos-
phate, and 10 mM Tris, pH 8.0. Column buffers B, C, D, and E consisted of 8 M urea, 0.1 M sodium phosphate, and 10 mM Tris; the pH of each buffer was 8.0, 6.3, 5.9, and 4.5, respectively. LB medium contained 1% (w/v) Bacto-tryptophane, 0.5% (w/v) Bacto-yeast extract, and 1% (w/v) NaCl. IDG medium contained 0.4% (w/v) amino acids, 0.06% NaHPO₄, 0.3% (w/v) glucose, 0.1% (w/v) poly(A⁺)-RNA, 0.1% NaCl, 0.1% MgSO₄, 0.05% (w/v) Tris-buffered saline (TBS) consisting of 20 mM Tris-HCl and 140 mM NaCl, pH 7.4, and TBS/C was TBS containing 5 mM CaCl₂. Coating buffer contained 15 mM Na₂CO₃, 35 mM NaHCO₃, and 0.05% (w/v) NaNO₃, pH 9.6.

**Generation of a Probe for Screening by the Polymerase Chain Reaction (PCR) and/or a Digoxigenin-labeled cDNA Probe (EST) Using a Potential New Collectin Gene Revealed a Novel Gene in EST Clone Clone ID 34472 of W72977**

The primers synthesized were 5'-CAATCTGA-

- and 3'-ACGAGGGCT-GATGGGACAT-3'. For the reverse primer, 5'-ACGAGGGCT-GATGGGACAT-3' for the forward primer. PCR was carried out using a PCR digoxigenin probe synthesis kit (Roche Molecular Biochemicals). The reaction mixture in 50 µl consisted of LA PCR Buffer II (Mg²⁺), TaKaRa LA Taq DNA polymerase (1.25 units), and RT reaction product amplified in a thermal cycler (TaKaRa PCR thermal cycler MP) by 28 cycles of PCR using degenerated primer sets (0.2 µM), TaKaRa LA Taq polymerase (1.25 units), and RT reaction product. The primers for PCR were 5'-TGGCCCTGGCCTGCGC-AATTG-3' (CL-P1), 5'-CCACGCAAGTAAGTGCTT-3' (CL-L1), 5'-ATGTTGTAATGCTGGTT-3' (MBP), 5'-GGGACGACGCTTCTGGAG-3' (SP-A), and 5'-GGGACGACGCTTCTGGAG-3' (SP-D) for the reverse primer sets and 5'-GGGACGACGCTTCTGGAG-3' (CL-P1), 5'-TAGCAATACATGATGGTGG-3' (CL-L1), 5'-TCAGATAGGG-

AUCTACAGAC-3' (MBP), 5'-CCCTGTTCACATGGCACAAAGCC-3' (SP-A), and 5'-TCAGAATCCGCAAGCAGACG-3' (SP-D) for the forward primers. Amplons were separated on 1.0% agarose gels.

**Ligiprotein Preparation—Human LDL was prepared from human plasma by stepwise sodium bromide density gradient centrifugation (20). All sodium bromide stock solutions contained 0.25 mM EDTA**. After centrifugation, LDL was recovered from the fractions with densities of 1.09-1.063 g/cm³. Prior to oxidation, an aliquot of LDL was passed through a 10DG resalting column (Bio-Rad) to remove EDTA. OxLDL was prepared by the incubation of LDL (2 mg/ml) at 37 °C for 24 h with 50 mM CuSO₄. The reaction was stopped by the addition of 300 mM Cu₆S₄O₆. The electrophoretic mobility of the OxLDL on SDS-PAGE was approximately 3 times higher than that of unmodified LDL. The OxLDL contained approximately 50 nmol of thiobarbituric acid-reactive substances (TBARS) in mg of protein (21). The TBARS of the native LDL was about 1 nmol/mg of protein. Acetylation of LDL (AcLDL) was performed as described previously (22). Acetylation resulted in the derivation of more than 75% of the free amino groups on the LDL that were labeled using the binding of LDL, OxLDL, and AcLDL with 11.1-diotadeacyl-3,3,3'-tetra-

methylindocarbocyanine perchlorate (DiI) (Molecular Probes) was performed as described previously (24).

**Antibodies—Expression of the CRD region in CL-P1 (amino acids 590-742 of human CL-P1) in E. coli (pPH13 and E. coli G712) was carried out as described previously (6). The fusion protein CL-P1/CRDhs was used to produce antiseria in chickens.**

**Cell Culture and Isolation of a Transfected Cell Line**—CHO-Id4A17 cells, kindly provided by Dr. M. Krieger (MIT), which lack functional LDL receptors, were maintained at 37 °C in Ham’s F-12 medium containing 5% fetal bovine serum (25). A full-length cDNA of human CL-P1 was amplified from a human placenta cDNA library by PCR using the forward primer 5'-AATGGCGGCGGCAATTGTAGGATTGCACTTGGAG-3' and the reverse primer 5'-GCTTATGGACGCGGAATTGCACTTGGAG-3'.
ATGACAGTAC-3'. The amplified human CL-P1 cDNA was subcloned into pcDNA3.1/Myc-His A vector (Invitrogen), sequenced, and transfected into CHO-ltdA7 cells using LipofectAMINE 2000 (LF2000) reagent (Life Technologies, Inc.) according to the manufacturer's protocol.

To select CL-P1 positive clones, cells were cultured in Ham's F-12 medium containing 5% fetal bovine serum and 0.4 mg/ml G418 (Life Technologies, Inc.). Positive cells were detected and sorted using a FACSVantage flow cytometer (Becton Dickinson) with anti-Myc monoclonal antibody (Invitrogen) and anti-mouse IgG-conjugated Alexa 594 (Molecular Probes). Positive clones were checked by the above method, and a stable clone (CHO/CL-P1) was established. CHO/SR-BI cells, which had been transfected with hamster SR-BI cDNA, were a gift from Dr. H. Arai (26). They were maintained at 37°C in Ham's F-12 medium containing 10% fetal bovine serum and 0.4 mg/ml G418.

Immunohistochemistry, Immunofluorescence Microscopy, and Western Blotting—Mice were anesthetized with 2.5% avertin and perfused through the left ventricle with 20 ml of ice-cold PBS containing 5 mM EDTA and then with 4% paraformaldehyde in PBS at 4°C for 10 h, and hearts were collected and treated as described elsewhere (27). Specimens were dehydrated and embedded in paraffin. Ultrathin sections were stained immunohistochemically and with Mayer's hematoxylin. Immunohistochemistry was done with anti-CL-P1 antibody (chicken IgY), anti-chicken IgY conjugated with HRP (Chemicon International, Inc.), biotinylated tyramide solution, and avidin-Alexa 488 solution using the TSATM biotin system (PerkinElmer Life Sciences). The fluorescent images were observed with an Olympus IX70–23 FL/DIC-SP and SPOT2-SP system (Olympus Optical Co. Ltd.). The transfected cells (CHO/CL-P1) were plated at a density of 3 x 10^5 cells/0.2 ml in 14-mm wells of 35-mm plastic culture dishes (Matsunami Glass Industries, Ltd., Japan) and cultured in Ham's F-12 medium containing 5% fetal bovine serum and 0.4 mg/ml G418. They were not fixed and directly incubated with anti-Myc murine monoclonal antibody and anti-CL-P1 chicken antibody, followed by anti-mouse IgG-conjugated Alexa 594 and anti-chicken IgY-conjugated Alexa 488 (Molecular Probes) as described previously (6). Immunofluorescent flow cytometry was performed with human umbilical vein endothelial cells (HUVEC) and human umbilical artery endothelial cells (HUAEc), both from ATCC. Cells were incu-

Fig. 1. a, the deduced amino acid sequences of human and mouse CL-P1. The amino acid residues are numbered in the N to C direction beginning with the first Met and ending with Leu. The underlined portions are the transmembrane domains, collagen-like domains, and CRDs. The nucleotide sequence data reported in this paper were submitted to the DDBJ, EMBL, and GenBank data libraries under the accession number AB005145. b, the structures of collectin, CL-P1, SR-AI, LOX-1, and SREC.
bated with anti-CL-P1 chicken antibody and anti-chicken IgY-conjugated Alexa 488 at 4°C for 30 min and assayed with a FACS Calibur (Becton Dickinson). Appropriate cell fractions were selected using a two-dimensional display of forward scatter and side scatter. Western blotting analyses were performed using CL-P1 transfected cells, HUVEC, placental tissue membrane extracts (BioChain Institute, Inc., CA) without or with de-glycosylation (Enzymatic Deglycosylation kit, Bio-Rad), and in vitro transcription/translation products (lane 6) were subjected to SDS-PAGE, Western blotting, and probing with chicken anti-CL-P1 antibody (lanes 1, 3, 4, 5, and 6) and mouse anti-Myc antibody (lane 2). The bound antibody was visualized with alkaline phosphatase-conjugated secondary antibody and a BCIP/NBT substrate system.

Analysis of Lipoprotein Binding—CHO/CL-P1, CHO/SR-BI, and CHO-ldlA7 cells were plated at densities of 3 × 10^4 cells/0.2 ml in 14-mm wells of 35-mm plastic culture dishes and cultured in Ham’s F-12 medium containing 5% fetal bovine serum with or without 0.4 mg/ml G418. Cells were incubated at 4°C for 30 min with DiI-OxLDL, DiI-AcLDL, and DiI-LDL. Fluorescent images were observed with the Olympus IX70–23 FL/DIC-SP and SPOT2-SP system (Olympus Optical Co. Ltd.). CHO/CL-P1 cells were incubated at 4°C for 2 h with 5 µg/ml DiI-OxLDL in the presence at 200 µg/ml of LDL, AcLDL, and OxLDL24, 10 µg/ml dextran sulfate, polycationic ligands (poly(A), poly(G)), and polyanionic ligands (poly(G), poly(I)) (29). To quantify the amount of DiI-OxLDL, cells were washed and then fixed with PBS containing 4% paraformaldehyde, pH 7.4, treated with 1 drop of SlowFade antifade.

**Fig. 2.** Detection of CL-P1 mRNA by RT-PCR and Northern blot analyses of poly(A)^+ RNAs from various human tissues. a, RT-PCR analyses using total RNAs (1 µg) from brain, heart, kidney, liver, lung, trachea, bone marrow, colon, small intestine, spleen, stomach, thymus, mammary gland, prostate, skeletal muscle, testis, uterus, cerebellum, fetal brain, fetal liver, spinal cord, placenta, adrenal gland, pancreas, salivary gland, and thyroid gland. b, Northern blot analyses of poly(A)^+ RNAs (2 µg) from heart, brain, placenta, lung, liver, skeletal muscle, kidney, and pancreas. Calculated sizes of the RNAs detected are indicated by arrows.

**Fig. 3.** Detection of CL-P1 protein by immunohistochemistry, flow cytometry, membrane immunofluorescence, and immunoblotting. a, paraformaldehyde-fixed sections of murine heart that was cut vertically were stained with chicken anti-human CL-P1-CRD antibody (left, × 10, × 40) and counterstained with hematoxylin eosin (right, × 10, × 40). The arrowheads indicate endothelial cells in micro and small vessels surrounding heart smooth muscle cells, and the arrows indicate endothelial cells in the coronary artery. b, flow cytometry showed membrane immunofluorescence in HUVEC and HUAEC. c, membrane immunofluorescence analyses showed CL-P1 on the surfaces of transfected CHO cells using anti-Myc tag and anti-CL-P1 antibodies. d, extracts of CHO/CL-P1 cells (lanes 1 and 2), HUVEC (lane 3), placenta (lane 4), deglycosylated placenta (lane 5), and in vitro transcription/translation products (lane 6) were subjected to SDS-PAGE, Western blotting, and probing with chicken anti-CL-P1 antibody (lanes 1, 3, 4, 5, and 6) and mouse anti-Myc antibody (lane 2). The bound antibody was visualized with alkaline phosphatase-conjugated secondary antibody and a BCIP/NBT substrate system.
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RESULTS AND DISCUSSION

Molecular Cloning of the CL-P1 Gene—We screened DNA data bases to identify novel members of the collectin family and identified a cDNA fragment from human EST data bases that showed carboxy-terminal sequence homology with the collectins. The EST clone W72977 from a fetal heart cDNA library was used to screen a human placenta cDNA library, and positive clones were isolated. In addition, “Cap-site hunting” (19) was performed to determine the complete 5’-terminal sequence including the transcription start site of a new collectin mRNA. Restriction mapping and sequencing of the clones revealed that they contained an open reading frame of 2226 base pairs encoding a sequence of 742 amino acids (Fig. 1a). The deduced amino acid sequence revealed a collectin structure consisting of a collagen-like region and a CRD. This new collectin, designated collectin placenta 1 (CL-P1), has an intracytoplasmic domain, a transmembrane domain with a coiled-coil region, a collagen domain, and a CRD (Fig. 1b). At the amino acid level the cloned mouse CL-P1 has high sequence identity (92%) and the same length and same domain sizes as human CL-P1 (Fig. 1a). The homology between human and mouse CL-P1 is the highest among the collectins. The collagen domain had the highest homology and has 49 more Gly-X-Y cycles than SR-AI (15). CL-P1 has three polycationic regions in a collagen domain that contain basic amino acids (arginine or lysine). These amino acid sequences are almost identical to those in human and mouse CL-P1. CL-P1 has a C-type lectin consisting of six cysteine residues, which is highly homologous to the CRDs in macrophage lectin 2 and the asialoglycoprotein receptor (1). Its ligand specificity is of the galactose type (Gln-Pro-Asp), which is different from the mannose and glucose types (Glu-Pro-Asn) (30). The whole structure of CL-P1 resembles that of SR-AI (Fig. 1b). The structures of other SRs, LOX-1 (31) and SREC (29), expressed in endothelial cells are completely different from those of SR-AI and CL-P1. SR-AI and CL-P1 can form oligomeric structures due to their collagen-like regions and coiled-coil structures. The polycharge islands in the collagen-polymer structure form a strong binding site for negatively charged substances. An endocytosis motif (Tyr-Lys-Arg-Phe) (32), like in the asialoglycoprotein receptor, is present in the intracytoplasmic domain.

Localization of CL-P1 in Tissues and Cells—RT-PCR analy-
cells was performed at 37 °C overnight under 5% CO₂. After the same staining as in a, phagocytosed bioparticles were observed under a confocal laser scanning microscope.

Fig. 5. Binding of microbes to CHO/CL-P1 cells. a, photographs of CL-P1 expression and microbe binding. CHO/CL-P1 cells were stained with anti-Myc antibody and anti-mouse IgG conjugated with Alexa Fluor™488. BioParticles of E. coli, S. aureus, and yeast (S. cerevisiae) conjugated with Texas Red or tetramethylrhodamine were used. b, the uptake of S. cerevisiae BioParticles by CHO/CL-P1 cells was performed at 37 °C overnight under 5% CO₂. The same staining as in a, phagocytosed bioparticles were observed under a confocal laser scanning microscope.

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