Chemokines Generally Exhibit Scavenger Receptor Activity through Their Receptor-binding Domain*

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Chemokines are a family of cytokines that induce directed migration of various types of leukocytes through specific interactions with a group of seven transmembrane receptors. Scavenger receptors are a heterogeneous family of transmembrane molecules that commonly bind and uptake oxidized low density lipoprotein and bacteria. Here, we show that not only CXC chemokine 16 (CXCL16)/SR-PSOX, a transmembrane chemokine with scavenger receptor activity, but also 12 out of 15 chemokines examined efficiently bound scavenger receptor ligands in competition with cells expressing their specific chemokine receptors. Furthermore both the chemotactic and scavenger receptor activities of SR-PSOX/CXCL16 were similarly impaired in a series of mutants altered in the chemokine domain, indicating that SR-PSOX/CXCL16 binds scavenger receptor ligands as well as CXCR6 using highly overlapping binding motifs. Taken together, chemokines generally have scavenger receptor-like activity through their receptor-binding domain, suggesting a close evolutionary relationship between chemokines and scavenger receptors.

The chemokine superfamily consists of small, heparin-binding cytokines that induce directed migration of various types of leukocytes through specific interactions with a group of seven transmembrane G protein-coupled receptors (1). More than 40 chemokines have been identified and classified into four subfamilies, C, CC, CXC and CX3C, based on the motif formed by the conserved cysteine residues in the amino-terminal region.

Chemokines have been considered to play important roles not only in the immune system but also in the development and remodeling of the body (2–4).

Scavenger receptors are a highly heterogeneous group of cell-surface molecules that commonly bind and take up oxidized low density lipoprotein (OxLDL) (5). Currently, scavenger receptors are categorized into almost 10 classes on the basis of their structures, even though there are few structural and primary amino acid sequence similarities among the classes (5). While scavenger receptors were primarily studied for their roles in foam cell formation and the pathogenesis of atherosclerosis, they have been considered a kind of so-called pattern recognition receptor, since most of them were also shown to bind a broad range of negatively charged molecules including bacteria and dextran sulfate (5–7).

Recently, SR-PSOX/CXC chemokine 16 (CXCL16) has been identified and characterized not only as a novel scavenger receptor for OxLDL, phosphatidylserine, dextran sulfate, and bacteria (7, 8) but also as a membrane-anchored chemokine for the G protein-coupled receptor CXCR6 (9, 10). Soluble SR-PSOX/CXCL16, which is released from macrophages and dendritic cells upon cleavage by membrane metalloproteinase (11), induces the migration of CXCR6-expressing activated T and NKT cells. In addition to this activity, cells expressing membrane-anchored SR-PSOX/CXCL16 can uptake OxLDL and phagocytose bacteria as well as adhere to CXCR6-expressing cells. These scavenger receptor activities have been implicated in foam cell transformation leading toatherogenesis and in protection from bacterial infection (6, 12).

In addition to the biological activities induced by chemokine receptor-mediated signal transduction, some chemokines such as NAP-2/CXCL7, MIG/CXCL9, IP-10/CXCL10, I-TAC/CXCL11, and MEC/CCL28 were recently reported to exhibit antimicrobial activity (13–15). Most of the antimicrobial chemokines were indicated to contain a positively charged COOH-terminal segment in addition to a chemokine domain (14), which was suggested to play a role in the antimicrobial activity (16).

Thus, some chemokines are suggested to be capable of binding their specific receptors and bacteria in the case of chemokines with antimicrobial activity. However, it has not been clear whether chemokines generally can bind bacteria. In addition, the precise relationships among these binding activities have not been analyzed at a molecular level. Here, we have shown that chemokines generally exhibit scavenger receptor-like activity using a highly overlapping binding domain to their specific receptors.

**EXPERIMENTAL PROCEDURES**

**Materials and Cells—**Human LDL and OxLDL were prepared and labeled with 1.1-dioctadecyl-3,3,3,3-tetramethylindocarbocyanin perchlorate (DiI) (Molecular Probes) or 125I as described previously (8). The murine pre-B cell line L1.2 was generously provided by Dr. E. Butcher at Stanford University School of Medicine. L1.2 cells stably expressing CXCR6,CCR1,CCR2,CCR4,CCR5,CCR7, and CCR10 (designated as L-CXCR6, L-CCR1, L-CCR2, L-CCR4, L-CCR5, L-CCR7, and L-CCR10 cells, respectively) were generated as described previously (7). Jurkat

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human T cells stably expressing CXC1R (L-CXCR1 cells) and Murine pre-B 300–19 cells stably expressing CXC6R3 (BC-CXCR3 cells) were generously provided by Dr. B. Moser at Theodor-Kocher Institute, University of Bern. 

Mutagenesis—Site-directed mutagenesis in the chemokine domain of human SR-PSOX was carried out using a QuikChange site-directed mutagenesis kit (Stratagene, La Jolla, CA). SR-PSOX mutants (K42A, R57A, R59A, K60A, R67A, and R73A) had an alanine substitution at lysine 42, arginine 57, lysine 59, arginine 60, arginine 67, and arginine 73 of SR-PSOX, respectively.

ELISA for SEAP-fused Chemokines Binding to OxLDL—OxLDL (10 μg/ml:50 μM/well) was loaded onto ELISA plates and incubated for 2 h at 37°C. The plates were washed and treated with 4-fold diluted BlockAce (Dainippon Seiyaku, Osaka, Japan) for 1 h at room temperature. After three washes with PBS, 10 ng of each chemokine fused at its COOH terminus with SEAP (100 μM/well) was loaded, and the plates were incubated for 2 h at room temperature. After three washes with PBS, polyclonal anti-SEAP antibody (50 μM/well) was loaded, and the plates were incubated for 1 h at room temperature. After three washes with PBS, anti-rabbit IgG-horseradish peroxidase (50 μg/well) was added to each well. After incubation for 5–30 min at room temperature, stop solution (100 μM/well) was added to each well, and the optical density at 450 nm was determined using a Wallac 1420 Fluoroscan (Wallac, Turku, Finland).

Phylogenetic Tree—A phylogenetic tree was constructed from the amino acid sequences of chemokines and chemokine domains using the Parallel prp and phylp programs (17).

RESULTS

Most Chemokines Have Potential Scavenger Receptor Activities—Since SR-PSOX/CXCL16 exhibits both chemokine and scavenger receptor activities (8), and some chemokine can kill bacteria probably through directly binding to bacteria (13–15), we examined whether chemokines generally have scavenger receptor activity. We selected 15 chemokines (as described in Table I) and prepared SEAP-tagged forms. We confirmed that the chemokines tagged with SEAP at their COOH terminus were chemotactic for cells expressing their receptors (data not shown). Then, we examined their binding to OxLDL immobilized to plate (Table I). Twelve chemokines (SR-PSOX/CXCL16, CXCL9, CXCL10, SDF/CXCL12, RANTES/CCL5, eotaxin/CCL11, TARC/CCL17, ELC/CCL19, LARC/CCL20, SLC/CCL21, CTACK/CCL27, and CCL28) clearly bound OxLDL (Table I) and prepared SEAP-tagged forms. We confirmed that values in the absence and presence of each chemokine without OxLDL were set as 100 and 0%, respectively. Bold characters indicate values for SEAP without chemokine. Bold characters represent binding activity comparable with or greater than that of SR-PSOX.

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Inhibitory activity of OxLDL for the binding and chemotactic activities of SEAP-fused chemokines to their specific receptors were quantified as described previously (7, 28). Values from SR-PSOX/CXCL16 were set as 100%. The control indicates values for SEAP without chemokine. Bold characters represent binding activity comparable with or greater than that of SR-PSOX.

The binding to E. coli or S. aureus (3 x 10^7 cells/well) was measured as described previously (7). Values from SR-PSOX/CXCL16 were set as 100%. The control indicates values for SEAP without chemokine. Bold characters represent binding activity comparable with or greater than that of SR-PSOX.

We further examined the binding of these chemokines to bacteria. Group A chemokines, except for CXCL9, CXCL12, and CCL5, were shown to bind to both Escherichia coli and Staphylococcus aureus, while CXCL9 could bind only to S. aureus and CXCL12, and CCL5 could bind only to E. coli (Table I). On the other hand, group B chemokines bound to neither of these bacteria at all (Table I). In addition, group A chemokines, except for CXCL12, were shown to efficiently adhere to dextran sulfate, while group B chemokines could not bind to dextran sulfate at all (data not shown). Collectively, most of the group A chemokines, which could bind to OxLDL, also efficiently bound to bacteria and dextran sulfate, while group B chemokines, which could not bind to OxLDL, did not bind to bacteria or dextran sulfate.

Scavenger Receptor Ligands Block Chemokine Activity—We next examined whether scavenger receptor ligands are capable of inhibiting the binding of various chemokines to their receptors (Table I). We confirmed the specific binding of each SEAP-tagged chemokine to its receptor-expressing cells except for CCL11-SEAP, which showed high levels of nonspecific binding to L1.2 cells (data not shown). Jurkat cells, whose endogenous expression of CXCR4 was confirmed by flow cytometry (data not shown), were used as target cells for CXCL12.

OxLDL blocks the specific binding of all examined group A chemokines to their receptors (Table I), while no such inhibitory effect was seen with native LDL (data not shown). On the other hand, OxLDL did not inhibit the specific binding of all group B chemokines to their receptors. Dextran sulfate showed the same inhibitory activity as OxLDL (data not shown).

We further examined whether scavenger receptor ligands

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**Table I**

| Chemokine | Relative binding activity to scavenger receptor ligands \( ^{a} \) | Inhibition of chemokine activities by OxLDL \( ^{b} \) |
|-----------|--------------------------------------------------|----------------------------------|
|           | OxLDL | E. coli | S. aureus | Receptor expressing cells | Binding | Chemotaxis |
| Group A   |        |        |           |                      |        |            |
| Control   | 1 ± 1  | 9 ± 6  | 13 ± 1    | L-CXCR6               | 85 ± 6  | ND          |
| CXCL16    | 100 ± 4 | 100 ± 12 | 100 ± 34 | B-CXCR3               | 89 ± 12 | 99 ± 1     |
| CXCL9     | 217 ± 2 | 35 ± 4  | 101 ± 2   | B-CXCR3               | 46 ± 12 | 35 ± 4     |
| CXCL10    | 128 ± 5 | 112 ± 24 | 10 ± 4    | Jurkat                | 82 ± 12 | 81 ± 7     |
| CXCL12    | 128 ± 32 | 79 ± 25 | 33 ± 24   | L-CRF1                | 75 ± 6  | 8 ± 12     |
| CCL5      | 79 ± 11  | 76 ± 11  | 73 ± 3    | L-CR3                 | ND      | ND         |
| CCL11     | 128 ± 11 | 71 ± 9  | 117 ± 7   | L-CR4                 | 65 ± 1  | 79 ± 5     |
| CCL17     | 128 ± 11 | 71 ± 9  | 117 ± 7   | L-CR7                 | 87 ± 2  | 58 ± 5     |
| CCL19     | 128 ± 11 | 71 ± 9  | 117 ± 7   | L-CRF6                | 93 ± 1  | 99 ± 1     |
| CCL20     | 128 ± 11 | 71 ± 9  | 117 ± 7   | L-CRF7                | 76 ± 2  | 71 ± 5     |
| CCL21     | 128 ± 11 | 71 ± 9  | 117 ± 7   | L-CR10                | 51 ± 36 | 98 ± 3     |
| CCL22     | 128 ± 11 | 71 ± 9  | 117 ± 7   | L-CR10                | 60 ± 3  | 100 ± 0    |

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\( ^{a} \) The binding of SEAP-fused chemokines to OxLDL-coated plates was quantified as described under “Experimental Procedures.” The binding to E. coli or S. aureus (3 x 10^7 cells/well) was measured as described previously (7, 28). Values from SR-PSOX/CXCL16 were set as 100%. The control indicates values for SEAP without chemokine. Bold characters represent binding activity comparable with or greater than that of SR-PSOX.

\( ^{b} \) Inhibitory activity of OxLDL for the binding and chemotactic activities of SEAP-fused chemokines to their specific receptors were quantified as described previously (11, 28). Values in the absence and presence of each chemokine without OxLDL were set as 100 and 0%, respectively. Bold characters indicate more than 35% inhibitory activity by OxLDL ND, not determined.
are capable of inhibiting the chemotactic activity of these chemokines (Table I). OxLDL inhibited the chemotactic activity of all group A chemokines except for CCL5, although the binding of CCL5 to its receptor was inhibited by OxLDL. In contrast, OxLDL did not inhibit the chemotactic activity of group B chemokines except for CXCL8. Although OxLDL inhibited the chemotactic activity of CXCL8 toward CXCR1-expressing Jurkat cells (J-CXCR1 cells), this effect could be nonspecific because 1) OxLDL did not block the binding of CXCL8 to J-CXCR1 cells and 2) OxLDL was reported to be toxic to Jurkat cells (18). Dextran sulfate inhibited the chemotactic activity of all group A chemokines, while it did not inhibit the chemotactic activity of group B chemokines (data not shown).

Collectively, both the receptor binding and chemotactic activities of most of the group A chemokines that recognized OxLDL were strongly inhibited by OxLDL and dextran sulfate.

**Mutational Analysis of SR-PSOX/CXCL16**—We then minutely examined the binding domain of SR-PSOX/CXCL16 to its chemokine receptor CXCR6 and various scavenger receptor ligands on the molecular level. SR-PSOX/CXCL16 is the first chemokine that has been identified as a scavenger receptor molecule (8). The extracellular part of SR-PSOX/CXCL16 is composed of two distinct domains, namely the chemokine domain and the mucin domain. We have confirmed that the chemokine domain of SR-PSOX without the mucin and transmembrane domains induces chemotaxis of CXCR6-expressing cells (data not shown). Furthermore, we have previously indicated that only the chemokine domain of SR-PSOX/CXCL16 is required for the recognition of bacteria, and bacterial phagocytosis via SR-PSOX is inhibited by OxLDL (7). In addition, the chemokine domain of SR-PSOX/CXCL16 is solely responsible for binding OxLDL (data not shown).

Both the chemotaxis and adhesion of CXCR1-expressing cells, by CXCL1 that is another transmembrane chemokine than SR-PSOX/CXCL16, were reported to be critically impaired by the replacement of basic amino acid residues in the chemokine domain of CXCL1 with alanine (19). To examine the recognition sites of SR-PSOX for bacteria, OxLDL, and CXCR6, we prepared a series of mutants of human SR-PSOX/CXCL16 by replacing a single basic amino acid residue in the chemokine domain, which is conserved in human, mouse, and pig SR-PSOX (lysine 42, arginine 57, arginine 59, lysine 60, arginine 67, and arginine 73 in human SR-PSOX), with alanine (designated K42A, R57A, R59A, K60A, R67A, and R73A, respectively). We generated soluble forms of these mutant SR-PSOX proteins as a SEAP fusion protein and examined their chemotactic activity on L-CXCR6 cells (Fig. 1A). Furthermore, we prepared COS-7 cells expressing each mutant of SR-PSOX and examined the phagocytosis of bacteria (Fig. 1B) and binding and uptake of OxLDL (Fig. 1C).

**Discussion**

Here, we have demonstrated that 12 out of 15 chemokines examined are capable of binding scavenger receptor ligands such as OxLDL and bacteria (Table I). OxLDLs were also shown to be able to competitively inhibit the binding and chemotactic activity of these chemokines toward cells expressing their specific chemokine receptors (Table I). We also analyzed scavenger receptor activity in the 15 chemokines, which were fused at their COOH terminus with the mucin, transmembrane, and cytoplasmic domains of SR-PSOX/CXCL16, expressed on the surface of COS-7 cells. Most of group A chemokines could, whereas group B chemokines could not, mediate the uptake of bacteria and OxLDL (data not shown). All the results indicate that most of chemokines have potential scavenger receptor activity. In addition, SR-PSOX/CXCL16, which is the first chemokine identified as a scavenger receptor, was suggested to bind not only its specific receptor CXCR6 but also OxLDL and bacteria by using an almost identical binding site (Fig. 1). All the results suggest that most of the chemokines examined, which bind to their chemokine receptors in a specific one to one manner, can also bind OxLDL and bacteria in a so-called pattern recognizing manner using almost the same chemokine receptor-binding domain.

Among the 15 chemokines examined, CXCL1, CXCL5, and CCL2 could not recognize OxLDL and bacteria. Interestingly, CXCL1 and CCL2 were reported to be the most important chemokines in the pathogenesis of atherosclerosis (20, 21). The lack of OxLDL binding activity in CXCL1 and CCL2 may allow these chemokines to exhibit chemotactic activity toward targeted leukocytes in atherosclerotic lesions even in the presence of high local concentrations of OxLDL. Conversely, OxLDL may suppress various immune reactions by binding to various che-
Chemokines Show Scavenger Receptor Activity

Fig. 2. Phylogenetic tree of chemokines. A phylogenetic tree of the chemokine family members was constructed from the amino acid sequences of chemokine domains using the Parallel prp and phylog programs (17). ZF-CXC, ZF-CC, and XCL indicate Zebrafish CXC chemokine, Zebrafish CC chemokine, and lymphotactin, respectively. Bold characters represent group B chemokines (see “Results”).

Some chemokines such as CXCL9, CXCL10, and CCL28 were reported to exhibit antimicrobial activity as well as chemotactic activity (13–15). Interestingly, CCL27, which is reported to have less antimicrobial activity than CCL28, exhibits strong binding activity toward bacteria and OxLDL (Table I). Similarly, SR-PSOX/CXCL16 with bacterial binding and phagocytic activities showed much less antimicrobial activity than previously reported antimicrobial chemokines. The bacterial binding and phagocytic activities of SR-PSOX/CXCL16 were indicated to require basic amino acid residues in the middle of its chemokine domain (Fig. 1), while the COOH-terminal cationic domains are likely to be involved in the antimicrobial activity of chemokines (16, 22). Indeed, SR-PSOX/CXCL16 and CCL27 do not have such COOH-terminal cationic domains. Taken together, these findings suggest that the bacterial binding activity and antimicrobial activity are not the same. The COOH-terminal cationic domain of antimicrobial chemokines may not be necessary to bind bacteria and Ox-LDL but may play an important role in the actual killing of bacteria (16, 22).

The potential dual functions of chemokines may be similar to those of β-defensin and galecston-3, which do not have the conserved chemokine motif in the primary amino acid sequence and therefore do not belong to the chemokine superfamily. β-Defensin was reported to contribute to host defense not only by disrupting the cytoplasmic membrane of microorganisms but also by recruiting dendritic cells and T cells to the site of microbial invasion through interaction with a chemokine receptor CCRL6 (23). Human galectin-3 was also reported to have not only activity to bind OxLDL and LPS (24, 25) but also chemotactic and scavenger receptor-like activity. In Drosophila, non-migrating fat body cells produce antimicrobial peptides (27). With the appearance of professional immune cells in higher vertebrates, it would be necessary to recruit these immune cells to the infection site. Antimicrobial molecules expressed at the infection site may have developed to be used to induce the migration and recruitment of immune cells to protect against invading microorganism. Further analysis of these multifunctional molecules should clarify the evolutionary relationships between chemokine, antimicrobial peptides, and scavenger receptor molecules.

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