Interleukin-13 (IL-13) is a cytokine secreted by activated T lymphocytes that shares many, but not all, biological activities with IL-4. These overlapping activities are probably due to the existence of common receptor components. Two proteins have been described as constituents of the IL-4 receptor, a 140-kDa glycoprotein (IL-4R) and the γc chain (γc) of the IL-2 receptor, but neither of these proteins binds IL-13. We have cloned a cDNA encoding an IL-13 binding protein (IL-13R) from the Caki-1 human renal carcinoma cell line. The cloned cDNA encodes a 380-amino acid protein with two consensus patterns characteristic of the hematopoietic cytokine receptor family and a short cytoplasmic tail. The IL-13R shows homology with the IL-5 receptor, and to a lesser extent, with the prolactin receptor. COS-7 cells transfected with the IL-13R cDNA bind IL-13 with high affinity but do not bind IL-4. COS-7 cells co-transfected with the cloned IL-13R cDNA and IL-4R cDNA resulted in the reconstitution of a small number of receptors that recognized both IL-4 and IL-13. Reverse transcription-polymerase chain reaction analysis detected the receptor transcript only in cell lines known to bind IL-13.

IL-13 is a cytokine secreted by activated T lymphocytes that regulates inflammatory and immune responses (1, 2). It shares several biological activities with IL-4, another T-cell-derived cytokine, in a variety of cell types such as B cells, monocytes, fibroblasts, and endothelial cells (for review, see Ref. 3). But contrary to IL-4, IL-13 does not regulate T-lymphocyte function (4). Recent studies have shown that IL-13 competitively inhibits binding of the IL-4 receptor antibodies X2/45 and CDW124 and the IL-4 antagonist hIL-4 Y124D were a generous gift of Dr. Sebald (Würzburg, Germany). Caki-1 cells (ATCC HTB 46) were cultured in RPMI 1640 medium, 20% fetal calf serum, 2 mM glutamine, and penicillin/streptomycin (100 units/ml), at 37°C in a humidified atmosphere containing 5% CO₂.

**Materials and Methods**

**Growth Factors, Antibodies, and Cells—**Recombinant IL-13 was produced and purified in our laboratory as described previously (2). Human IL-6 was obtained from Tebu (Le Perray en Yvelines, France). The anti-IL-4 receptor antibodies X2/45 and CDW124 and the IL-4 antagonist hIL-4 Y124D were a generous gift of Dr. Sebald (Würzburg, Germany). Caki-1 cells (ATCC HTB 46) were cultured in RPMI 1640 medium, 20% fetal calf serum, 2 mM glutamine, and penicillin/streptomycin (100 units/ml), at 37°C in a humidified atmosphere containing 5% CO₂.

**Binding and Biological Activity Assays—**Binding and cross-linking experiments were performed as described with [³⁵S] labeled [Phe⁴³] IL-13-Gly-Tyr-Gly-Tyr (6) or [¹²⁵I]-IL-4. For the induction of IL-6 secretion, Caki-1 cells were plated in 24-well plates at a density of 5 × 10⁴ cells/well, and after 3 days of culture, confluent monolayers were washed three times with Dulbecco’s modified Eagle’s medium without fetal calf serum. Stimulation of Caki-1 cells was performed with 30 ng/ml of IL-4 or IL-13 in the absence or in the presence of hIL-4 Y124D or an anti-goat140 monoclonal antibody. The level of IL-6 released into the culture media after 24 h incubation was measured by enzyme-linked immunosorbent assay (ECL, Lintes, Besançon, France).

**cDNA Library Constructions, Isolation of cDNAs, and Sequence Analysis—**Total RNA was extracted from Caki-1 cells or tissues as described (2). Poly(A) RNA was isolated from total RNA with oligo(dT)₅₂ magnetic beads (Dynal, Inc.). The Caki-1 size fractionated cDNA library containing 2 × 10⁶ d needs was constructed using the primer-adapter procedure (18) and the vector pSE1 (19). The expression cloning strategy used was reported previously (20). The human cDNA sequence for the IL-13R has been submitted to the GenBank (EMBL Data Bank (accession number X93502).

**Cell Transfection and Receptor Characterization—**For the functional characterization of the recombinant receptor, COS-7 cells were transfected in 25-cm² plates using 0.6 µg of different plasmids. After 24 h COS-7 cell monolayers were trypsinized and plated at 8 × 10⁴ cells per well in 12-well plates. Three days later, binding and cross-linking experiments were performed as described with [³⁵S]-labeled [Phe⁴³] IL-13-Gly-Tyr-Gly-Tyr (6) or [¹²⁵I]-IL-4.
experiments using $^{125}$I-IL-13 in the absence (lane a) and in the presence of a 100-fold excess of IL-13 (lane b) or IL-4 (lane c).

Tissue Distribution—cDNAs were generated from RNA samples with reverse transcriptase and a mixture of labeled d-NTP of known specific activity for quantification. Then, for each sample, 10 ng of cDNA was submitted to PCR using a sense primer corresponding to the sequence of 103 bases. A canonical AATAAA polyadenylation signal at the predicted location (Fig. 3a). PCR-amplified products were hybridized with a probe complementary to sequence +445 to +461 of the cDNA.

RESULTS

Characterization of IL-4 and IL-13 Binding and Activity in Caki-1 Cells—The Caki-1 cell line, among several human cell lines tested, expressed the highest number of binding sites for IL-13. Saturation experiments with labeled IL-13 on Caki-1 cells showed the presence of one class of binding sites with a $K_d$ of 446 ± 50 pM and 7.2 × 10^4 receptors/cell (Fig. 1a). In competition experiments, unlabeled IL-13 totally displaced the labeled IL-13 in a dose-dependent manner, whereas IL-4 displaced with high affinity (~10% of the labeled IL-13). Higher concentrations of IL-4 (>100 nM) did not displace the remaining 90% of the IL-13 binding (Fig. 1b). These results are in line with the existence of two sites with similar affinity for IL-13, of which only one (~10% of the total binding) recognizes IL-4. Affinity cross-linking of IL-13 showed a ~70-kDa complex consistent with the complex observed in similar cross-linking experiments with IL-13 on several cell types (6). The labeled IL-13 was completely displaced from the complex by IL-13 but not by IL-4, which is in agreement with the competition experiments (Fig. 1b).

We also analyzed the IL-4- or IL-13-induced IL-6 secretion from Caki-1 cells. Both cytokines induced the secretion of similar levels of IL-6, and this secretion was inhibited by antibodies against the IL-4Rα chain and by the IL-4 antagonist hIL-4.Y124D (Fig. 2), suggesting that only the shared receptors in Caki-1 cells are responsible for the induction of IL-6 secretion. Similar results were observed when the IL-4- and IL-13-induced phosphorylation of the IRS1/4PS was analyzed in the presence or the absence of the anti-IL-4R antibodies and the IL-4 antagonist (results not shown).

Cloning of the IL-13 Binding Protein—We constructed a cDNA library containing 2 × 10^6 recombinant clones from Caki-1 cells. The library was divided into pools of 1000 cDNAs, and plasmid DNA from each pool was introduced into COS-7 cells. Binding of labeled IL-13 to transfected COS-7 cells was used to identify pools of clones encoding an IL-13R. Positive pools were partitioned and rescreened until a single clone was identified that directed synthesis of a cell surface protein capable of binding IL-13. Two independent IL-13 receptor cDNAs of identical sequence were finally isolated. The cDNA is 1299 bases long, excluding the poly(A) tract, and has a short 3′-untranslated region of 103 bases. A canonical AATAAA polyadenylation signal is found at the predicted location (Fig. 3a).

The open reading frame between nucleotides 53 and 1192 defines a polypeptide of 380 amino acids. The sequence codes for a membrane protein with a putative signal peptide of 26 amino acids, a single membrane-spanning domain, and a short cytoplasmic tail. Four sites for potential N-linked glycosylation are located in the extracellular region. Importantly, two consensus patterns considered as signatures of the hematopoietic cytokine receptor family (for review, see Ref. 21) are also found, as are four conserved cysteines in the amino-terminal half of the extracellular domain and the WSXWS motif located in the COOH-terminal region of the extracellular domain (Fig. 3b). Alignment studies reveal homologies with the human IL-5Rα chain (51% similarity and 27% identity; Fig. 3c) and to a lesser extent with the prolactin receptor (not shown).

Distribution of the mRNA for the IL-13—A ~1.4-kilobase IL-13 transcript was detected when the cDNA for the IL-13 was labeled with $^{32}$P and used to probe a Northern blot containing polyadenylated RNA from Caki-1 cells (not shown). Surprisingly, in Caki-1 cells, similar amounts of IL-13R and IL-4R mRNAs are detected by Northern analysis, although a large excess of IL-13 is expressed. This observation suggests a higher translatability of this mRNA versus IL-4R transcripts and may explain the absence of IL-13R mRNA detection in cell
Fig. 3. Nucleotide sequence of the IL-13R cDNA and comparison of the IL-13R and IL-5R protein sequences.

a, nucleotide sequence of the IL-13R cDNA. The human IL-13R cDNA sequence has been submitted to the GenBank/EMBL Data Bank (accession number X95302). The amino acids corresponding to the predicted transmembrane domain are underlined. Potential N-glycosylation sites (Asn-X-Ser/Thr) are underscored.

b, schematic representation of the protein. S, predicted signal peptide; C, conserved cysteines in the hematopoietic cytokine receptor family; W, the WSXWS motif; TM, transmembrane domain; CD, cytoplasmic domain. The black circles indicate the potential N-glycosylation sites.

c, amino acid alignment of the IL-13R and IL-5R sequences. Human IL-13 and IL-5 receptor protein sequences are aligned as described (27). Cysteine residues and the WSXWS motif characteristic of this family of receptors are boxed.
Figure 5. Characterization of the recombinant IL-13 receptor. COS-7 cells were transfected with the IL-13 cDNA and assayed for binding of \( ^{125}\text{I}-\text{IL-13} \). Bars, S.D. (n = 3). Inset, Scatchard analysis of the saturation curve, which indicated the presence of ~560,000 sites/cell; \( K_d \sim 250 \text{pm} \).

Lines expressing low number of IL-13 binding sites. Reverse transcription-PCR analysis (Fig. 4) showed that the transcript found in Caki-1 cells is also present, at lower levels, in the keratinocytic A431 cells, premyeloid TF-1 cells, premonocytic U937 cells, and the B-cell line IM9. No transcript was detected in the T-cell line Jurkat. These results are in agreement with our previously reported binding studies (6) and with the known biological targets for IL-13.

Expression and characterization of the IL-13 binding protein—COS-7 cells transfected with the isolated cDNA encoding the IL-13R showed specific binding of labeled IL-13. Scatchard analysis of the saturation curve showed a single component with a \( K_d \) value of 250 ± 30 pm and a maximal binding capacity of 5.6 × 10^6 receptors/cell (Fig. 5). The affinity displayed by the recombinant receptor is in good agreement with the \( K_d \) value of 446 pm for the Caki-1 IL-13 receptor and for that described in several other cells (6).

In competition studies, IL-13 was effective in inhibiting the binding of labeled IL-13 to the dimerized receptor, with an inhibitory affinity constant (\( K_i \)) of 1.5 ± 0.5 nm, whereas IL-4 did not inhibit binding (not shown). Cross-linking experiments with labeled IL-13 showed a radioactive band of ~70 kDa similar to the one obtained with the Caki-1 cells, shown in Fig. 1b, inset. The radioactivity was completely displaced by a 100-fold excess of IL-13 but not by a 100-fold excess of IL-4 (results not shown), in agreement with the binding experiments. Thus, the pharmacology and the electrophoretic mobility of the covalently cross-linked labeled receptor are those expected from the characterization of the IL-13R present in Caki-1 cells.

**DISCUSSION**

Compelling evidence supports the idea that IL-13 and IL-4 share receptor components as well as signal transduction elements (5, 6, 9–13). However, neither of the two proteins described as components of the high affinity IL-4 receptor, the IL-4R and the \( \gamma_c \), is responsible for the binding cross-competition of IL-4 and IL-13. Thus, another protein(s), part of the IL-4 complex, is clearly necessary for the recognition of IL-13. Here we describe the characterization and cloning of an IL-13R.

We used Caki-1 cells as source of mRNA for the molecular cloning of the IL-13R because our results suggested that these cells expressing low number of IL-13 binding sites. Reverse transcription-PCR analysis (Fig. 4) showed that the transcript found in Caki-1 cells is also present, at lower levels, in the keratinocytic A431 cells, premyeloid TF-1 cells, premonocytic U937 cells, and the B-cell line IM9. No transcript was detected in the T-cell line Jurkat. These results are in agreement with our previously reported binding studies (6) and with the known biological targets for IL-13.

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**TABLE I**

| cDNAs transfected | \( ^{125}\text{I}-\text{IL-13} \) | Bound displaced by |
|-------------------|-----------------|-----------------|
|                  | total bound     | IL-13 | IL-4 |
| Mock              |                 |      |      |
| IL-13R            | 344             | 117  | 116  |
| IL-4R             | 22,895          | 22,612| 55   |
| IL-13R + IL-4R    | 21,119          | 20,942| 1772 |
| IL-13R + IL-4R + \( \gamma_c \) | 7769 | 7481 | 389  |
| IL-13R + \( \gamma_c \) | 8402 | 8130 | 484  |
| IL-4R + \( \gamma_c \) | 351  | 107  | 109  |

*Each value is the average of triplicates, the S.D. values of which were less than 10%. The results shown are representative of three independent experiments.

Co-expression of IL-13R and IL-4R in COS Cells—We investigated whether the IL-13R and IL-4R would interact in the cell membrane to reconstitute a receptor that allowed cross-competition of both cytokines. The results of the co-expression experiments are shown in Tables I and II. Table I shows the binding of radiolabeled IL-13 to COS cells transfected with cDNAs coding for IL-13R, IL-4R, and \( \gamma_c \) alone and in various combinations. Mock transfected cells displayed low IL-13 binding that is displaced by both IL-13 and IL-4, as described previously (6). Cells transfected with the cDNA for IL-13R showed high IL-13 binding that was completely displaced by IL-13 but not by IL-4; and the expression of IL-4R resulted in IL-13 binding values similar to those observed in mock transfected cells. Thus, IL-13R recognizes IL-13 but not IL-4, and IL-4R does not bind IL-13, as described previously (5, 6). COS cells expressing both IL-13R and IL-4R showed similar binding for IL-13 to that of the cells expressing IL-13R alone; however, about 8–10% of that binding can be displaced by IL-4, suggesting the reconstitution of a binding site shared by both IL-13 and IL-4. The co-expression of \( \gamma_c \) did not improve the reconstitution of shared sites, but resulted in a diminution of IL-13 binding. Similarly, the expression of the \( \gamma_c \) with the IL-13R also resulted in a diminution of IL-13 binding. Work is in progress to investigate an eventual interaction of \( \gamma_c \) and IL-13R that may explain these observations. The co-expression of IL-4R and \( \gamma_c \) did not result in binding sites recognized by IL-13, as described previously (6).

Cells from the same transfection experiments used for binding of labeled IL-13 were also analyzed for binding of labeled IL-4. The binding results with labeled IL-4 (Table II) showed that IL-13 cannot displace labeled IL-4 from the IL-4R alone or from the IL-4R–\( \gamma_c \) complex. But when the IL-4R and the IL-13R were co-expressed, the IL-13 was able to displace a fraction of the labeled IL-4 binding, suggesting again an interaction of both chains to reconstitute shared binding sites for both cytokines.

**TABLE II**

| cDNAs transfected | \( ^{125}\text{I}-\text{IL-4} \) | Bound displaced by |
|-------------------|-----------------|-----------------|
|                  | total bound     | IL-13 | IL-4 |
| Mock              |                 |      |      |
| IL-13R            | 105             |      |      |
| IL-4R             | 60000           | 2000 | 250  |
| IL-13R + IL-4R    | 20000           | 250  | 461  |
| IL-13R + IL-4R + \( \gamma_c \) | 18000 | 2500 | 461  |
| IL-13R + \( \gamma_c \) | 16000 | 2000 | 461  |
| IL-4R + \( \gamma_c \) | 14000 | 2000 | 461  |

*Each value is the average of triplicates, the S.D. values of which were less than 10%. The results shown are representative of three independent experiments.
cells express an IL-4/IL-13 receptor complex responsible for the binding cross-competition and biological activities of IL-4 and IL-13 similar to that previously described in other cells and, in addition, that these cells overexpress the IL-13R component that binds IL-13.

The cloned cDNA for the IL-13R codes for a protein of 380 amino acids with characteristics of the hematopoietic cytokine receptor family; and the pattern of IL-13 mRNA expression is consistent with the receptor distribution estimated by IL-13 binding and with the known IL-13 target cells.

The new receptor has a short cytoplasmic sequence, which is in line with the observation that probably only the receptor complex that is shared by IL-4 and IL-13 transduces a signal to the cells. However, further work is needed to address this issue, whether this cytoplasmic domain plays a role in association with other chains and contributes to signal transduction as described for the IL-5Rα (22).

The homology of the IL-13R with the IL-5Rα chain is interesting because the latter binds IL-5 but needs another protein, the β chain shared with the IL-3 and GM-CSF (granulocyte macrophage colony-stimulating factor) receptors, to form a high affinity receptor complex that is capable of signal transduction (23). However, it should be noted that the IL-13R, when expressed in COS-7 cells, does not need a second chain to reconstitute a high affinity binding site. It therefore resembles the recently described IL-15 binding protein, which also displays high affinity binding for IL-15 in the absence of the other components of the IL-15 receptor complex (24).

The recombinant receptor recognized IL-13 with high affinity, but was not able to bind IL-4, as expected from the results obtained in Caki-1 cells. The radiolabeled complex observed in the cross-linking experiments with labeled IL-13 has the same mobility as that observed in other cell lines (6). It most probably corresponds to the 60–70-kDa band observed in cross-linking experiments performed with labeled IL-4 (6, 15) in addition to the 140-kDa IL-4R complex, arguing in favor of the interaction of both proteins in the functional receptor complex.

The results of the co-expression of the IL-13R and the IL-4R in COS-7 cells suggest that these proteins can interact with each other in the cell membrane to reconstitute a receptor where IL-13 and IL-4 can compete with each other. However, it should be noted that only a fraction of the expressed IL-4R and IL-13R resulted in shared sites for both cytokines. Several reasons may explain the low yield of reconstituted shared sites. For example, the existence of another protein(s), present in limiting amounts in COS cells, necessary to reconstitute the IL-4/IL-13 receptor complex. Another explanation could be an incorrect stoichiometry of IL-4R and IL-13R in the cell membrane, but this is unlikely because co-transfections producing different relative amounts of the IL-4R and IL-13R did not show major differences in the number of reconstituted receptors. Finally, our results do not exclude the existence of another IL-13R with a better capacity to interact with the IL-4R for the reconstitution of shared binding sites for IL-4 and IL-13. If this is the case, the IL-13R described here could either play a regulatory role in the IL-4/IL-13 receptor complex, or require a protein, different from the known IL-4R, to reconstitute a functionally independent IL-13 receptor complex.

The results obtained in the co-transfection experiments with the γc demonstrate, as previously suggested (5, 25), that this protein is not the limiting factor for the reconstitution of an IL-4R/IL-13R complex, a conclusion also supported by the absence of detectable γc mRNA in Caki-1 cells (7). It should be noted that the expression of the γc improved IL-4 binding as described previously (16), but decreased the binding of IL-13, suggesting complex interaction between the different receptor components. Work is in progress to address this problem.

The cloning of the IL-13R described here should help to address the question of the regulation of the expression of the receptor under normal and pathological conditions, such as allergy, where IL-13 may play a key role. Furthermore, the availability of the cDNA will facilitate the cloning of other proteins necessary for the reconstitution of the IL-4/IL-13 receptor complex or of an eventual independent IL-13 receptor complex and may help the rational design of new drugs that specifically antagonize IL-4 or IL-13 activities.

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