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Regulation of Cell Type-Specific Mouse FcεRI β-Chain Gene Expression by GATA-1 Via Four GATA Motifs in the Promoter

Keiko Maeda,* Chiharu Nishiyama,2* Tomoko Tokura,* Yushiro Akizawa,* Makoto Nishiyama,† Hideoki Ogawa,* Ko Okumura,* and Chisei Ra†

The FcεRI, a subunit of two related multisubunit receptor complexes, the FcεRI and FcγRIII, amplifies the mast cell response and is necessary for the cell surface expression of FcεRI in mouse. The transient reporter assay indicated that −69/+4 region is required for cell type-specific transcriptional regulation of mouse β-chain gene. EMSA using Abs against transcription factors or competitive oligonucleotides demonstrated that −58/−40 region (containing overlapping three GATA-1 sites, −53/−48, −46/−51, and −42/−47) and −31/−26 region (containing one GATA-1 site) are recognized by GATA-1. The promoter activity of β-chain was decreased by nucleotide replacements of the GATA-1 sites in mouse mast cell line PT18. Furthermore, exogenously produced GATA-1 up-regulated the promoter activity in CV-1 cells, which are negative in the β-chain production and the up-regulation was apparently suppressed by GATA-1 site mutations. These results indicate that cell type-specific transcription of mouse β-chain gene is regulated by GATA-1. The Journal of Immunology, 2003, 170: 334–340.

In this report, we describe the analysis of transcriptional regulatory elements for the mouse FcεRI β-chain gene expression by luciferase reporter assay and EMSA, and demonstrate that GATA-1 transactivates β-chain gene expression by recognizing repeating GATA-1 motifs present in the promoter in a cell type-specific manner.

Materials and Methods

Cell culture

PT18 (mouse mast cell line), A20.2/J (mouse B lymphoma cell line), RAW264.7 (mouse mono-macrophage cell line), and L5178Y (mouse lymphoma cell line) cells were cultured in RPMI 1640 medium (Sigma-Aldrich, St. Louis, MO) containing 10% FCS (Biological Industries, Haemek, Israel), 10 μM nonessential amino acid (Invitrogen, Leek, The Netherlands), 100 U/ml penicillin, and 100 μg/ml streptomycin. CV-1 (simian kidney cell line) cells were purchased from RIKEN Cell Bank (Tsukuba, Japan) and were cultured in DMEM medium (Sigma-Aldrich) containing 10% FCS, nonessential amino acids, penicillin, and streptomycin.

Plasmid construction

The mouse genomic DNA of FcεRI β-chain was prepared from mouse genomic library and the transcription start site was determined by 5′-RACE analysis (S. Hiraoka, M. Watanabe, Y. Takagaki, K. Fujita-Suzuki, K. Okumura, and C. Ra, manuscript in preparation; accession no. AB033617). The 2.4 kb of 5′-flanking region was subcloned into BglII/Ncol site of reporter plasmid pG3L-Basic (Promega, Madison, WI) and the resulting plasmid was named −2.4k/pG3L-Basic. The plasmids containing a variety of 5′-deletion were constructed by an endonuclease/exonuclease III deletion kit (TAKARA BIO, Otsu, Japan).

The plasmids in which several bases were replaced were constructed by site-directed mutagenesis using a Quick Change site-directed mutagenesis kit (Stratagene, La Jolla, CA). All mutations were verified by sequencing analysis.

The expression plasmid pCR-GATA-1 generated in our previous study (9, 10) was used to produce GATA-1.

Transfection and luciferase assay

Cells of 5 × 10⁶ (PT18, A20.2/J, RAW 264.7, and L5178Y) were transfected with 5 μg reporter plasmid and 25 ng pRL-CMV (Promega) by electroporation using Gene Pulsar II (Bio-Rad, Hercules, CA) set at 700 V and 950 μF. CV-1 cells of 1 × 10⁶ were transfected with 500 ng reporter plasmid, 0.5 ng pRL-null (Promega), and 100 ng of pCR-GATA-1 expression plasmid or pCR3.1 empty plasmid (9, 10) by FuGENE 6 (Roche Diagnostics, Indianapolis, IN) according to the manufacturer’s instruction.

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FIGURE 1. Cell type specificity of mouse FcεRI β-chain promoter. Reporter plasmid, −2.4k/pGL3-Basic, containing 2.4 kb 5′-upstream from translation initiation codon of mouse β-chain gene or pGL3-Basic was transfected into mouse cells with internal control plasmid, pRL-CMV. The relative luciferase activity of −2.4k/pGL3-Basic is represented as the ratio to the activity driven by pGL3-Basic. Each experiment was conducted in duplicate for each sample, and the results are expressed as mean ± SE for more than three independent experiments in Figs. 1, 2, 6, and 7.

The pRL-CMV and the pRL-null plasmids were used as the internal control of transfection efficiency.

After 20–24 h of cultivation, the cells were harvested and treated with a Dual-luciferase assay kit (Promega) for the measurement of luciferase activity. The luminescence was measured with Micro Lumat Plus (Berthold, Postfach, Germany).

EMSA
Probes for EMSA were prepared by annealing FITC-labeled synthesized oligonucleotides. Each nucleotide sequence of sense strand was shown as follows: probe-1, 5′-GGAGTCACCTGATATCAGGGGAC-3′, and probe-2, 5′-CTGGAGACCTTCTGAGCAAGTATGGTCTCCCATGGAAGATAATC-3′ (In vitrogen). The nonlabeled competitor oligonucleotides were prepared by a similar way. The sequences of mutant competitors were shown in Fig. 3A. The sequences of sense strand of consensus oligonucleotides were as follows: GATA, 5′-CATTGATAACAGAAAGTGATAACTCT-3′, and AP-1, 5′-CGCTTGACTCAGCCCGAAA-3′.

Nuclear extract from PT18 cells were prepared by using the method described in our previous report (9, 10). Briefly, PT18 cells were washed with PBS and incubated in ice-cold buffer A containing 0.4% Nonidet P-40 for 1 min. After centrifugation at 3,000 × g for 1 min, the pellet was resuspended in extraction buffer including 1/10 volume of 3 M (NH₄)₂SO₄ and incubated on ice for 30 min. After centrifugation at 6,000 × g for 15 min, equal volumes of 3 M (NH₄)₂SO₄ were added to the supernatants to precipitate the nuclear protein. The pellets obtained by centrifugation at 10,000 × g for 15 min were resuspended with appropriate volume of extract buffer, and protein concentrations of the nuclear extract were determined with a BCA Protein Assay Reagent kit (Pierce, Rockford, IL).

Nuclear extract of 5 µg was incubated with 5 pmol FITC-labeled probe for 20 min in the reaction buffer containing 500 ng poly(dI-dC), 1 mM MgCl₂, 30 mM KCl, 10 mM HEPES (pH 7.9), 1 mM DTT, and 5% glycerol. The protein–DNA complexes were separated on a 4% polyacrylamide gel in 0.5 × TBE buffer (45 mM Tris-HCl, 32.3 mM boric acid, and 1.25 mM EDTA). For competition experiments, 50-fold molar excess of unlabelled competitor oligonucleotide were added to the binding reaction mixture. For inhibition experiments by Abs, 1 µg of Abs against GATA-1, GATA-2, GATA-3, and c-Jun purchased from Santa Cruz Biotechnology (Santa Cruz, CA) were used. All gels were subjected to fluorescence detection, Fluorimager (Amersham Pharmacia Biotech, Uppsala, Sweden).

For in vitro transcription and translation, pCR-GATA-1 was used for the reaction using a TNT T7 Quick coupled transcription/translation system (Promega).

RT-PCR analysis
Detection of the mRNA for GATA-1 or G3PDH was performed by RT-PCR using 1 µg of total RNA prepared from each cell line using Trizol Reagent (Invitrogen) as a template. For detection of GATA-1, the following primer set was used: 5′-ATGGATTTTCCTGGTCTAGGGGC-3′ and 5′-TCAGAAACTGAGTGGGGCGATCACG-3′, and a primer set to detect G3PDH was purchased from Clontech Laboratories (Palo Alto, CA).

Western blot analysis
Five micrograms of nuclear extract prepared from each cell line was subjected to Western blotting analysis. Anti-GATA-1 Ab, the same as that for EMSA analysis, was used as the primary Ab to detect GATA-1 protein. Anti-YY1 Ab, control rat IgsG1 (control for anti-GATA-1 Ab), and control mouse IgG3 (control for anti-YY1 Ab) were purchased from Santa Cruz Biotechnology and were used as the primary Abs. Peroxidase-conjugated anti-rat IgG goat Abs (Wako Pure Chemical Industries, Osaka, Japan), or anti-mouse IgG sheep Abs (Santa Cruz Biotechnology) were used as the detector, Fluorimager (Amersham Pharmacia Biotech, Uppsala, Sweden).

FIGURE 2. Mapping of cell type-specific cis-enhancer elements in β-chain promoter. A. 5′-deleted constructs of β-chain promoter were introduced into PT18 by electroporation. The relative luciferase activity is represented as the ratio to the activity driven by pGL3-Basic. Relative length of upstream region in each construct is denoted by each solid line. Nucleotide numbers when the transcription start site is expressed as +1 are also attached. B. 5′-deleted constructs were introduced into PT18, A20.2J, L5178Y, and RAW264.7.

RT-PCR analysis
Detection of the mRNA for GATA-1 or G3PDH was performed by RT-PCR using 1 µg of total RNA prepared from each cell line using Trizol Reagent (Invitrogen) as a template. For detection of GATA-1, the following primer set was used: 5′-ATGGATTTTCCTGGTCTAGGGGC-3′ and 5′-TCAGAAACTGAGTGGGGCGATCACG-3′, and a primer set to detect G3PDH was purchased from Clontech Laboratories (Palo Alto, CA).

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secondary Abs. Membrane was soaked with the ECL+ plus Western blotting detection reagent (Amer sham Pharmacia Biotech), and its chemiluminescence was detected by LAS-1000 plus (Fuji Film, Tokyo, Japan).

Results
The β-chain promoter is activated in mast cell line
To examine the cell type specificity of the mouse FcεRI β-chain promoter, we generated a reporter plasmid, −2.4k/pGL3-Basic, by introducing ∼2.4 kb 5′-upstream region of the putative translational initiation codon of mouse β-chain gene (−2357/+103) at just upstream of the luciferase gene of promoterless plasmid pGL3-Basic. The luciferase activity in the PT18 cells which were transiently transfected with −2.4k/pGL3-Basic was ∼10-fold higher than that from PT18 cells transfected with pGL3-Basic, whereas no difference was observed between the luciferase activities directed by −2.4k/pGL3-Basic and that of pGL3-Basic when the reporter plasmids were introduced into FcεRI β-chain negative cell lines A20.2J, L5178Y, or RAW264.7 (Fig. 1). This result suggests that the −2357/+103 region principally contains all the elements necessary for the expression and regulation of the promoter of β-chain which is active only in β-chain positive cells.

To screen cis-acting elements within the mouse β-chain promoter region, several 5′-deletion constructs each carrying a portion of 5′-flanking region from −2357 to +103 were generated and introduced into PT18 cells (Fig. 2A). The deletion of −2357/+103 region had no apparent effect on the promoter activity. In contrast, the promoter activity was slightly decreased by deletion of −116/−70, and markedly decreased by further deletion (see −69/−33 and −32/+4). The luciferase activity directed by +3/pGL3-Basic was reduced to the level which was equivalent to that of pGL3-Basic. These results indicate that elements essential for the activation of mouse β-chain promoter are present between −69 and +4.

To further substantiate the finding that the enhancer elements necessary for the activation of β-chain promoter are functional in PT18 but not A20.2J, L5178Y, or RAW264.7, the deletion constructs containing −69/+103, −32/+103, or +3/+103 of β-chain gene were introduced into each cell. As expected, all the constructs gave the luciferase activity almost equal to that given by the empty plasmid in A20.2J, L5178Y, and RAW264.7 (Figs. 1 and 2B). In contrast, the distinct activity was observed when pGL3-Basic containing the β-chain promoter region −69/+103 or −32/+103 was introduced into PT18 cells. We also found that the promoter activity directed by −61/+4 was almost the same as that of −69/+4 in our similar reporter assay (data not shown). From all these data, we assume that −61/+4 contains elements essential for the activation of mouse β-chain promoter, which is activated in specific cells.

GATA-1 binds to both of the regions −58/−38 and −31/−26
To identify the transcription factors binding the −61/+4 region, EMSA was performed by using PT18 nuclear extract and FITC-labeled probes in the presence or absence of the various competitive fragments. The EMSA using probe-1 (−61/−32) gave several shifted bands (Fig. 3B). Among them, the shifted band shown with an arrow was lost by addition of competitors −61, −39, and −37. On the contrary, the shifted band apparently remained even by addition of other competitors −58, −54, −52, −47, and −43 (Fig. 3B, lanes 5–9). This indicated that the transcription factor responsible for this band shift recognized the region −58/−38. Motif analysis using a program TFSEARCH (http://www.cbrc.jp/research/db/TFSEARCH.html) revealed the presence of three overlapping GATA-binding motifs in this region: two TGATAT and one TGATTG (−53/−48, −46/−51, and −42/−47). The shifted band shown with an arrowhead was apparently observed in the presence of competitors −61 and −58, suggesting that the nuclear protein(s) recognizing −61/−55 also presents in nuclear of PT18 cells. Other shifted bands, shown with asterisks in Fig. 3B, lane 2, might be nonspecific because they disappeared equally by addition of any competitors.

To confirm that GATA-1 is responsible for the band shift, similar EMSA was performed in the presence of anti-GATA-1, GATA-2, GATA-3, or c-Jun Abs, or oligonucleotides with GATA-1- or AP-1-recognition sequence. The intensity of the shifted band marked with the arrow was decreased by addition of anti-GATA-1 Ab or oligonucleotides with GATA-1 consensus sequence (Fig. 4A, lanes 4 and 8), whereas Abs against GATA-2, 3, anti-c-Jun, and oligonucleotide competitor with AP-1 consensus sequence had no effect on this band (Fig. 4A, lanes 5, 6, 7, and 9). To confirm further the binding of GATA-1 to this probe, GATA-1 was produced by in vitro transcription/translation system used for EMSA. Although several shifted bands were seen, the shifted band that showed the mobility identical with that seen with the nuclear protein from PT18 was found (Fig. 4B). As expected, the band disappeared by addition of anti-GATA-1 Ab (Fig. 4B). These results indicate that the transcription factor GATA-1 binds probe-1.

By using a different probe, probe-2, similar EMSA was performed (Fig. 5). Probe-2 (−39/+4 fragment) contains a possible GATA motif (AGATAA) at −31/−26. The shifted band indicated by an arrow disappeared by addition of Ab against GATA-1, but not by Abs against GATA-2.

FIGURE 3. Competition assay on −61/−33 region. A. Nucleotide sequences of probe-1 and competitors used in this study. Only the nucleotides that differ from the original were shown in the nucleotide sequences of competitors and lines represent unchanged nucleotides. B. EMSA was performed with probe-1 and nuclear extracts from PT18 in the presence or absence of unlabeled competitors shown in A. The self-competitor had the nucleotide sequence the same as probe-1. Lane 1, probe only; lanes 2–11, probe with nuclear extract; lane 2, without competitor; lane 3, with unlabeled probe; lanes 4–11, with each mutant competitors. Specific band caused by binding between probe-1 and nuclear protein via −58/−38 region was shown with an arrow.
The β-chain promoter is transactivated by GATA-1 via multiple GATA-1 binding sites

To examine the contribution of the putative four GATA1-binding sites to the promoter activity, we measured the luciferase activities directed by the wild-type (−69/+103) and various altered promoters in which nucleotide replacements were introduced at the four putative GATA-binding sites by site-directed mutagenesis (Fig. 6A). The luciferase activity directed by the mutant promoter M4 lacking a GATA-1 site at −31/−26 was apparently lower than that of the wild-type promoter (Fig. 6B). The mutant promoters (M12, M23, and M123) which lacked two or three of GATA-1 sites in the region −53/−42 also gave lower activities (Fig. 6B). When the additional mutation at −31/−26 was introduced into M12 and M23, resulting promoters showed further reduced activity (M124 and M234). The luciferase activity directed by the mutant promoter M1234 lacking all of four GATA-1 motifs was decreased to the level equivalent to that of promoterless plasmid (pGL3-Basic). These results suggest that all four GATA-1-binding motifs in −53/−26 are necessary for full transcriptional activity of β-chain promoter in mouse mast cells.

To confirm the involvement of GATA-1 to the β-chain promoter activity, the reporter plasmids constructed in this study were co-transfected with pCR-GATA-1 expression plasmids or pCR3.1 empty plasmid into CV-1 cells that do not express endogenous GATA-1 (Fig. 7). The β-chain promoter was markedly up-regulated ~50-fold by the cotransfection of GATA-1 expression plasmid when the reporter plasmid containing all the four GATA-1 motifs (−69/+103 region) was used. The up-regulating effect of exogenously expressed GATA-1 was apparently decreased when the fourth GATA-1 motif (M4), or two of the other three GATA-1 motifs (−47/+103, −40/+103, −32/+103, M12, and M23) was mutated. The altered promoters (+3/+103 and M1234) lacking all four GATA-1 gave further decreased level of transactivation even by exogenous GATA-1. These results indicate that GATA-1 actually transactivates the β-chain promoter.

Correlation of cell type-specific expression between β-chain and GATA-1

Above results indicated that GATA-1 transactivates the promoter of β-chain which is expressed in mast cells specifically. To confirm cell type specificity of GATA-1 expression, we performed both RT-PCR and Western blotting analysis. The transcript of GATA-1 was detected in PT18 cells but not in A20.2J, L5178Y, and RAW264.7 (Fig. 8A). Similarly, we detected GATA-1 protein in nuclear extract of PT18 by Western blotting analysis, whereas no specific band corresponding to GATA-1 protein was observed in other cell lines examined (Fig. 8B), which is in contrast with the case of an ubiquitous transcription factor YY1 found in each cell line (Fig. 8C). These results suggest that cell type-specific expression of β-chain is mediated by cell type-specific expression of GATA-1.
Discussion

In this study, we indicated that the mouse β-chain promoter is functional only in PT18 mouse mast cell line, but not in monomacrophage and lymphoma cell lines. By using a series of 5’-deletion promoter constructs, the cell type-specific promoter was assigned within −69/−103, although a slight decrease in the promoter activity was observed by the deletion of −116/−70. This may suggest the presence of additional cis-enhancing elements recognizing the deleted region. Anyway, by EMSA using antitranscription factor Abs and various competitive oligonucleotides, GATA-1 protein was identified to bind the promoter region (−61/+3) of the β-chain where three possible GATA-1-binding (−53/−48, −46/−51, and −42/−47) and a single possible GATA-1-binding site (−31/−26) are present. Furthermore, reporter assay using PT18 cells (β-chain⁺, GATA-1⁺) and coexpression analysis using CV-1 cells (β-chain⁻, GATA-1⁻) indicated that those four GATA-1 sites are required for full transcriptional activation by GATA-1 protein.

GATA-1 is abundantly expressed in mast cells (11) and recognizes mast cell-specific cis-enhancing elements in several genes such as FcεRI α-chain (9, 10), carboxypeptidase A (12), IL-4 (13), and chymase (14). In this study, expression of GATA-1 was observed in PT18 cells (β-chain positive), but not in other cell lines examined (β-chain negative) (Fig. 8). Therefore, GATA-1 is one of the most probable candidates which regulate cell type-specific expression of mouse β-chain gene. When we performed coexpression analysis in the cell lines L5178Y and RAW264.7 (β-chain⁻/GATA-1⁻), exogenously expressed GATA-1 had no positive

FIGURE 6. Involvement of GATA-1-binding sites in the transcriptional activation of β-chain promoter. A. The nucleotide sequences of mutant promoters. Mutations were introduced into −54/−28 region of −69/pGL3-Basic reporter plasmid. Only the nucleotides that differ from the original were shown, and lines represent unchanged nucleotides. GATA-1 recognition sites are shown with arrows. B. The various reporter plasmids shown in A were introduced into PT18 cells. Relative luciferase activities are represented as the ratio to the activity driven by pGL3-Basic.

FIGURE 7. The exogenously produced GATA-1 up-regulated the transcriptional activity of β-chain promoter via four GATA-1 sites. Each reporter plasmid shown in Fig. 6A was transfected into CV-1 cells with either pCR-GATA-1 expression plasmid or pCR3.1 empty plasmid. The ratio of luciferase activity of each constructs in the presence of GATA-1 expression plasmid to that of the empty plasmid was represented as fold activation.
The effect on β-chain promoter activity (data not shown). This may be explained by the possibility that PU.1, which is expressed in limited cells such as lymphoid cells and monocyte/macrophages, may inhibit function of GATA-1 as reported by several studies (15, 16). Actually, we found transcript of PU.1 in all cell lines used in Fig. 1, but not in CV-1 (data not shown). Alternatively, an unknown transcription factor that is involved in the transactivation of β-chain promoter is absent in these two cell lines.

Recently, β-chain gene was found to be expressed in eosinophils in human (17). It contrasts with the case of mouse or rat where the expression of FcεRI or β-chain was not observed in eosinophils, respectively (18, 19). These reports suggest the different mechanisms for cell type-specific expression of β-chain between human and rodent. Consistent with this, our recent study suggested that transcriptional activation of human β-chain promoter was observed in various types of cells and the cis-enhancing elements and transcription factors different from those of mouse identified in this study were involved in the regulation of human β-chain promoter (Y. Akizawa, C. Nishiyama, M. Hasegawa, K. Maeda, T. Nakahata, K. Okumura, H. Ogawa, and C. Ra, manuscript in preparation).

The motif (T/A)GATA(A/G) has been well-known as consensus recognition sequence of GATA-family proteins. However, recently, it was reported that GATA protein binds a variety of motifs with high affinity equivalent to that of conventional GATA motif and activates the transcription activity (20, 21). In this report, we suggest that GATA-1 protein prepared from PT18 cells binds and transactivates the β-chain promoter via motifs TGATA(T/G) (−53/−48), −46/−51) and TGATG (−42/−47) as well as the canonical consensus sequence, AGATAA (−26/−31). A single GATA-1 protein that occupies either of the overlapping GATA-1 motifs may prevent different GATA-1 proteins from binding the other two GATA-1-motifs by possible steric hindrance. However, palindromic or overlapping GATA sites were found in several promoters and were shown to give higher GATA-1-binding affinity than a promoter with a single GATA site (22). Therefore, it may be possible to assume that two of the three, at least, GATA-1 motifs are recognized by two GATA-1 proteins. For elucidation of the mechanism for GATA-1-mediated up-regulation of the promoter via multiple GATA-1 motifs, detailed analysis including the determination of three-dimensional structure of whole GATA-1/DNA complex will be obviously required.

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