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Regulation of YB-1 gene expression by GATA transcription factors

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Abstract

GATA-1 is a transcription factor essential for erythroid cell development, and knockdown of GATA-1 gene results in maturation arrest and transformation of erythroblasts. To clarify the mechanism that gives rise to this abnormal phenotype, genes that are aberrantly expressed in the spleen of heterozygous GATA-1 knockdown mutant mouse (referred to as GATA-1 mutant mouse) were identified by using cDNA array. One of these genes, YB-1, was found to be highly expressed in the spleen of GATA-1 mutant mouse. Reporter and electrophoretic mobility shift assays revealed that the proximal GATA element in 5′-UTR region of YB-1 gene functions positively in K562 cells. Furthermore, both GATA-1 and GATA-2, which were transiently expressed in COS-7 cells, bound to this element and activated the YB-1 promoter through this element. These results suggest that YB-1 functions under the regulation of GATA factors in erythroid differentiation and aberrant expression of YB-1 gene may result in dyserythropoiesis.

Keywords: Erythropoiesis; YB-1 gene; GATA factors

Hematopoiesis is maintained under a proper balance between self-renewal and differentiation of hematopoietic stem cells. Although it remains unclear how the commitment to the specific lineage is induced during the differentiation process, it is known that transcription factors, which control the expression of lineage-specific genes, play key roles [1]. GATA transcription factor family consists of lineage-specific transcription factors with a unique protein structure, Zn finger motif, as a DNA binding domain that recognizes (A/T)GATA(A/G) sequence [1,2]. Among these GATA factors, GATA-1 is the first member identified and has been confirmed to be essential for erythropoiesis [3]. Interestingly, erythroid cells derived from GATA-1 knockdown ES cells showed maturation arrest at proerythroblast stage and transformed both in vivo and in vitro [4,5]. These observations suggest that not only maturation arrest but also transformation is led by single GATA-1 mutation. The first step to clarify the molecular mechanism of this phenomenon is to identify the genes that are aberrantly regulated by GATA-1 deficiency.

For this purpose, we screened for genes that are differentially expressed between wild type and heterozygous GATA-1 knockdown mutant (termed GATA-1 mutant) mouse spleen, and found that YB-1 gene is strongly expressed in GATA-1 mutant mouse spleen. YB-1 has been shown to be a multifunctional gene functioning on the transcriptional or post-transcriptional gene expression [6–14], but its role and the regulation of the gene expression in hematopoiesis have not been reported to date. In this study, the regulation of YB-1 gene expression, especially by GATA factors, in erythroid cells was examined.

Experimental procedures

Cell culture. K562 and COS-7 cells were maintained in RPMI1640 and DMEM, respectively. Each medium was supplemented with 10% fetal bovine serum.

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Northern blot analysis, RT-PCR, and cDNA array hybridization. For RT-PCR, total RNA was extracted from mouse spleen with ISOGEN-LS (Nippongene, Tokyo, Japan) according to the protocol recommended by the manufacturer. Extracted RNA was reverse-transcribed and amplified by PCR as described previously [15]. Quantitative PCR was performed using QuantiTect SYBR Green PCR kit (Qiagen, GmbH, Germany) according to the manufacturer's protocol. Primers used for PCR are as follows. mouse GATA-1 forward: 5′-ACTC GTCATACCACTAAGGT-3′, reverse: 5′-AGTGTCTGTAGGCCTC AGCT-3′, mouse GATA-2 forward: 5′-TGCAACACACCACCCGAT ACC-3′, reverse: 5′-CAATT TGGACAACAGGTGCCC-3′, β-actin forward: 5′-GT GACGAGGCCCAGAGCAAG-3′, reverse: 5′-AGGG GCCGGACTCATCGTAC-3′, human GATA-1 forward: 5′-TTTCCA GTACCTTCTTTTCTCCC-3′, reverse: 5′-ACAGTGGAGTGGCTGT TGCTC-3′, human GA TA-2 forward: 5′-ATCAAGCCCAAGCGAA GACT-3′, reverse: 5′-CATGGTCAGTGGCCTGTTAAC-3′, and GAPDH forward: 5′ACCACAGTCCATGCCATCA3-3′, reverse: 5′-TCCACCACCCTGTTGCTGTA-3′. For Northern blot analysis, total RNA (10–20 μg/lane) was electrophoresed on a 1% formaldehyde–agarose gel in MOPS buffer and transferred to Hybond-N^+ nylon membranes (Amersham Pharmacia Biotech, Piscataway, NJ). cDNA labeling, hybridization, washing, and detection of signals were carried out by using Gene Images AlkPhos Direct labeling kit and AlkPhos Direct detection system (Amersham Pharmacia Biotech) under the instructions provided by the manufacturer. Mouse YB-1 cDNA, an 879-bp fragment corresponding to nt 26–904, was obtained by PCR. cDNA array hybridization was performed using the procedure recommended by the supplier (Atlas cDNA expression arrays, Clontech, Palo Alto, CA) using mRNA extracted from spleen of wild-type and GATA-1 mutant mouse [5].

Promoter activity assays. Series of DNA fragments of the human YB-1 gene promoter region were obtained from human genomic DNA of peripheral blood mononuclear cells (PBMC) by PCR using the following forward primers: 5′-GAATGGTACCGTTTGCTA CCTCTGCTCC-3′ (spanning positions −1856 to −1839), 5′-ACAGG GTACCGATCAGATAGATGTT-3′ (−1663 to −1646), 5′-TTCTGG TACCAACTTTCTGGCTGTTGTG-3′ (−1640 to −1623), 5′-TAGT GGTACCTCTATCACGTGGCTG TTG-3′ (−660 to −642), 5′-TCA CGGTACCGTTGCAGGAATAAAGTGA-3′ (−646 to −629), and the common reverse primer, 5′-ACTACTCGAGCTACCGATCGAA CTAGCG-3′ (+189 to +206). 5′UTR deletion DNA fragments were also obtained by PCR using the common forward primer 5′-TCA CGGTACCGTTGCAGGAATAAAGTGA-3′ (−646 to −629) and the following reverse primers: 5′-ATCACTCGAGCATTCTCATT GGACCTTT-3′ (+38 to +55), 5′-TATTCTCGAGTATTTTATCAG TCCTCCA-3′ (+54 to +71), 5′-GCGACTCGAGCTAGGCGTTG TTCACTGG-3′ (+128 to +145), and 5′-ATGGCTCGAGAGGC GGGATAAGCCCTAC-3′ (+158 to +175). All forward primers contain an artificial *Kpn*I recognition sequence, whereas all reverse primers contain an artificial *Xho*I recognition sequence. PBMC were collected from healthy volunteers after obtaining informed consent. The amplified DNA fragments were digested with *Kpn*I and *Xho*I, and inserted into the *Kpn*I/*Xho*I site of the pGL3-Basic plasmid (Promega, Madison, WI). These constructed plasmids were termed −1856PGL, −1663PGL, −1640PGL, −660PGL, −646PGL, −646/+55 PGL, −646/+71PGL, −646/+145PGL, and −646/+175PGL, respectively (Fig. 2). +166GATAmut was created with QuickChange Site-Directed Mutagenesis Kit (Stratagene, La Jolla, CA) using the mutagenesis primer, 5′-TCGCTCGTAGGGCTTAAACCGCCTGTC CCGCCAT-3′. Nucleotide sequences of amplified fragments were verified by sequencing. A human GATA-1 expression plasmid, pEF-GATA-1, and a human GATA-2 expression plasmid, pEF-GATA-2, were kindly provided by Dr. N. Minegishi, University of Tsukuba.

Transient promoter activity assays were performed using these plasmids using the procedure provided by the supplier (FuGENE6, Boehinger–Mannheim, Germany). Briefly, for K562 cells, cells (5×10^5 cells/well) in 12-well plate were transfected with 3 μg FuGENE6 with 1 μg of each YB-1 promoter construct and 0.05 μg pRL-SV40 plasmid (Promega). After 24 h, the cells were collected and lysed in 500 μl lysis buffer (Promega). The luciferase activity of 10 μl of each lysate was determined by Dual-Luciferase Reporter Assay System (Promega). For COS-7 cells, cells (1×10^5 cells/well) in 12-well plate were incubated for 16 h. After incubation, cells were transfected with 0.5 μg of each YB-1 promoter construct, 0.01 μg pRL-SV40 plasmid or pRL-TK plasmid (Promega), combined with GATA-1 or GATA-2 expression plasmid. Total amount of transfected plasmid was equalized by the addition of pBluescript plasmid DNA (Stratagene). Experiments were carried out three times independently, and averages and standard deviations (SD) were calculated.

Electrophoretic mobility shift assay. EMSA analysis was performed as described previously [16]. Briefly 2–4 μg of nuclear extracts of K562 or COS-7 was incubated with the ^{32}P-labeled oligomer, 5′-CGCT CGTAGGGCTTATCCCGCCTGTCCCG-3′, corresponding to nucleotides +152 to +181 of the human YB-1 promoter in a reaction mixture for 30 min on ice. In competition assays, 300–200 molar excess of the following unlabeled oligomers: +166GATAwt, 5′-TCGCTC GTAGGGCTTATCCCGCCTGTCCCG-3′, +166GATAmut, 5′-TCG CTCGTAGGGCTTAAACCGCCTGTCCCGCCAT-3′, and GATA MαP (mouse α1-globin GATA binding sequence) [17] 5′-GATC TCCGGCAACTGATAAGGATTCCCTG-3′ were added prior to the addition of the probe to the mixture. For the inhibition assay with antibodies, an aliquot of nuclear extracts was incubated on ice with 2–4 μg mouse IgG against human GATA-1 (M-20) (Santa Cruz Biotechnology, Santa Cruz, CA), goat IgG against human GATA-2 (CG2-96) (Santa Cruz Biotechnology), and control murine or goat IgG for 1 h before the addition of the probe. Electrophoresis was performed on 4% acrylamide gel and then gels were dried and exposed to X-ray film.

Results

YB-1 mRNA is abundantly expressed in GATA-1 mutant mouse spleen

First, we identified the genes that were expressed differentially between GATA-1 mutant and wild type mouse spleen, using cDNA array. Fig. 1A shows mRNA expression profiles of each spleen. Arrows show the dots of mouse YB-1 gene and YB-1 mRNA that was expressed more strongly in mutant mouse than wild type. This result was confirmed by Northern blot analysis (Fig. 1B). Besides YB-1, several genes were found to be expressed highly in mutant mouse spleen by cDNA array, but the difference of expression level of these genes between wild-type and mutant mouse spleen was not confirmed by Northern blot analysis.

In addition, the expression of GATA-1 mRNA was decreased in GATA-1 mutant mouse spleen, whereas the expression of GATA-2 mRNA was increased in GATA-1 mutant mouse spleen (Fig. 1C). These results are consistent with those in previous reports which showed complemented upregulation of GATA-2 expression along with downregulation of GATA-1 gene in GATA-1 mutant erythroblasts.

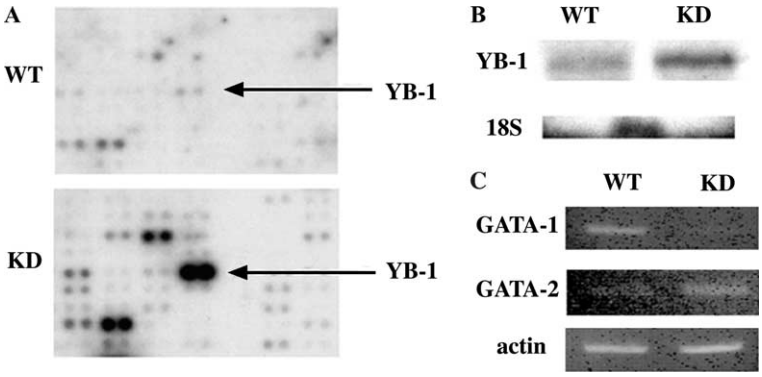

Fig. 1. Expression of YB-1, GATA-1, and GATA-2 mRNA in wild type and GATA-1 mutant mouse spleen. (A) The gene expression profile of wild-type and GATA-1 mutant mouse spleen was examined by using cDNAarray. Arrows indicate spots of YB-1 mRNA. (B) Expression of YB-1 mRNA in the wild-type and GATA-1 mutant mouse spleen examined by Northern blot analysis. (C) The expression of mRNA expression of GATA-1, GATA-2, and YB-1 in the wild-type and GATA-1 mutant mouse spleen was examined by RT-PCR. WT and KD indicate wild-type and GATA-1 knockdown mutant mouse, respectively.

A 5′-UTR GATA element is critical for the human YB-1 promoter activity in K562 cells

Then, in order to determine whether GATA factors directly regulate YB-1 gene expression in erythroid cells, we cloned the regulatory region of YB-1 gene by PCR and performed functional analysis. Sequencing analysis identified two GATA consensus sequences in the promoter region and one in 5′-UTR region. To clarify whether these cis-GATA elements alter the promoter activity, transient transfection assays were performed in K562 cells using a series of GATA deletion constructs

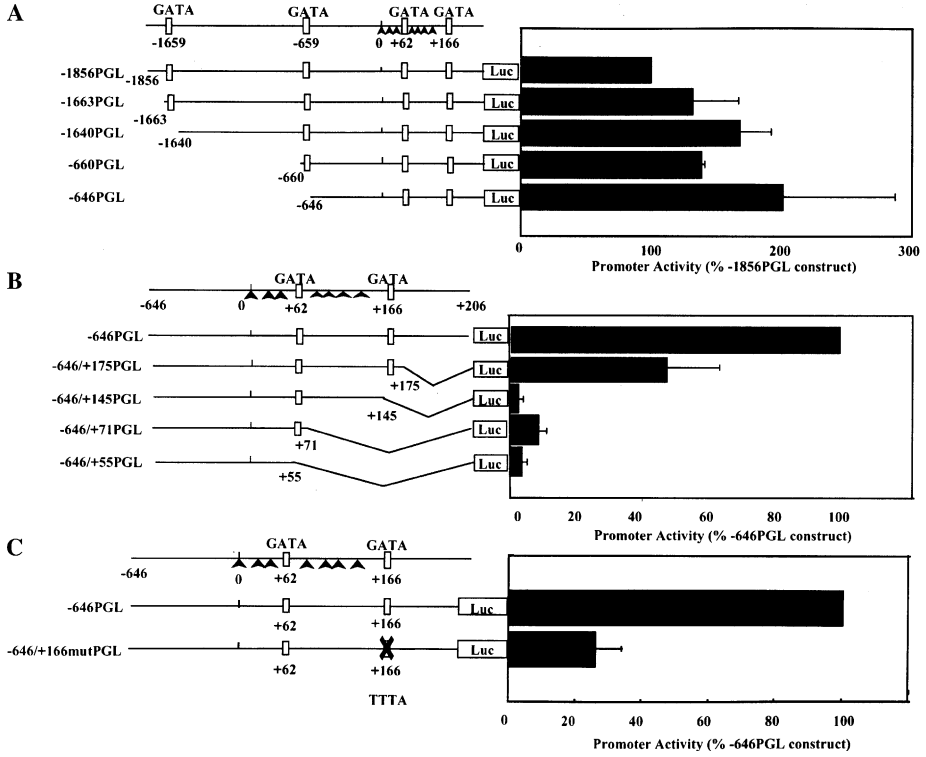

Fig. 2. Transient promoter analysis of the human YB-1 gene in K562 cells. (A) A series of 5′-deletion mutants of the human YB-1 gene promoter were fused to the firefly luciferase gene and transfected into K562 cells. Luciferase activity of cell extract was analyzed and normalized by the control sea pansy luciferase activity. Data are mean values of three separate experiments. Relative luciferase activity of each construct was expressed relative to the activity of −1856 PGL. (B) Reporter analysis of the 5′-UTR region was performed as described in Materials and methods. Data are mean values of three separate experiments. Relative luciferase activity of each construct was expressed relative to the activity of −646 PGL. Triangles represent the transcriptional start sites that have been reported previously [18]. (C) Two-base mutation was introduced in +166GATA (5′-CTTAAACCG) and the promoter activity was compared to the wild-type. Data are mean values of three separate experiments. Luciferase activity of mutant was expressed relative to wild-type activity.

fused to a luciferase reporter gene. As shown in Fig. 2A, deletion of both GATA consensus sequences in the promoter region did not significantly change the activity. Since it has been reported that the +24 to +281 of 5′-UTR region is important for YB-1 expression [18], we further studied the function of 5′-UTR including two GATA elements, a consensus GATA element (+62GATA) and a GGATAA element (+166GATA). Deletion of +206 to +176 did not change the activity significantly, while the deletion of +175 to +146 including the +166GATA site decreased the activity to 20% of the control (Fig. 2B). This decrease of the activity was not due to the deletion of transcriptional start site, because RT-PCR with primers, which anneal the sequence upstream of +166 and luciferase gene, respectively, showed a clear amplified product. (data not shown). When mutation was introduced into this GATA element (GATA to TTTA), the activity was decreased to a level similar to that when it was deleted (Fig. 2C). Next, EMSA analysis using K562 nuclear extracts was performed to confirm the binding of GATA factors to this element. When the radiolabeled oligomer was incubated with nuclear extracts from K562 cells, a single major retarded band was observed (Fig. 3A, lane 2). This band disappeared by the addition of a 300-fold molar excess of the unlabeled +166GATA oligomer (Fig. 3A, lane 3) and the oligomer of GATA binding site in mouse α1 globin promoter region (Fig. 3A, lane 5), while the addition of a mutated +166GATA oligomer had no effect (Fig. 3A, lane 4). Furthermore, the band was abolished mostly by the addition of anti-GATA-1 antibody (Fig. 3A, lane 6) and the intensity became slightly weaker by the addition of anti-GATA-2 antibody (Fig. 3A, lane 7). These results suggest that this band contains mainly the GATA-1 protein, reflecting the findings of quantitative RT-PCR that the copy number of GATA-2 mRNA was much lower than that of GATA-1 in K562 cells (16.3 copies/1000 copies of GAPDH vs 3.6 copies/1000 copies of GAPDH). Together with the results of the promoter assay, it is suggested that this GATA element plays a critical role for YB-1 expression.

Both GATA-1 and GATA-2 proteins activate the YB-1 gene promoter

Although the regulation of YB-1 gene expression by GATA-2 was not confirmed in K562 cells, it is possible that GATA-2 also regulates the YB-1 gene expression in erythroid development, because (1) GATA-1 and GATA-2 were expressed redundantly in the early stage of erythroid differentiation and (2) GATA-2 was complementarily upregulated in GATA-1 mutant mouse in which the expression level of YB-1 mRNA was

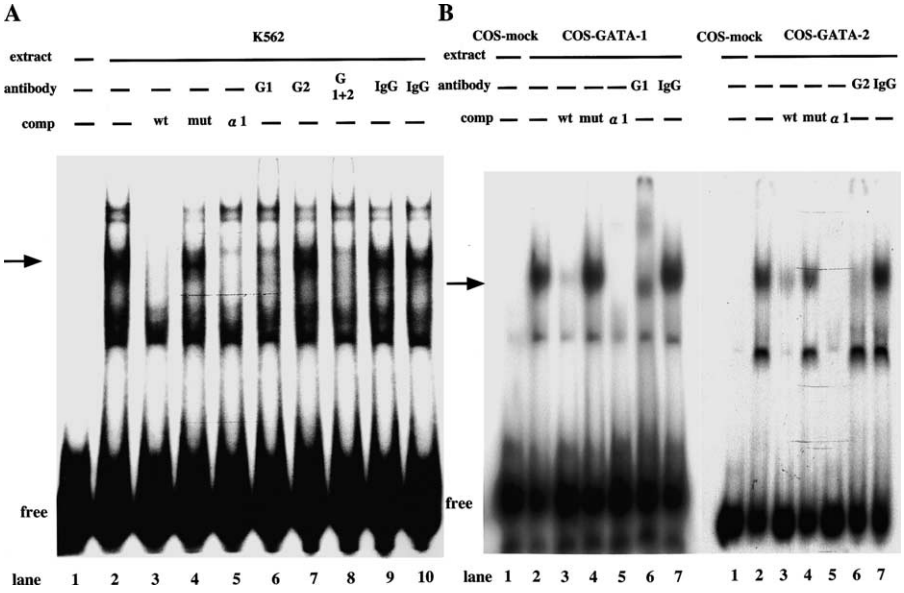

Fig. 3. Binding of GATA-1 and GATA-2 proteins to +166GATA element. (A) Three μg aliquots of nuclear extract from K562 cells were incubated with end-labeled oligomers corresponding to +166GATA in the absence (lanes 1–2, 6–10) or presence of a 200-fold molar excess of unlabeled wild-type (lane 3) or mutant oligonucleotides (lane 4) or GATA binding site of mouse α1 globin promoter region (lane 5). For the inhibition assays with antibodies, 4 μg of anti-GATA-1 (lane 6) or anti-GATA-2 (lane 7) or both (lane 8) was added to the reaction mixture. Alternatively, 4 μg of goat IgG (lane 9) or mouse IgG (lane 10) was added as a control. Arrow indicates the complex of the probe and GATA protein. (B) Three μg aliquots of nuclear extract from COS-7 cells with transfected Mock vector (lane 1) or that expressed GATA-1 protein or GATA-2 protein were incubated with end-labeled oligomers corresponding to +166GATA in the absence (lanes 1, 2) or presence of a 300-fold molar excess of unlabeled wild-type (lane 3), mutant oligonucleotides (lane 4) or a GATA binding site of mouse α1 globin gene (lane 5). For the inhibition assays with antibody, 4 μg of anti-GATA-1 or anti-GATA-2 (lane 6) and 4 μg of control goat or mouse IgG (lane 7) were added to the reaction mixture. Arrow indicates the complex of the probe and GATA protein.

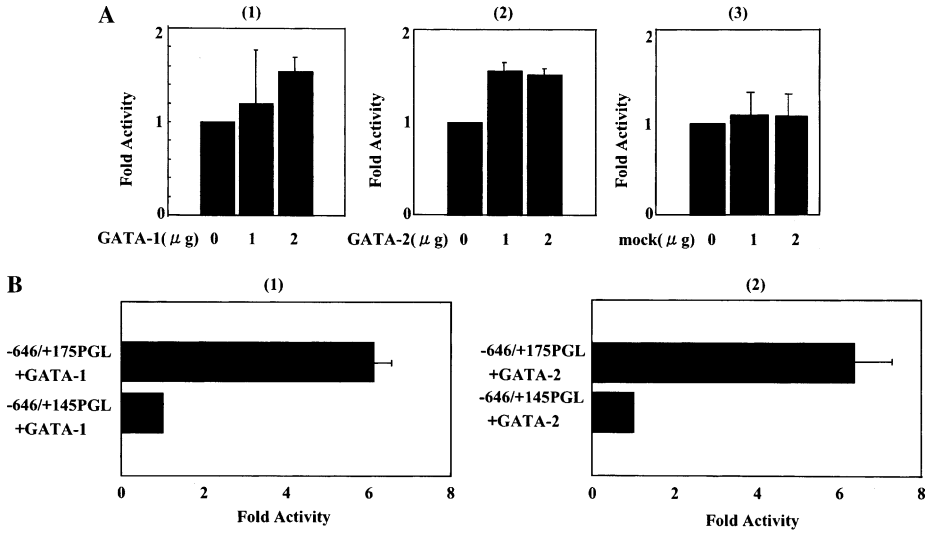

Fig. 4. Activation of human YB-1 gene by GATA-1 and GATA-2 proteins transiently expressed in COS-7 cells. (A) Various amounts of GATA-1 expression vector, GATA-2 expression vector and mock vector were transfected with the −1856PGL construct into COS-7 cells, and luciferase activities were examined. Total amount of plasmids was adjusted by the pBluescript II plasmids. (1) GATA-1 expression vector (pEF-GATA-1) alone, (2) GATA-2 expression vector (pEF-GATA-2) alone, and (3) mock vector alone. (B) −646/+175PGL construct, which has +166GATA site, or −646/+145PGL constructs, which lack +166GATA site, was transfected into COS-7 cells combined with GATA-1 expression vector (1) or GATA-2 expression vector (2). Luciferase activity was expressed relative to that of −646/+145PGL construct. Data are mean values of three separate experiments.

increased. Therefore, we performed EMSA and luciferase activity assays using COS-7 cells that transiently expressed either human GATA-1 or GATA-2 to characterize the individual functions of GATA-1 and GATA-2 on driving the YB-1 gene promoter. As shown in Fig. 3B, binding of these two GATA proteins to +166GATA was confirmed by EMSA using nuclear extracts of COS-7 cells expressing GATA-1 and GATA-2 transiently. Furthermore, both GATA-1 and GATA-2 activated the luciferase activity of −1856PGL construct in COS-7, and the promoter activity of −1856PGL construct increased proportionally along with the increase of the amount of expression vector of each GATA-1 and GATA-2 (Fig. 4A). Of note was the fact that activation of YB-1 promoter by GATA-1 and GATA-2 was abolished by the deletion of +166GATA site (Fig. 4B). These results suggest that both GATA-1 and GATA-2 can activate the YB-1 gene promoter through +166GATA element, and that upregulation of YB-1 expression in GATA-1 mutant mouse spleen in vivo may be due to the complemental upregulation of GATA-2 (Fig. 1C).

Discussion

Analysis of GATA-1 mutant mouse suggests that reduction of GATA-1 activity induces maturation arrest and transformation of erythroid cells [4,5]. In order to clarify the mechanism that induces this abnormal phenotype, we sought to identify the gene, which is differentially expressed between GATA-1 mutant mouse spleen and wild-type spleen, and identified a novel target gene of GATA proteins, YB-1.

YB-1 is a highly conserved gene from bacteria to vertebrates and belongs to cold shock domain (CSD) protein family, and it has been shown to be a multifunctional protein involved in transcriptional regulation, stabilizing or splicing RNA, and translation for several growth or differentiation genes [6–13]. In addition to these functions, YB-1 has been shown to interact with iron binding protein2 (IRP2) and regulate the translation of ferritin gene through iron responsive element (IRE) [14]. Since IRE exists in the 3′ or 5′-UTR region of several genes involved in heme biosynthesis and iron metabolism, it is possible that YB-1 may play a role in erythropoiesis.

By functional analysis, we demonstrated that +166G ATA in 5′-UTR region element, not GATA elements in the promoter region, plays an important role in the regulation of YB-1 gene expression. It has been also shown that GATA factors activate flk-1/KDR gene expression through 5′-UTR palindrome GATA sites [19]. Interestingly, the element in 5′-UTR of flk-1/KDR gene is also GGATA sequence same as the critical element in 5′-UTR of YB-1 gene, which is not a consensus sequence (A/T)GATA(A/G) for GATA factors. Although it has been shown that both GATA-1 and GATA-2 can bind to this sequence [19], GATA factor might change their preference of the binding sequence depending on the location.

In erythroid cells, both GATA-1 and GATA-2 are redundantly expressed, although the expression levels of GATA-1 and GATA-2 change along with the differentiation. Namely, GATA-2 is expressed strongly in the immature stage and the expression level diminishes with

differentiation. Conversely, the expression level of GATA-1 is high in the more maturate stage [20]. Interestingly, the expression level appears to be closely related to each other, because GATA-2 expression was complementarily upregulated in GATA-1 mutant erythroblasts [21]. In fact, the level of GATA-2 mRNA was increased in GATA-1 mutant mouse spleen in the present study. Since we found that not only GATA-1 but also GATA-2 can bind GATA site in 5′-UTR element of YB-1 gene and activate gene transcription through this element, it may be speculated that upregulated YB-1 expression in GATA-1 mutant mouse spleen was due to the complemented upregulation of GATA-2 which binds to GATA site in 5′-UTR element of YB-1 gene in the absence of GATA-1.

In conclusion, we found that YB-1 is a novel target gene of GATA factors. To clarify the biological consequence of aberrant expression of YB-1 gene, the phenotype of YB-1 overexpressing erythroid cells is under investigation.

Acknowledgments

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