Polyomavirus Enhancer-binding Protein 2/Core Binding Factor/Acute Myeloid Leukemia Factors Contribute to the Cell Type-specific Activity of the CD11a Integrin Gene Promoter*

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The CD11a/CD18 leukocyte integrin (LFA-1; also known as αL/β2) mediates leukocyte transendothelial migration during immune and inflammatory responses and participates in lymphoma metastasis. CD11a/CD18 leukocyte-restricted expression is controlled by the CD11a gene promoter, which confers tissue-specific expression to reporter genes in vitro and in vivo. DNase I protection analysis of the CD11a proximal gene promoter revealed DNA-protein interactions centered at position −110 (CD11a-110). Disruption of CD11a-110 reduced CD11a promoter activity in a cell type-specific manner, as it reduced its activity by 70% in Jurkat lymphoid cells, whereas the effect was considerably lower in K562 and HepG2 cells. Electrophoretic mobility shift assays showed evidence of cell type-specific differences in CD11a-110 binding and indicated its specific recognition by members of the polyomavirus enhancer-binding protein 2/core binding factor (CBF)/acute myeloid leukemia (AML) family of transcription factors. AML1B/CBFα transactivated the CD11a promoter, with AML1B/CBFα-mediated transactivation being completely dependent on the integrity of the CD11a-110 element. Therefore, CBF/AML factors play a role in the cell type-restricted transcription of the CD11a integrin gene through recognition of CD11a-110. The involvement of CBF/AML factors in CD11a expression raises the possibility that CD11a/CD18 expression might be deregulated in acute myeloid and B-lineage acute lymphoblastic leukemias, thus contributing to their altered adhesion and metastatic potential.

CD11a/CD18 (LFA-1; also known as αL/β2) is a member of the β2 integrin subfamily, the leukocyte-restricted expression of which is developmentally regulated (reviewed in Ref. 1).

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The CD11a/CD18 mediates leukocyte interactions required for immune and inflammatory responses through the recognition of at least one of its three identified counterreceptors, i.e. CD50, CD54, and CD102 (1–6). The importance of CD11a/CD18 for leukocyte extravasation is exemplified by the existence of an inherited disease (leukocyte adhesion deficiency) in which leukocytes exhibit a deficient expression of the three leukocyte integrins and the clinical symptoms of which are secondary to the lack of phagocyte migration into inflammatory sites (6, 7). Conversely, under certain circumstances, CD11a/CD18 activity might become detrimental; CD11a/CD18 participates in T cell and lymphoma metastasis (1, 8), and ischemia-reperfusion syndromes, myocardial infarction, and allograft rejection have their origin in an excessive and uncontrolled CD11a/CD18-dependent phagocyte extravasation into the tissues (9, 10). To understand the mechanisms controlling transcription of each leukocyte integrin subunit, the proximal regulatory region of the CD11a gene has been isolated (11–13) and shown to confer leukocyte-restricted expression to reporter genes both in vitro and in vivo (11–14). So far, Sp1- and cts-binding sites have been located within the CD11a proximal promoter (12, 15), although their functional contribution to the promoter activity remains unknown.

The members of the polyomavirus enhancer-binding protein 2 (PEBP2)1/core binding factor (CBF)/acute myeloid leukemia (AML) family of heterodimeric (αβ) transcription factors play important roles in hematopoiesis and osteogenesis (16, 17). To date, three distinct α subunits (AML-1/CBFα-2/PEBP2α-B, AML-2/CBFα-3/PEBP2α-C, and AML-3/CBFα-1/PEBP2α-A) and one β subunit (CBFβ/PEBP2β) have been reported in mammalian cells (16). The α subunits exhibit sequence-specific DNA binding ability, whereas the β subunit does not bind to DNA by itself but interacts with the α subunit and increases its DNA binding affinity (18). The AML-1/CBFα transcription factor complex is one of the most frequent targets of chromosomal translocations in acute leukemias (17–19), as a high percentage of AML and B-lineage acute lymphoblastic leukemias have altered AML1 or CBFα alleles. Some of these translocations transform AML-1 into a constitutive transcriptional repressor and disrupt normal hematopoietic cell differentiation (17).

In the present study, we report the identification of a cis-acting element (CD11a-110) specifically recognized by members of the PEBP2/CBF/AML family of transcription factors and implicated in the cell-type restricted activity of the CD11a promoter. Our results indicate the involvement of PEBP2/CBF/AML

1 The abbreviations used are: PEBP2, polyomavirus enhancer-binding protein 2; AML, acute myeloid leukemia; CBF, core binding factor; EMSA, electrophoretic mobility shift assay; CMV, cytomegalovirus.
AML factors in the restricted expression of the CD11a leukocyte integrin and suggest that CD11a/CD18 expression might be deregulated in leukemic cells harboring AML1 or CBFβ gene rearrangements.

**EXPERIMENTAL PROCEDURES**

**Cell Culture**—The cell lines HepG2 (hepatoma), HeLa (epithelial carcinoma), Jurkat (T cell lymphoma), JY (lymphoblastoid B), U937 (histiocytic lymphoma), and K562 (chronic myelogenous leukemia) were cultured in RPMI supplemented with 10% fetal calf serum, 2 mM glutamine, and 50 μg/ml gentamicin at 37 °C in a humidified atmosphere with 5% CO2. Induction of differentiation of K562 cells was accomplished in the presence of phorbol myristate acetate at 10 ng/ml for 24 h.

**Transfections, Plasmids, and Site-directed Mutagenesis**—Transfections in COS-7, HepG2, Jurkat, and K562 cells was performed with Superfect (Qiagen) according to the manufacturer’s instructions. Transfections were carried out using 1 µg of reporter plasmid in 24-well plates and with 4 × 104 (COS-7 and HepG2) or 8–15 × 104 (K562 and Jurkat) cells/well. In all cases, the amount of DNA in each transfection was normalized by using the corresponding insertless expression vector (CMV-β-galactosidase) expression plasmid pCMV-β-galactosidase, and β-galactosidase levels were determined using the Galacto-Light kit (Tropix).

The CD11a-based reporter gene construct pcID11A170-Luc, in which the expression of the firefly luciferase cDNA is directed by the CD11a promoter region −170/+83, has been previously described (11). The promoterless plasmid pXP2 was used as a control in some transfection experiments. Drs. S. Hiebert (Vanderbilt Cancer Center, Nashville, TN), M. A. Vega (Consejo Superior de Investigaciones Científicas, Madrid, Spain) and Y. Ito (Kyoto University, Kyoto, Japan) generously provided the expression plasmids CMV-AML1B, CMV-AMLF1, and pEF-BOS-as1 (AML3), respectively.

Site-directed mutagenesis was performed on the CD11a promoter construct pcID11A170-Luc using a polymerase chain reaction-based approach. For mutation of the CD11a-110 site, oligonucleotides MSTMUTS (5′-CTCCCCGAAACCGATTCTTTCACACTCTTG-3′ (−89)) and MSTMUTAS (5′-GGACAGGTGTGGAAGAAGGTCCGGGTTCAGGGA-3′ (−119)) were synthesized substituting the sequence (−111) 5′-CCCTGCGGTTT-3′ (−100) for the EcoRI-containing sequence 5′-CCGAGATTTT-3′. Polymerase chain reaction was performed on CD11A170-Luc using either oligonucleotides MSTMUTS and LFA-1 promoter in distinct hematopoietic cell lines has previously been revealed that the −170/+43 fragment retains most of the basal and tissue-specific activity (11). To locate cis-acting elements in the CD11a proximal promoter, DNA I protection experiments were performed on the −170/+43 DNA fragment using Jurkat (CD11a+), K562 (CD11a−), and HeLa (CD11a−) nuclear extracts. Several areas of DNA-protein interaction were identified, the boundaries and intensities of which differed among the cell lines tested. Protected sequences included the major transcriptional start site, a consensus Sp1-binding sequence GGAA, and a previously identified Sp1-binding sequence (24), except that cells were lysed in 10 mM Hepes, pH 7.6, 10 mM KCl, 0.1 mM EDTA, 0.1 mM EGTA, 0.75 mM spermidine, 0.1 mM spermine, 2 μg/ml aprotinin, 2 μg/ml leupeptin, and 10 mM Na3MoO4. Samples were subjected to electrophoresis on 6% denaturing polyacrylamide gels in parallel with a G + A sequence ladder generated with the same probe.

**RESULTS**

**DNA-Protein Interactions at the CD11a Gene Proximal Promoter**—Functional analysis of 5′ deletion mutants of the CD11a promoter in distinct hematopoietic cell lines has previously revealed that the −170/+43 fragment retains most of the basal and tissue-specific activity (11). To locate cis-acting elements in the CD11a proximal promoter, DNA I protection experiments were performed on the −170/+43 DNA fragment using Jurkat (CD11a+), K562 (CD11a−), and HeLa (CD11a−) nuclear extracts. Several areas of DNA-protein interaction were identified, the boundaries and intensities of which differed among the cell lines tested. Protected sequences included the major transcriptional start site, a consensus Sp1-binding sequence GGAA, and a previously identified Sp1-binding site at −70 (data not shown). The most prominent footprint, spanning from −90 to −115, could be detected in both strands and was always stronger in Jurkat and HeLa than in K562 cells (Fig. 1). Increasing amounts of Jurkat nuclear extracts were used to determine the boundaries of this footprint (−90−115) (Fig. 1), further demonstrating the existence of DNA-protein interactions around position −110 within the CD11a integrin promoter.

**DNA-Protein Interactions at the CD11a Gene Promoter Element (CD11a−110)**—To characterize the factor(s) giving rise to the −90−115 footprint, EMSA was performed using partly overlapping probes spanning the region between −120 and −79 (Table I). This region is located immediately upstream from the
Sp1-binding element at −70 (15) and downstream from the sequence recognized by an unidentified and ubiquitous ets-related factor (−130) (12). Using the MS7 probe (−120/−88), a low mobility complex, which resolved as a doublet in some experiments, was produced by Jurkat nuclear extracts (indicated by an asterisk in Fig. 2 and hereafter termed AML-110). A specific complex of higher mobility was detected with extracts from the B lymphoblastoid cell line JY (Fig. 2A). By contrast, no retarded complex was produced by nuclear extracts from K562, whereas phorbol myristate acetate-differentiated K562 cells exhibited a complex similar to that seen in Jurkat extracts (Fig. 2A). The specificity of the AML-110 complex was demonstrated as it was completely inhibited in the presence of a 100-fold molar excess of unlabeled MS7, it was partially competed by AP2CONS and unaffected by the unrelated NFkBCONS oligonucleotide (Fig. 2A). In addition, formation of the AML-110 complex was prevented by oligonucleotides MS6 (−110/−79) or MS8 (−130/−99) (Fig. 2, A and B) but was not affected by MS4 (−80/−51), MS5 (−95/−64), or MS9 (−140/−111) (Fig. 2, B and C), thus demonstrating the dependence of the AML-110 complex on the sequence (−110) 5′-CCCTGCGGTTTC-3′ (−99). In fact, EMSA of MS6 (−110/−79) also yielded the AML-110 complex, and its formation was similarly abrogated by either MS8 (−130/−99), MS7 (−120/−88), or MS6 (−110/−79) but not altered by either MS5 (−95/−64) or MS9 (−140/−111) (data not shown). The involvement of the identified sequence in AML-110 complex formation was further confirmed by the lack of inhibitory activity of a mutated MS7 probe (MS7MUT), in which the core of the sequence (−110) 5′-CCCTGCGGTTTC-3′ (−99) had been replaced by an EcoRI site (−110) 5′-CCCTGCGGTTTC-3′ (−99)) (Fig. 2C).

**Figure 2.** Nuclear factors interacting with the MS7 probe and identification of nucleotides involved in recognition of CD11a-110. All panels show EMSA performed on the MS7 oligonucleotide using nuclear extracts from Jurkat leukemic T cells, B lymphoblastoid JY cells, erythroleukemic K562 cells, and phorbol myristate acetate-differentiated K562 cells, as indicated. Major specific retarded complexes are indicated by an asterisk (AML-110). Unlabeled competitor oligonucleotides (MS4, MS6, MS7, MS9, MS7MUT, AP2CONS, and NFkBCONS) were added at 100-fold molar excess.

**Figure 3.** The CD11a-110 element affects the CD11a promoter activity in a cell type-specific manner. Jurkat, K562, and HepG2 cells were transfected with pCD11A170-Luc, the corresponding mutant at the CD11a-110 element (pCD11A170(−110mut)-Luc), or the promoterless vector pXP2, and luciferase activity was determined. Each experiment was performed at least three times using distinct DNA preparations, and a representative experiment is shown. Promoter activity is expressed relative to the activity produced by the wild-type pCD11A170-Luc reporter plasmid in each transfected cell line, after normalization for transfection efficiency. Wild-type and mutant CD11a-110 sites are depicted as filled and open circles, respectively.
AML1; a polyclonal antiserum against the N-terminal region of AML1B produced a supershift, and the R-3034 polyclonal antiserum, which recognizes the AML1B DNA-binding domain, also inhibited complex formation (Fig. 4). Therefore, proteins structurally related to the PEBP2/CFB/AML family of transcription factors specifically bind the CD11a-110 element and give rise to the AML-110 complex.

To further demonstrate recognition of the CD11a-110 element by CBF/AML factors, AML1B, AML3, and CBFβ were overexpressed in COS-7 cells, and the resulting extracts were assayed for binding to MS7. MS7 was recognized by either AML1 or AML3 alone, and coinuciation with CBFβ-containing extracts produced a strong retarded complex (Fig. 5). The intensity of the AML3-containing complex was considerably higher, an effect that might reflect differences between the levels of expression obtained from pEF-BOS and pCDM8 plasmids. More importantly, AML1/CFBβ or AML3/CFBβ recognition of CD11a-110 was competed by either MS7 or AML1CONS, but unaffected by the MS7MUT oligonucleotide. Therefore, CBF/AML factors recognize the CD11a-110 element within the CD11a promoter and participate in formation of the AML-110 complex.

Transactivation of the CD11a Integrin Promoter by CBF/AML Factors—Because mutation of CD11a-110 has a cell type-dependent effect on the activity of the CD11a promoter, we tested the functional effect of overexpressing CBF/AML factors in K562 cells, which express extremely low levels of CBF/AML (23). Expression of AML1B alone had a minimal effect on the CD11a promoter activity, whereas expression of CBFβ always produced a considerable reduction in the activity of the promoter (Fig. 6). However, co-expression of both AML1B and CBFβ produced a considerable increase (12–16-fold) in the activity of the CD11a promoter (Fig. 6). The CD11a promoter transactivation was observed at distinct reporter:vector ratios (Fig. 6), and its dependence on the co-expression of AML1B and CBFβ was in agreement with the structural data shown in Fig. 5 and the known functional activities of CBF/AML factors (16–19). Therefore, CBF/AML factors directly contribute to the activity of the CD11a integrin gene promoter.

Because CD11a-110 mutation prevented its recognition by CBF/AML factors, we analyzed the effect of disrupting CD11a-110 on the CBF/AML-mediated transactivation of the CD11a promoter. As shown in Fig. 7, mutation of CD11a-110 greatly reduced the AML1B/CFBβ-mediated transactivation of the CD11a promoter. On average, disruption of CD11a-110 reduced the transactivation to 25% of the level observed on the wild-type promoter (Fig. 7). In addition, AML3 was also capable of transactivating the CD11a promoter in a CD11a-110-dependent manner, although its level of transactivation was always lower than that produced by AML1 (data not shown). Therefore, CBF/AML transactivation depends on the integrity of CD11a-110, and CBF/AML factors contribute to CD11a promoter activity by recognizing the CD11a-110 element.

DISCUSSION

The CD11a/CD18 integrin mediates essential adhesive interactions during leukocyte transendothelial migration, in CTL- and NK-mediated killing, in antigen presentation, and in T cell hybridoma and lymphoma metastasis (reviewed in Ref. 1). CD11a/CD18 expression is leukocyte-restricted by transcriptional mechanisms acting on the regulatory regions of the CD11a and CD18 genes (11–14, 26–28). We have previously shown that deletion of the –170/–100 fragment greatly reduces the basal activity of the CD11a promoter (11). In the present report, we demonstrate the presence of a CBF/AML-binding site at –110 (CD11a-110) that is specifically recognized by members of the CBF/AML family of transcription factors and contributes to the cell type-specific activity of the CD11a promoter. Consequently, CBF/AML factors appear as essential players in the control of the cell type-specific transcription of the CD11a gene through recognition of the CD11a-110 element. CD11a-110 is the first functionally characterized cis-acting element within the CD11a gene promoter of which the contribution to the cell type-specific activity of the CD11a promoter...
resembles that of Sp1-binding sites within the CD11b and CD11c promoters (15, 29).

The AML-1/CBFβ transcription factor complex is one of the most frequent targets of chromosomal translocations in acute leukemias (17–19). Both genes are affected by as many as 11 chromosomal translocations in either AML or B-lineage acute lymphoblastic leukemia, and some of these rearrangements transform AML-1 or CBFβ into constitutive transcriptional repressors (e.g., AML1/ETO and CBFβ/MYH11) that disrupt normal hematopoietic cell differentiation (17, 30). AML1/ETO can act as a dominant negative inhibitor of AML1 transactivation (31), although AML1/ETO and AML1 can also synergistically transactivate the macrophage colony-stimulating factor receptor promoter (32). Thus, the involvement of AML1 and CBFβ in the CD11a gene transcription raises the possibility that CD11a/CD18 integrin expression might be altered in some of these lymphoproliferative disorders. In this regard, diminished or absent CD11a/CD18 expression has been noted in cases of B-lineage acute lymphoblastic leukemia (33), and the expression of CD11a/CD18 significantly correlates with splenomegaly, resistance to induction chemotherapies and short survival periods in AML patients (34). Therefore, it will be of interest to determine whether an association exists between AML1 or CBFβ chromosomal translocations and the expression of the CD11a/CD18 integrin, a task we are currently undertaking.

AML1 products are expressed in most tissues and at high levels in hematopoietic cells (16–19, 30), in which they collaborate in the organization of promoters prior to transcriptional activation and are transcriptional activators of myeloid and lymphoid-specific genes, including T cell receptor subunits, (35), myeloperoxidase (36), interleukin-3 (37), and neutrophil elastase (38). However, CBF/AML proteins are relatively weak transcriptional activators in isolation, and they potently enhance transcription rates in cooperation with several factors (e.g., Ets-1, PU.1, c-Myc, and CCAAT enhancer-binding protein a) via cooperative DNA binding or interactions with co-activators (17, 39). In fact, CBF/AML-binding sites are usually flanked by sites for CCAAT enhancer-binding protein, Myb, or Ets factors (16–19). In the case of the CD11a promoter, the CD11a-110 element is adjacent to an Ets-binding element and to putative CCAAT enhancer-binding protein- and Myb-binding sites, suggesting that some of these interactions may participate in the transactivation of the CD11a promoter by CBF/AML proteins and contributing to the cell type-specific expression of the CD11a integrin. On the other hand, a different type of interaction might also affect the involvement of CBF/AML factors in the transcriptional activity of the CD11a promoter. Thus, in agreement with the partial inhibitory effect of AP2CONS on AML-110 complex formation (shown in Fig. 2), we have obtained evidence that AP-2α and AP-2β can transactivate the CD11a promoter and that recombinant AP-2 factors bind the CD11a-110 element in vitro, although with lower affinity than CBF/AML factors (data not shown).

Because AP-2 factors are capable of preventing the binding of other transcription factors to overlapping or adjacent cis-acting sequences (NF1, AP-3, and NFkB) (40–42), it is conceivable that AP-2 could also be regulating the access of CBF/AML factors to the CD11a gene promoter in certain cell types and thus regulating CD11a integrin expression.

The CD11a/CD18 integrin plays a key role in the triggering of immune and inflammatory responses (1). However, under certain circumstances (e.g., lymphoma metastasis and ischemia-reperfusion injuries), the functional activity of CD11a/CD18 and related integrins becomes detrimental to the host, and in fact, anti-CD11a antibodies can inhibit these processes (8–10). The involvement of CBF/AML factors in the transcription of the CD11a gene implies that CD11a/CD18 expression might be deregulated (either positively or negatively) in leukemic cells with chromosomal translocations affecting either AML1 or CBFβ alleles (AML M2 and B-lineage acute lymphoblastic leukemia), thus contributing to altered adhesive and/or metastatic phenotypes. The identification of CBF/AML factors as key players in the transcription of the CD11a gene will allow the dissection of the signaling pathways that regulate CD11a/CD18 integrin expression and, subsequently, the development of strategies to modulate the adhesive and migratory capabilities of leukocytes and tumor cells.

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