Renin gene expression is subject to complex developmental and tissue-specific regulation. A comparison of the promoter sequences of the human, rat, and mouse renin genes has revealed a highly conserved sequence homologous to the DNA recognition sequence for CBF1 (CSL/RBP-Jκ/Su(H)/LAG1/RBPJsu). Electrophoretic mobility shift assays document that As4.1 cell nuclear protein complex binding to the putative rat renin CBF1-binding site (~175 to ~168 bp) contains CBF1. Transient transfection analyses in COS-7 cells further document that a CBF1-VP16 fusion protein and the intracellular domain of Notch1 robustly activate a promoter containing multiple copies of the rat renin CBF1-binding site. An Ets-binding site (~143 to ~138 bp) has also been identified in the rat renin promoter by sequence comparisons and electrophoretic mobility shift assays. Transcription factor Ets-1 is capable of activating the rat renin promoter through the Ets-binding site. Mutation of the CBF-binding site significantly increases transcriptional activity of the rat renin promoter in Calu-6 and COS-7 cells but not in As4.1 cells, whereas mutation of the Ets-binding site reduces promoter activity of the rat renin gene in all three cell lines. Finally, we show that the intracellular domain of Notch1, Ets-1, and HOXD10-PBX1b-PREP1 activate the rat renin promoter cooperatively in COS-7 cells. These results strongly suggest that the renin gene is a downstream target of the Notch signaling pathway.

Renin, through its participation in an enzymatic cascade that results in the production of angiotensin II, the major effector molecule of the renin-angiotensin system, plays a major role in blood pressure regulation and electrolyte homeostasis. Expression of the renin gene is subject to complex tissue-specific and developmental regulation. Progress has been made recently in understanding the mechanisms of this regulation (1). An enhancer (2, 3) and a proximal promoter region (4) have been identified as critical for expression of the mouse renin gene (Ren-1*) in the mouse kidney tumor-derived As4.1 cell line. This cell line was developed from a Ren-2-T antigen transgenic line and has been shown to retain many features characteristic of renin-expressing juxtonglomerular cells in the kidney (5). A HOXD-PBX-binding site has been located within the Ren-1* proximal promoter region (6). Mutation of the site in a construct containing 4.1 kb of the Ren-1* 5′-flanking sequence caused a more than 10-fold decrease in transcriptional activity. HOXD10 is capable of pairing with PBX1b and binding to the Ren-1* HOXD-PBX site with high affinity. Moreover, PREP1 has been shown to form a ternary complex with HOXD10 and PBX1b on the Ren-1* promoter. The HOXD-PBX-binding site is also present in human and rat renin promoters, suggesting an important role of this site in the regulation of renin expression.

Notch is a transmembrane receptor that regulates expression of genes in a cell type-specific fashion to determine cell fate and patterning through cell-cell communication (see Refs. 7–9 for reviews). There are four Notch genes (Notch1–Notch4) in mammals. Upon receipt of extracellular signals mediated via binding of the specific ligands, Jagged and Delta-like, the intracellular domain of Notch is released by proteolytic cleavages and translocates to the nucleus, where it subsequently interacts with the transcriptional repressor CBF1 and converts it to a transcriptional activator through replacement of the CBF1-bound co-repressor complex with a co-activator complex. Several co-repressors have been identified, including CIR, SMART, and N-CoR (10, 11), whereas Mastermind appears to be a major co-activator for Notch signaling (12, 13).

In this study, we identified two new transcription factor-binding sites, a CBF1- and an Ets-binding site, in the promoter region of the rat renin gene in addition to the HOXD-PBX-binding site identified previously (6). The CBF1-binding site acts as a negative regulatory element in renin-expressing Calu-6 cells and non-renin-expressing COS-7 cells, whereas the Ets-binding site is a positive regulatory element in all cell lines tested. Moreover, we showed that N1IC1 and Ets-1 activated the renin promoter through the CBF1- and Ets-binding sites, respectively. Finally, we demonstrated that N1IC, Ets-1, and HOXD10-PBX1b-PREP1 were capable of cooperating with each other to activate the rat renin promoter.

Materials and Methods

Plasmids—Plasmid rR240 was constructed by inserting a fragment containing the rat renin gene region from −241 to +16, which was amplified by the polymerase chain reaction from a bacterial artificial chromosome, CH230 101.37, containing the rat renin genomic sequence, into the Xhol/HindIII-digested pGL2-basic (Promega). Plasmids containing mutations in the transcription factor-binding sites of the rat renin promoter (see Fig. 3) were created using the QuikChange site-directed mutagenesis kit (Stratagene). The oligonucleotides used to generate mutations in the CBF1-, Ets-, and HOXD-PBX-binding site are 5′-CTCGGTTCAGGCATGTATTTAagaaCAGCAGATTGACTGTTGCCACCT-3′, 5′-CCTGCACTCTGTTCAAGGTTCTCTTACATTTACCTCCCCT-3′, and 5′-GGACCCCTGGAATAAcAaaCTAGACGGCTGCGCTG-3′, respectively. Plasmid 7XrCBF1-SV40 or 5XmCBF1-SV40 was constructed by inserting seven tandem copies of a double-stranded oligonucleotide

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containing the rat renin CBF1-binding site (5'-tcagGCCATGGTTTC-
CCACACTCGATTC-3') or five tandem copies of an oligonucleotide con-
taining the mutated CBF1-binding site (5'-tcagGCCATGGTTTasag-
CAGTGATTC-3'), respectively, into the Xhol site of pG2L-Promoter
(Promega).

The expression vectors for Ets-1, Ets-2, and CBF1 contain the
IMAGE full-length cDNA clones inserted in pCMV-Sport6 and
were purchased from Open Biosystems. The mouse N1IC expression
plasmid contains the intracellular domain of Notch1 under the control
of a CMV promoter and was kindly provided by Dr. R. Kopan (Wash-
ington University School of Medicine, St Louis, MO) (14). VP16-CBF1
was constructed by inserting the polymerase chain reaction-amplified
cDNA encoding CBF1 into the EcoRI/XbaI-digested pVP16 (BD Bio-
sciences). The expression vectors for HOXD10, PBX1 and PREP1
were purchased from Open Biosystems. The mouse N1IC expression
sequences (Promega).

Identification of a putative CBF1-binding site
The EMSA was performed as described previously (3). The
EMSA was performed using rRen as the labeled probe and nuclear
extracts prepared from As4.1 cells. Competitors (100×) and anti-CBF1 serum (anti-CBF1) added are indicated on the top of the gel. The
rRen-CBF1 complex is labeled as CBF1. SS indicates the supershifted complex. The free probe is not shown.

RESULTS

Identification of a CBF1-binding Site in the Rat Renin Prom-
er—A phylogenetic footprinting comparison of human, rat, and
mouse renin promoter sequences (rRen, mRen, and hRen, respectively) reveals a conserved CBF1-binding site. Shown also are sequences of oligonucleotides used as competitors in EMSA. 23/24 contains two CBF1-binding sites (16). HES-1 is a promoter sequence of the mouse Hairy enhancer of split gene containing two CBF1-binding sites. Both NF-kb and IL-6kB contain an NF-
competitors in EMSA. 23/24 contains two CBF1-binding sites (16). HES-1 is a promoter sequence of the mouse

Identification of an Ets-binding Site within the Rat Renin Prom-
er—An Ets-binding site has been identified in the hu-
notch signaling pathway, a construct containing seven tandem copies of the rat renin CBF1-binding site inserted immediately upstream an SV40 promoter (7XrCBF1-SV40) was cotransfected into COS-7 cells with an expression vector for CBF1, VP16-CBF1, or N1IC. Results showed that overex-
expression of CBF1 did not have a significant effect on promoter
activity, whereas VP16-CBF1 or N1IC activated the promoter.

Identification of a CBF1-binding site in the rat renin promoter. A, alignment of the rat, mouse, and human renin promoter
sequences (rRen, mRen, and hRen, respectively) reveals a conserved CBF1-binding site. Shown also are sequences of oligonucleotides used as competitors in EMSA. 23/24 contains two CBF1-binding sites (16). HES-1 is a promoter sequence of the mouse Hairy enhancer of split gene containing two CBF1-binding sites. Both NF-kb and IL-6kB contain an NF-
competitors in EMSA. 23/24 contains two CBF1-binding sites (16). HES-1 is a promoter sequence of the mouse

Identification of a putative CBF1-binding site for the transcriptional repressor, CBF1. Results from EMSA showed
that a double-stranded oligonucleotide, rRen, which represents the rat promoter sequence from −185 to −166 bp containing putative CBF1-binding site, formed a complex with nuclear proteins prepared from As4.1 cells (Fig. 1B). This complex could be efficiently competed by 100-fold molar excess of unla-
beled rRen itself, mRen, a corresponding mouse renin promoter
sequence, and oligonucleotides containing previously identified
CBF1-binding sites, including 23/24, HES-1, NF-kb, and IL-
6kB, whereas it could not be competed by oligonucleotides that
do not contain the CBF1-binding site such as IL-2kB and IgkB
(Fig. 1, A and B) (16). Furthermore, a CBF1-specific antibody
supershifted the DNA-protein complex. These results demon-
strate that the rat promoter sequence from −175 to −168 is a
CBF1-binding site.

To test in vivo whether the rat renin CBF1-binding site functions as a binding site for CBF1 and target for activation by
the Notch signaling pathway, a construct containing seven tandem copies of the rat renin CBF1-binding site inserted
immediately upstream an SV40 promoter (7XrCBF1-SV40) was cotransfected into COS-7 cells with an expression vector
for CBF1, VP16-CBF1, or N1IC. Results showed that overex-
pression of CBF1 did not have a significant effect on promoter
activity, whereas VP16-CBF1 or N1IC activated the promoter
by >20- or >200-fold, respectively (Fig. 1C). Moreover, neither
VP16-CBF1 nor N1IC has any stimulatory effect on activity of
the promoter containing five tandem copies of the mutated rat
renin CBF1-binding site (5XmrCBF1-SV40). These results indi-
cate that the rat renin CBF1-binding site is capable of bind-
ing the CBF1-N1IC complex in vivo. Results from transfection
analysis also showed that N1IC-induced promoter activity of
construct 7XrCBF1-SV40 was reduced by overexpression of
CBF1, consistent with previous reports (17, 18).

Identification of an Ets-binding Site within the Rat Renin Prom-
er—An Ets-binding site has been identified in the hu-
man renin promoter, which resides immediately 3’ to the TATA
box (19). However, no Ets-binding site is identifiable at the
same position in the rat renin promoter. The rat renin pro-
mot er sequence does, however, contain several GGA(A/T) (or inverting (T/A))TCC motifs, which are the consensus binding sequences for the Ets family transcription factors. Oligonucleotide (rR150), which contains a putative Ets-binding site located between -143 and -138 bp of the rat renin promoter, was found to bind nuclear proteins prepared from As4.1 cells (Fig. 2). Competition EMSA using a 100-fold molar excess of unlabelled competitor oligonucleotides containing truncations and mutations within rR150 confirmed that the critical base pairs for nuclear protein binding include TTCC. Moreover, the DNA-protein complexes in EMSA are efficiently competed by an oligonucleotide (Fig. 2, conEts) containing the consensus Ets-binding site. These results demonstrate that the rat renin promoter contains an Ets-binding site located between -143 and -138 bp.

**Effect of Mutation in the CBF1-, Ets-, or HOX-PBX-binding Site on Promoter Activity of the Rat Renin Gene**—To test whether mutation of the CBF1-, Ets-, or HOX-PBX-binding site affects activity of the rat renin promoter, constructs containing single, double, or triple mutations in these sites (Fig. 3) were transfected into Calu-6, As4.1, or COS-7 cells. Calu-6 cells were developed from a human pulmonary carcinoma and express their endogenous renin gene (20), whereas COS-7 cells do not express their endogenous renin gene but have been widely used to study the Notch signaling pathway. Consistent with this, a 60-fold higher basal expression from the rat renin promoter was observed in Calu-6 cells than in COS-7 cells, indicating that CBF1 binds to the rat renin promoter and acts as a transcriptional repressor. However, mutation of the CBF1-binding site resulted in a significant increase in promoter activity in both Calu-6 and COS-7 cells, indicating that CBF1 binds to the rat renin promoter and acts as a transcriptional repressor. However, mutation of the CBF1-binding site had no effect on promoter activity in As4.1 cells, suggesting that one or more co-repressors necessary for CBF1-mediated repression may be absent in As4.1 cells. Alternatively, the Notch pathway may be constitutively active in these cells. Mutation of the Ets-binding site reduced promoter activity by 63, 33, and 78% in Calu-6, As4.1, and

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**FIG. 2.** Identification of an Ets-binding site in the rat renin promoter. A, sequences of oligonucleotides used in EMSA. The Ets-binding sites are in **bold**. B, EMSA was performed using the double-stranded oligonucleotide rR150 as the labeled probe and nuclear extracts prepared from As4.1 cells. Competitors (100×) added are indicated on the top of the gel. Specific DNA-protein complexes are indicated by arrows. The free probe is indicated by FP. conEts, an oligonucleotide containing the consensus Ets-binding site.

**FIG. 3.** Schematic representation of reporter constructs used in transfection assays. Shown are the reporter constructs containing wild-type (WT) or mutated rat renin promoter with single, double, or triple mutations in the transcription factor-binding sites. LUC, luciferase.

**FIG. 4.** Mutational analysis of transcription factor-binding sites in the rat renin promoter. Calu-6 (A), As4.1 (B), or COS-7 (C) cells were transfected with 2 μg of reporter as indicated and 25 ng of RSV-β-galactosidase. Luciferase (Luc) activity for construct rR240 in As4.1 or COS-7 cells is expressed relative to that in Calu-6 cells (arbitrarily set to 100). Luciferase activity for each of the rR240-based mutant constructs in each cell line is expressed relative to that of rR240. * and **, significantly different (p < 0.05) relative to rR240 in the same cell line and to each other, respectively, as measured by Student’s t test.
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Promoter activity of the rat renin gene. The enhancement by N1IC of the HOXD10-PBX1b-PREP1 induction was reduced to 1.7- or 2.5-fold when the CBFL- or HOXD10-PBX1b-PREP1-binding site, respectively, was mutated.

When COS-7 cells were cotransfected with expression vectors for Ets-1, HOXD10, PBX1b, and PREP1 altogether with rRen240, a 14.4-fold induction over basal promoter activity of the rat renin gene was observed (Fig. 5C). N1IC further enhanced this induction to 31.4-fold. Interestingly, N1IC decreased Ets-1-HOXD10-PBX1b-PREP1-mediated induction by 2-fold in COS-7 cells transfected with the rat renin promoter containing the mutated CBFL-binding site. Although Ets-1 alone had no effect on activity of the rat renin promoter containing the mutated Ets-1-binding site, it is capable of further increasing the HOXD10-PBX1b-PREP1-mediated activation of the same promoter. It is possible that Ets-1 may directly or indirectly interact with the HOXD10-PBX1b-PREP1 complex. Moreover, overexpression of N1IC did not alter the effect of Ets-1-HOXD10-PBX1b-PREP1 on activity of this mutant promoter, demonstrating again that not only is the activation of the rat renin promoter by N1IC dependent on the presence of the CBFL-binding site but on the Ets-1-binding site as well. On the contrary, mutation of the HOX-PBX-binding site did not change the effect of N1IC. A 2-fold induction by N1IC over the Ets-1-HOXD10-PBX1b-PREP1-mediated activation of the promoter was still observed. Finally, when the construct rRen240/mPHEC, in which the HOX-PBX-, Ets-1-, and CBFL-binding sites are all mutated, was tested in COS-7 cells, no significant effect from N1IC, Ets-1, or HOXD10-PBX1b-PREP1 by themselves on promoter activity was detected. When compared with the 31.4-fold activation of the wild-type rat renin promoter, only a 2-fold activation of this promoter was observed in COS-7 cells simultaneously expressing Ets-1, HOXD10, PBX1b, PREP1, and N1IC, suggesting critical roles for these three cis-regulatory sites in regulating renin gene expression.

DISCUSSION

In this study, a CBFL- and an Ets-binding site were identified in the promoter region of the rat renin gene. Transcription factor Ets-1 binds to the Ets-binding site and activates the rat renin promoter, whereas CBFL acts as a transcriptional repressor in renin-expressing Calu-6 and non-renin-expressing COS-7 cells. Moreover, N1IC is capable of counteracting the negative effect of CBFL1 and activating the rat renin promoter via the CBFL-binding site in cooperation with Ets-1 and HOXD10-PBX1b-PREP1.

The Ets transcription factors are implicated in cellular proliferation, differentiation, migration, apoptosis, and cell-cell interactions (21). An Ets-binding site has also been identified in the human renin promoter (19). It is located immediately 3’ to the TATA box and capable of binding Ets-1 (22, 23). However, whether it contributes to promoter activity of the human renin gene has not been determined. Ets-1 has been reported to play a role in vascular development and angiogenesis (24). Mice deficient for Ets-1 have severe kidney abnormalities and/or lethal angiogenic defects (25). Considering that renin-expressing cells are associated with the branching of renal arterioles (26, 27), Ets-1 may be one of transcription factors critical for renin gene expression. Whether any of the Ets consensus sequences present in the mouse renin promoter is a functional Ets-binding site remains to be investigated.

The Notch signaling pathway also plays an important role in forming the vasculature (28). Mice deficient in genes encoding Notch, Notch ligands, and components of the Notch signaling pathway all show vascular defects. Notch and Notch ligands are expressed throughout the vasculature early in embryonic development but restricted to arterial vessels later. This is
consistent with the notion that renin expression is only detected in developing arteries in the mouse or rat fetal kidney (29, 30).

Identification of the renin gene as the downstream target of the Notch signaling pathway provides us with some clues in understanding the tissue-specific regulation of renin gene expression. We have previously hypothesized that the binding of transcriptional repressors to the renin promoter may be the reason that renin transcription is turned off in non-renin-expressing cells (2). Here, we show that CBF1 is one of those
hypothesized repressors. Upon activation of notch signaling, the direct interaction of NIC with CBFI1 turns CBFI1 from a repressor to an activator. However, the renin gene is not expressed in every cell in which Notch signaling is activated, suggesting that Notch is necessary but not sufficient for renin gene expression. Cooperation between CBFI1-Notch and other transcription factors such as Ets-1 and HOX D10-PBX1b-PREP1 may be necessary for renin expression. This fits a model proposed by Furriols and Bray (31) for Notch target gene regulation. They suggest that in the absence of Notch, DNA-bound CBFI prevents transcriptional activators from enhancing transcription. Notch activation can alleviate the repression so that CBFI1 is capable of cooperating with other DNA-bound activators to promote transcription.

We observed that N1IC had reduced but still significant stimulatory effect on the promoter activity of the rat renin gene even when the CBFI1-binding site is mutated. However, when the Ets-binding site is mutated, no significant effect by N1IC is observed. It is possible that N1IC directly or indirectly interacts with DNA-bound Ets-1 to promote this increase. Interactions of N1IC with transcription factors other than CBFI1 have been reported. For example, N1IC is capable of inhibiting NF-kB activity in the nucleus by a direct interaction (32). Moreover, N1IC can act as a co-activator for transcription factor LEF-1 (33).

We showed that the wild-type rat renin promoter or the promoter containing triple mutations in the HOX gene regulatory region enhances transcription factors such as Ets-1 and HOX D10/PBX1 binding site in the nucleus (34). However, we have found that N3IC can also cooperate with Ets-1 or HOXD10-PBX1b-PREP1 to activate the rat renin promoter, although the effect by N3IC is smaller than that of N1IC (data not shown). Moreover, an additive effect was obtained when both N1IC and N3IC were cotransfected with the promoter-reporter construct in the presence or absence of other expression vectors (data not shown). These results suggest that members of the Notch family may have redundant roles in regulating renin gene expression. We are currently studying the renin expression pattern in mice deficient for Notch3 expression (35), which have been shown to have defects in arterial differentiation and maturation of vascular smooth muscle cells (36), to further understand the mechanisms of regulation of renin gene expression by Notch.

A screen of the Drosophila X chromosome for genes whose dosage affects the function of the HOX gene Deformed revealed that Notch is one of these genes (37). Notch also affects the function of another HOX gene, Ultrabithorax. These results suggest that Notch may be generally involved in homoeotic function. Here, we show that Notch signaling and HOX-PBX-PREP complex functionally interact with each other to activate renin gene expression. The finding suggests a role for renin in the development of renal vasculature.

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Activation of the Rat Renin Promoter by HOXD10·PBX1b·PREP1, Ets-1, and the Intracellular Domain of Notch
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