Expression of the cdc25B gene is up-regulated late during cell cycle progression (S/G2). We have cloned the murine cdc25B promoter to identify elements involved in transcriptional regulation. A detailed structure-function analysis led to the identification of several elements that are located upstream of a canonical Inr motif at the site of transcription initiation and are involved in transcriptional activation and regulation. Activation of the promoter is largely mediated by NF-Y and Sp1/3 interacting with one and four proximal binding sites, respectively. In addition, NF-Y plays an essential role in cell cycle regulation in conjunction with a repressor element (cell cycle-regulated repressor) located −30 nucleotides upstream of the putative Inr element and overlapping a consensus TATA motif. The cell cycle-regulated repressor is unrelated to the previously described cell cycle-regulated repressor elements. Taken together, our observations suggest that expression of the cdc25B gene is controlled through a novel mechanism of cell cycle-regulated transcription.

Cell cycle progression in mammalian cells is associated with the phase-specific transcription of defined sets of genes (1). Such periodically expressed genes frequently encode proteins that either directly control cell cycle progression or function in periodically occurring metabolic processes, such as nucleotide and DNA biosynthesis. A major regulator of the cell cycle-dependent expression of these genes is the transcription factor E2F (2–4). Transcriptionally inactive complexes of E2F with pocket proteins of the Retinoblastoma protein (prb) family assemble in G1/early G1, but during cell cycle progression these complexes dissociate, and the release of transcriptionally active “free E2F” leads to the activation of E2F-responsive genes. It has become clear, however, that E2F can also act as an active repressor, which, at least in part, appears to be due to the retinoblastoma protein-mediated recruitment of histone deacetylases. The first example of a gene that is repressed by E2F is the mouse B-myb gene (5), but a number of other genes repressed via E2F sites in their promoters have been identified, for example E2F-1 (6, 7), arc-1 (8), cdc 6 (9–11), cdc25A (12, 13), and p107 (14). Interestingly, structure-function analysis of the B-myb promoter identified an E2F binding site close to the transcription start sites, which is necessary but not sufficient for cell cycle regulation (15, 16). Mutational analyses showed that an adjacent element, termed Bmyb-CHR,1 is indispensable for repression and acts as a corepressor element together with the E2F-binding site.

cdc25C exemplifies a group of cell cycle genes whose transcription is up-regulated later than that of B-myb, i.e. in S/G2. cdc25C was originally discovered in Schizosaccharomyces pombe as a regulator of the G2 to M progression (17, 18). Higher eukaryotes contain at least three genes with a high degree of similarity to cdc25, encoding the Cdc25A, Cdc25B, and Cdc25C protein phosphatases (19–28). The Cdc25C phosphatase activates the Cdc2 cyclin B complex and thereby enables the entry into mitosis (20, 24, 28–30). Cdc25A appears to play a role in regulating entry into S phase (13, 26, 31), whereas Cdc25B is required for the G2 to M progression (32–36).

For the cdc25C promoter, repression of upstream activators via a bipartite site, consisting of the “cell cycle-dependent element” and the “cell cycle genes homology region” (CHR), has been established as the major regulatory mechanism (37, 38). As shown by genomic footprinting, both elements are cooperatively bound in a periodic fashion by a repressor that has been designated CDF-1 (37, 39). A similar mechanism seems to be of global relevance, because a number of other similarly regulated cell cycle genes, such as cyclin A (37, 40), cdc2 (37, 41), CENP-A (42), polo-like kinase (43), and survivin (44), have been identified. Recently, a factor (CHF) interacting with the CHR in the cyclin A promoter has been described (45).

Cell cycle regulation of cdc25B resembles that of cdc25C, which is in agreement with its function at the final stages of the cell cycle (32–36). The cdc25B gene is of interest also in view of its possible involvement in human cancer (19, 46–48), and its oncogenic potential in transgenic mice (49, 50). However, to date, the promoter of the cdc25B gene has not been analyzed, and consequently the mechanism controlling the cell cycle-regulated expression is unknown. In the present study, we have addressed this question. We have cloned the murine cdc25B promoter and have identified regulatory elements and interacting transcription factors required for cdc25B transcription and contributing to its regulation of expression during the cell cycle.

**EXPERIMENTAL PROCEDURES**

**Cell Culture—** The murine cell line NIH3T3 (kindly provided by R. Treisman, ICRF, London) was maintained at 37 °C in 5% CO2 in Dulbecco’s modified Eagle’s medium supplemented with 10% fetal bovine

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1 This abbreviation is used: CHR, cell cycle genes homology region; CDF, cell cycle-dependent element-binding factor; PCR, polymerase chain reaction; EMSA, electrophoretic mobility shift assay; CCRR, cell cycle-regulated repressor; NF-Y, nuclear factor-Y; DMS, dimethyl sulfate; Inr, Initiator; CCRE, cell cycle-regulated repressor element.
serum, penicillin, and streptomycin.

Transfections and Luciferase Assays—Cells were plated on 35-mm (diameter) tissue culture plates at a density producing 60–80% confluence at the time of the transfection and transfected using the cationic lipid DOTAP as described by the manufacturer (Roche Molecular Biochemicals). For synchronization in G0, cells were maintained in serum-free medium for 3 days. Stimulation was carried out for the indicated times with 10% fetal calf serum. Luciferase activity was determined as published elsewhere (38, 51).

Library Screening—The murine genomic λ-Fix phage library 129 FVJ (Stratagene) was screened with a 69-base pair oligonucleotide (probe 1, 5'-TCTAGCTACCTGTCGCCGCGGGGTACCACTGTCGAGCT-3') annealing to the 5'-end of the murine cdc25B eDNA (947) (mutated bases 167 or 223), and the DNA was amplified and further mapped in the 3'-end of the murine cdc25B cDNA (52). Three phage clones were isolated and subcloned in the pBluescriptIISK vector (Stratagene). The experimental strategy included the following precautions. (i) The number of PCR cycles was kept low to obtain a linear amplification of the PCR products, which was possible by the incorporation of radioactive precursor nucleotides and evaluation by autoradiography and β-ray detection scanning. (ii) All results were standardized using the signal obtained with glyceraldehyde-3-phosphate dehydrogenase, whose expression is independent of cell proliferation. (iii) All experiments were performed with at least two independent cDNA preparations.

cdc25B Promoter Constructs—Prizers carrying restriction sites were used for PCR with pBliskcdc25B as the template to generate a series of 5' terminal deletions with compatible ends for cloning as KpnI/NheI fragments into the multiple cloning region of the promoterless luciferase vector pGL3-basic (Promega, Madison, WI). All PCR-amplified fragments were verified by DNA sequencing. 1–7-base pair mutations were introduced into the regions of the promoters, and the noncoding sequence was isolated and subcloned in the EcoRI/SalI sites of the pBluescriptISK vector (Stratagene).

Primer Extension Analysis—32P-labeled primer (10 pmol) and total cellular RNA, isolated from normal cycling NIH3T3 cells, were denatured for 10 min at 65 °C and then incubated for 30 min at 37 °C. Primer extension was carried out in a total volume of 50 μl containing 50 mM Tris, pH 8.3, 75 mM KCl, 10 mM dithiothreitol, 3 mM MgCl2, 400 μM dNTPs, 2 units of RNasin, and 400 units of Moloney murine leukemia virus reverse transcriptase (Life Technologies, Inc.). After incubation for 1 h at 37 °C, the reaction was stopped with EDTA followed by RNase treatment, redissolved, and separated by electrophoresis on a 6% acrylamide, 7% urea gel.

Reverse Transcription PCR—For cDNA synthesis (53), 4 μg of total RNA were annealed to 1 μg of oligo(dT)20 and incubated with 200 units of Moloney murine leukemia virus reverse transcriptase for 1 h at 37 °C in a final volume of 20 μl. One-tenth of the reaction mixture was amplified by 25 cycles of PCR in the presence of 0.5 μCi of [α-32P]dCTP (54). The experimental strategy included the following precautions. (i) The experimental strategy included the following precautions. (i) The number of PCR cycles was kept low to obtain a linear amplification of the PCR products, which was possible by the incorporation of radioactive precursor nucleotides and evaluation by autoradiography and β-ray detection scanning. (ii) All results were standardized using the signal obtained with glyceraldehyde-3-phosphate dehydrogenase, whose expression is independent of cell proliferation. (iii) All experiments were performed with at least two independent cDNA preparations.

Fig. 1. A genomic structure of the murine cdc25B locus. The map was assembled on the basis of the three analyzed phage clones shown below the map. The subcloned fragment used for promoter analysis is depicted at the bottom. kb, kilobases. B, schematic of the cdc25B promoter showing putative protein binding sites. E, E2F site; Sp1, binding site for Sp1 family members; NF-Y site (reverse CCAAT box); TATA, TATA box. C, nucleotide sequence of the proximal promoter region. The major site of transcription initiation was designated position +1 (see also Fig. 3). Functional element (Sp1/3 and NF-Y sites) motifs identified in the present study, as well as the TATA and Inr, are highlighted.
Cell Cycle Regulation of cdc25B Transcription

underlined) was generated by PCR with the primer 5'-AGCTTGTTAAGCTTTCCCACTAGGTCCTTCCCAG-3' and the primer cdc25B NheI (see below). The resulting fragments carrying the mutations were cloned into the KpnI/NheI sites of the promoterless luciferase vector pGL3-basic (Promega) and verified by DNA sequencing.

The following oligonucleotides were used as primers: cdc25B KpnI, 5'-AGCTTGTTAAGCTTTCCCACTAGGTCCTTCCCAG-3'; cdc25B NheI, 5'-GGGCAAAAGCTTTCCCACTAGGTCCTTCCCAG-3'; cdc25B NheI, 5'-GGGCAAAAGCTTTCCCACTAGGTCCTTCCCAG-3'; cdc25B KpnI, 5'-AGCTTGTTAAGCTTTCCCACTAGGTCCTTCCCAG-3'.

Electrophoretic Mobility Shift Assays—Preparation of nuclear extracts and electrophoretic mobility shift assays (EMSA) were performed as described (55, 56) using poly(dI-dC) or poly(dA-dT). The following oligonucleotides were used as probes and/or competitors: cdc25B KpnI, 5'-AGCTTGTTAAGCTTTCCCACTAGGTCCTTCCCAG-3'; cdc25B KpnI, 5'-AGCTTGTTAAGCTTTCCCACTAGGTCCTTCCCAG-3'; cdc25B KpnI, 5'-AGCTTGTTAAGCTTTCCCACTAGGTCCTTCCCAG-3'.

Electrophoresis—Time course of luciferase activity in G0-synchronized NIH3T3 cells after transfection of the B950 construct and serum stimulation. B, kinetics of endogenous cdc25B mRNA expression in serum-stimulated NIH3T3 cells. The analysis was performed by reverse transcriptase-PCR. For comparison, the induction of cdc25B mRNA was also measured.

Fig. 2. Cell cycle regulation of cdc25B transcription. A, time course of luciferase activity in G0-synchronized NIH3T3 cells after transfection of the B950 construct and serum stimulation. B, kinetics of endogenous cdc25B mRNA expression in serum-stimulated NIH3T3 cells. The analysis was performed by reverse transcriptase-PCR. For comparison, the induction of cdc25B mRNA was also measured.

Fig. 3. Mapping of the 5' of cdc25B mRNA by primer extension in normally cycling NIH3T3 cells. As a negative control, yeast tRNA was used. A sequencing reaction was run alongside (lanes labeled G, A, T, C) to be able to accurately determine the nucleotide positions.

Fig. 4. Delineation of functionally important regions in the cdc25B promoter. Terminally truncated cdc25B promoter-luciferase constructs were analyzed in transient expression assays in both quiescent (G0) and normally growing (N) NIH3T3 cells. Values are given as relative luciferase activities normalized to 100 for the longest promoter construct (−950) in normally growing cells.
CGGCCGCTGCCGCTGTTATTTTTCGAATATATAAGGAG-3'; 64/−20 3mCCRR, 5'-GGGCTCGGCCGCTGCCGCTGTTATTTTATCATATATAAAGGAG-3'; ns, 5'-GAAATAAGTATTCTGATTCTTTGAGACA-3'. Shown are the top strand oligonucleotides. For radioactive labeling by filling in with [32P]dCTP, an additional G was added to the 5'-end of the bottom strand oligonucleotides. Underlined letters represent mutated bases.

**RESULTS**

**Cloning of the Mouse cdc25B Promoter**—A mouse embryo genomic DNA library was screened with an oligonucleotide representing the mouse cdc25B coding region. Several recombinant phage spanning ~30 kilobases of genomic DNA were isolated and mapped (Fig. 1A). One phage clone (designated III in Fig. 1A) was used to subclone a 1.1-kilobase fragment representing the sequence 5' to the translation start codon. This fragment (B950) was linked to the firefly luciferase gene and transfected into NIH3T3 cells to test whether the isolated promoter fragment was functional in a transient expression assay. As shown in Fig. 2A, B950 was cell cycle-regulated after serum stimulation of cells that had been synchronized in G0. Thus, hardly any luciferase activity was detectable in G0 cells and stimulation was carried out for the indicated times with 10% fetal calf serum. The cells were then treated with 0.2% DMS for 2 min. After DMS treatment, cells were washed three times with cold phosphate-buffered saline, and the DNA was isolated. As reference, NIH3T3 genomic DNA was methylated in vitro with 0.2% DMS for 10–30 s. Piperidine cleavage was performed as described. Genomic DNA (3 μg) was used for ligation-mediated PCR as described. The Stoffel fragment of Taq polymerase (PerkinElmer Life Sciences) was used instead of the native enzyme. Samples were phenol-extracted and ethanol-precipitated before primer extension with 32P-labeled primers.

**Sequence of B950**—The nucleotide sequence of B950 was determined for both strands (GenBankTM accession number AJ296019). The most relevant part of the sequence, as determined below, is shown in Fig. 1B. Inspection of the sequence revealed a match with a canonical TATA box motif 190 nucleotides 5' to the ATG (Fig. 1C). A single transcription start site cluster was identified by primer extension analysis ~30 nucleotides downstream of this motif and overlapping with an Initiator (Inr) consensus sequence (Figs. 1C and 3). Although we cannot formally rule out the presence and absence of antibodies specific for the A subunit of NF-Y (aNF-Y A). No effect was seen with irrelevant anti-serum (data not shown). Competitors were identical to the respective probes (self-competition) or represented a bona fide NF-Y site (MHC), a GT box, or the mutated cdc25B NF-Y site (MY).

**Identification of functionally important elements in the cdc25B promoter.** cdc25B promoter-luciferase constructs with point mutations in defined elements (E boxes, NF-Y site) were analyzed in transient expression assays in both quiescent (G0) and normally growing (N) NIH3T3 cells. Values are given as relative luciferase activities normalized to 100 for the wild-type construct (~950) in normally growing cells. N/G0 gives the factor of cell cycle regulation. Sites are labeled as in Fig. 1B.

**NF-Y site**

![NF-Y site diagram](Image 129x569 to 476x729)

![Fig. 5](http://www.jbc.org/)

![Fig. 6](http://www.jbc.org/)

**Structure of the Mouse cdc25B Promoter**—The nucleotide sequence of B950 was determined for both strands (Gen-
formal possibility that the *cdc25B* gene contains additional initiation sites outside the region analyzed, these observations strongly suggest that a TATA box and/or an Inr element direct the initiation of transcription and define the transcriptional start site. The A within the Inr motif was therefore designated position 1 (see Fig. 1C). A search for potential regulatory sites revealed the presence of additional putative transcription factor binding sites: two E boxes (−947 and −800), three E2F sites (−232, −58, and −50), five Sp1 sites (−570, −217, −200, −105, and −95), and an NF-Y binding site (−70).

**Delineation of Functional Regions in the Mouse *cdc25B* Promoter by Truncation Analysis**—To identify functionally relevant regions in *cdc25B* promoter, a series of terminal truncations was generated from the B950 construct (−950/1167) and analyzed for expression in G0 versus normally cycling cells (N) (Fig. 4). This analysis led to the following conclusions. (i) The terminal deletion of 10 nucleotides, which removes a potential E box, led to an increase in transcriptional activity of 40% but had no effect on cell cycle regulation. Truncation of the adjacent fragment spanning positions −2980 to −2768, which harbors another potential E box, had no detectable effect on transcriptional activity or cell cycle regulation. (ii) The region from −340 to −250 seems to have a negative effect on transcriptional activity. However, because no putative binding sites could be identified in this region, and there was no effect on cell cycle regulation, we did not pursue this finding. (iii) Further deletion of a fragment spanning nucleotides −250 to −223 and harboring a potential E2F site had no detectable effect. (iv) Truncation of a fragment spanning positions −223 to −180, which contains two potential Sp1 sites, led to a clear reduction in transcriptional activity. This was further decreased by truncation of the adjacent region spanning nucleotides −180 to −87, which harbors two more potential Sp1 sites. The loss of these
four potential Sp1 sites led to a total decrease in transcriptional activity of 60%, with only a marginal effect on cell cycle regulation. (v) The terminal deletion of an additional 20 nucleotides resulted in a further drop in transcriptional activity but also led to a clear decrease in cell cycle regulation, indicating that this promoter region, which harbors a potential NF-Y site, is of particular functional relevance. (vi) Further truncations had no additional effect on cell cycle regulation, presumably because these constructs all lacked the NF-Y site.

Identification of Functional Upstream Elements in the Mouse cdc25B Promoter—To confirm and extend the findings obtained by promoter truncation, the putative E boxes and NF-Y binding site were altered by point mutations, and the functional consequences were analyzed in transient transfection assays. The proximal potential E2F sites were not included in this analysis because no binding of E2F-1, E2F-3, or E2F-4 to the cdc25B promoter could be detected in EMSA using either normal NIH3T3 cells or retrovirally transduced cells overexpressing B-myb (5, 15). Further evidence that NF-Y interacts with the C/EBP or NF-I/CTF (59), were used (data not shown). To obtain any competition. Likewise, no effect on complex formation was observed. Neither the GT box nor the mutated site both for transcriptional activity and cell cycle regulation.

Interaction of NF-Y and Sp1/Sp3 with the Mouse cdc25B Upstream Activating Sequence—To investigate protein interactions at the potential NF-Y site in the cdc25B promoter, we performed EMSAs with nuclear extracts from normally cycling NIH3T3 cells. A synthetic oligonucleotide encompassing this element was used as a probe, and competitors representing either the same site (self-competition), a bona fide NF-Y site from the MHC class II promoter (Ea-Y) (58), an Sp1 binding site (GT box), or a mutated cdc25B element (MY) were also used. As shown in Fig. 6, only the former two oligonucleotides were able to prevent the formation of a DNA-protein complex. Neither the GT box nor the mutated cdc25B element showed any competition. Likewise, no effect on complex formation was seen when binding sites for other CAAT box-binding factors, i.e. C/EBP or NF-I/CTF (59), were used (data not shown). To obtain further evidence that NF-Y interacts with the cdc25B promoter, we analyzed the effect of a monoclonal antibody (aNF-Y A) against the A subunit of NF-Y (kindly provided by D. Muth) (58). This antibody led to the expected supershift of the observed complex (58, 59). Taken together, these data clearly suggest that the protein complex interacting with the cdc25B site is NF-Y.

Similar experiments were performed to analyze protein binding to the four functionally relevant Sp1 sites at positions −217, −200, −105, and −95. EMSAs were performed using four different probes representing these sites in conjunction with a specific (self) or nonspecific competitor (unrelated sequence) and antibodies specific for Sp1 or Sp3 (kindly provided by G. Suske, IMT, Marburg, Germany) (60, 61). The data in Fig. 7 clearly show that all four sites specifically interact with Sp1 and Sp3, leading to the formation of the expected com-
plexes (60).

Identification of a Proximal Repressor Element—Finally, we scanned the proximal promoter for the presence of additional sites that might play a role in cell cycle regulation. Toward this end, we introduced point mutations into this region in the context of an otherwise intact promoter fragment (−223/+167 construct). Construct 2mCCR harbors two mutations at positions −32 and −33, whereas construct m30G is mutated at position −30, i.e. the first nucleotide of the TATA motif. As shown in Fig. 8, both these mutations led to a 3- to 4-fold increased activity in G0 cells, resulting in a 50–60% loss in cell cycle regulation. These results indicate that this region of the promoter functions as a cell cycle-regulated repressor. Previous studies have shown that other S/G2 genes are regulated by two promoter functions as a cell cycle-regulated repressor. These results indicate that this region of the promoter, its sequence (TATATAA) is functional in the cdc25B promoter, but the precise underlying mechanism remains to be investigated.

Another interesting aspect relates to the fact that the CCRE apparently overlaps the TATA motif. Although there is no formal proof at present that the putative TATA element is functional in the cdc25B promoter, its sequence (TATATAA) exactly fits that of a canonical TATA box, and its spacing relative to the transcriptional start site and the putative Inr element is within the expected range. This raises the intriguing possibility that a CCRE-interacting repressor functions by interfering with the basal transcriptional machinery, e.g. by inhibiting the assembly of a functional initiation complex. Future analyses will have to address these mechanistic questions in detail. The present study provides the basis for such studies.

Acknowledgments—We are grateful to R. Bernards for retrovirally transduced cells overexpressing specific E2F family members and to Dr. M. Krause for synthesis of oligonucleotides.

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Cell Cycle Regulation of the Murine cdc25BPromoter: ESSENTIAL ROLE FOR NUCLEAR FACTOR-Y AND A PROXIMAL REPRESSOR ELEMENT
Kathrin Körner, Valérie Jérôme, Thorsten Schmidt and Rolf Müller

J. Biol. Chem. 2001, 276:9662-9669.
doi: 10.1074/jbc.M008696200 originally published online December 4, 2000

Access the most updated version of this article at doi: 10.1074/jbc.M008696200

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