A Role of the C-terminal Part of p44 in the Promoter Escape Activity of Transcription Factor IIIH*

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The p44 subunit plays a crucial role in the overall activity of the transcription/DNA repair factor TFIIH: on the one hand its N-terminal domain interacts with and regulates the XPD helicase (1, 2); on the other hand, as shown in the present study, it participates with the promoter escape reaction. Mutagenesis along with recombiant technology using the baculovirus/insect cell expression system allowed us to define the function of the two structural motifs of the C-terminal moiety of p44: mutations within the C4 zinc finger motif (residues 291–308) prevent incorporation of the p62 subunit within the core TFIIH. Double mutations in the RING finger motif (residues 345–385) allow the synthesis of the first phosphodiester bond by RNA polymerase II, but prevent its escape from the promoter. This highlights the role of transcription factor IIIH in the various steps of the transcription initiation process.

Accurate transcription of class II genes requires formation of a preinitiation complex composed of RNA polymerase II (RNA pol II) and several general transcription factors including TFIIH (3). TFIIH, also involved in DNA repair and cell cycle control (4), plays a central role in the initiation of transcription due to its numerous enzymatic activities: the 3'-5' XPB helicase opens DNA around the promoter (5–7), whereas the 5'-3' XPD helicase is more likely devoted to the opening of DNA around a damage (8, 9). In transcription the role of XPD seems to be more structural, since it allows the anchoring of CAK (cdk-activating kinase) complex to the core TFIIH, for optimal phosphorylation of RNA pol II (10), and nuclear receptors (11–13). p62, p52, and p34 have no defined functions, whereas p44 plays a crucial role in the overall activity of the transcription/DNA repair factor TFIIH: on the one hand its N-terminal domain interacts with and regulates the XPD helicase (1, 2); on the other hand, as shown in the present study, it participates with the promoter escape reaction. Mutagenesis along with recombinant technology using the baculovirus/insect cell expression system allowed us to define the function of the two structural motifs of the C-terminal moiety of p44: mutations within the C4 zinc finger motif (residues 291–308) prevent incorporation of the p62 subunit within the core TFIIH. Double mutations in the RING finger motif (residues 345–385) allow the synthesis of the first phosphodiester bond by RNA polymerase II, but prevent its escape from the promoter. This highlights the role of transcription factor IIIH in the various steps of the transcription initiation process.

Experimental Procedures

Construction of Recombinant Baculoviruses Expressing TFIIH Subunits—Baculoviruses expressing the TFIIH subunit were constructed in the pVL1392 or pACAB4 vectors (PharMingen). The cDNAs coding for XPB, XPD, p62, p52, p34 subunits of TFIIH, and for cyclin H, MAT1 subunits of the CAK complex, were inserted into the pVL1392 expressing vector (22, 23). Each p44 single mutant was obtained by PCR mutagenesis using two pairs of oligonucleotides on a wild type plasmid containing the p44 open reading frame: CCTCCTGCTAGTC-AAGTTTCTGAA in combination with either GACACTGTGGAGCGAAAATAGCCCT, CCAAGATAGCCAGCGTTTCTATACGTAA, CGTACATCAATAAGCAGGAAAAGTTTTCA, GGCACACAGCAGAACTACATATGAT, AGAAGAACTTTTGGGCACACGACA, GCAACAGTGATGGACTACAGC-CAACAAAAAC, AGCCAGGCAAGACAGGCTAGAACGAACT, or GAATTCAAGCCAGGGGCACTGTTAG (to synthesize the 5' part of p44 C291A, C308A, C345A, C360A, C363A, H376A, H380A, and C382A, respectively) and CGGGTCCCAGGAAAGGATCCTCA in combination with either AGGCTATTCGCGCTCCACAGTTCG, TGGATTAGTAAATC- GCTCTGTCTTCTTTGG, GGAGAAAGATTGTTCTTATGGAAGGC, CATGTTTAATGGCTGCTGGTGGCC, TGGCTGTTGGCGCACAAAGT-TTCT, GATTGGTTGCTGATTTCCTACACTGTTCG, GATTTCTCTGCGTTGCTGCGG, CAGACGTTTGCTGCTGCTGTTAGTGTGTGGTTTGAAGTTTCTGAGGTTTCTGTT, or CTACACTGCTGGCTGCTGCTGTTATCCCAGGAGAAGATCTGTTAG (to synthesize the 3' remaining part of these p44 mutants). The double mutants (C360A/C363A and H376A/H380A) were obtained by the same PCR procedure. The mutated fragment was then substituted to the wild type open reading frame using the BamHI/HindIII restriction sites. Both XPB and cyclin H were fused to a 6-histidine tag at their N-terminal extremity. The resulting vectors were transfected with linearized baculovirus DNA (PharMingen) in S9 cells (Spodoptera frugiperda 9). The recombinant viruses were purified from isolated plaques, and viral stocks were prepared by a three-step growth amplification. The calculation of the multiplicity of infection of each virus stock solution as well as the determination of the best ratio for the expression of each subunit allowed us to optimize the coexpression of the different subunits stoichiometrically.

Purification of TFIIH Complexes—Cobalt chelate affinity purification: S9 cells were infected with combinations of baculoviruses expressing the different subunits of TFIIH as described previously (23). 48 h after infection, cells were collected, washed, and dosed in buffer A (20 mM Tris-HCl, pH 7.8, 20% glycerol, 150 mM NaCl) containing 0.1% Nonidet P40, 5 mM β-mercaptoethanol, 0.5 mM phenylmethylsulfonyl...
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fluoride, and 1 × protease inhibitor mixture, and then the extracts were centrifuged at 14,000 × g during 30 min. The supernatants were first applied on a heparin–Ultrogel column. After a five-resin volume wash with buffer A containing 0.4 mM NaCl, the proteins were eluted with this buffer containing 0.5 mM NaCl. After a 3-h dialysis against buffer B (50 mM Tris–HCl, pH 7.8, 20% glycerol) containing 50 mM KCl fractions were incubated for 1 h at 4 °C with 1/50 fraction volume of cobalt chelate affinity resin (CLONTECH). After a 20-volume wash with buffer B containing 10 mM imidazole, proteins were eluted with 100 mM EDTA and dialyzed against buffer C (50 mM Tris–HCl, pH 7.8, 20% glycerol, 0.1 mM EDTA, 0.5 mM dithiothreitol) containing 50 mM KCl for 3 h at +4 °C. Recombinant CAK was purified as described previously (22).

Immunopurification of rIIH6/p44-C291A and rIIH6/p44-C308A—Infected Sf9 cell extracts were incubated overnight at 4 °C with protein A–agarose beads (Amersham Pharmacia Biotech) cross-linked to p44 antibody. The resin was washed twice with buffer C containing 150 mM KCl, and the elution was performed by incubating the resin 6–7 h at 4 °C in 0.15 ml of the same buffer in the presence of 2 mg/ml p44 epitope peptide and 0.2 mg/ml insulin.

Enzymatic Activities of TFIIH—The run-off transcription assay was performed by incubating all general transcription factors (TFIIF-A, -B, -E, -F, -H, -TBP), RNA pol II, and the adenovirus major late promoter (MLP) for 15 min at 25 °C in 50 mM Tris–HCl, pH 7.8, 10% glycerol, 1 mM EDTA, 0.5 mM dithiothreitol, 50 mM KC1, and 5 mM MgCl2 (24).

Transcription was then carried out for 45 min at 25 °C in the presence of ribonucleotides including radiolabeled CTP (Amersham Pharmacia Biotech). RNA transcripts were resolved on 5% polyacrylamide/urea gel and analyzed by autoradiography. The abortive initiation as well as the ATPase and the helicase assays were done as described earlier (5, 25).

The promoter escape reaction was done as described in Ref. 25; briefly RNA pol II and the basal transcription factors, including mutants or wild type rIIH6, were incubated with a premelted DNA (–8/+2) for 30 min at 28 °C in the presence of 0.4 mg/ml bovine serum albumin, 5 mM MgCl2, and dinitocleotide ApG as an initiate substrate. After preinitiation complex formation the RNA synthesis was initiated upon addition of CTP, GTP, in–32PUTP, cordycepin, and 6.5 mM MgCl2 for 30 min at 28 °C. The samples were applied on a 12% polyacrylamide/urea gel and analyzed by overnight autoradiography.

RESULTS

Mutations in the Cysteine-rich Domain of p44 Cause Transcriptional Defects—The C-terminal part of p44 binds three zinc atoms through two independent modules: the first contains a C4 zinc finger motif (from aa 291 to aa 308) and a zinc through Cys-291, Cys-294, Cys-305, and Cys-308. The second module is a RING finger motif (from aa 345 to aa 385) and contains two zinc through Cys-345, Cys-348, Cys-368, and Cys-371 and through Cys-360, Cys-363, and as strongly suggested in Ref. 26, through two of these four residues: His-376, Cys-382, and Cys-385 (Fig. 1). Whereas the first motif does not exhibit a typical three-dimensional structure, NMR spectroscopy (26) of p44 (residues 345–385) determined by NMR spectroscopy (26). The cysteines and histidines that are the best candidate for the chelation of the two zinc ions are indicated. Cysteines 382 and 385 are also indicated.

To analyze the role of the C4 zinc finger motif, we have mutated cysteines to alanine at position 291 and 308 (Fig. 1A). Immunopurified rIIH6 complexes, carrying wild type or mutated p44, were washed at 150 mM salt and further analyzed by Western blotting (Fig. 2A, lanes 1–3). Although all the rIIH6 subunits are present in the baculoviruses extracts (data not shown), both mutated recombinant IIH6/p44-C291A and IIH6/p44-C308A complexes lack p62 (lanes 2 and 3). When either p44-C291A or p44-C308A are coexpressed with p62, they are able to interact with this latter one (data not shown). It is worthwhile to mention that even at low salt concentration (150 mM), both C291A and C308A mutations in p44 do not allow integration of p62 within the core TFIIH. Furthermore, as expected in the absence of one TFIIH subunit, none of these two mutated rIIH6 complexes exhibit any transcriptional activity (data not shown).

We then focused our attention on the RING finger motif and generated alanine point mutations in the following residues: Cys-345, Cys-360, Cys-363, His-376, His-380, and Cys-382 (Fig. 1A). The rIIH6 complexes carrying these p44 mutations were purified from infected cell extracts and further analyzed by Western blotting. None of the p44 mutations were found to be expected in the absence of one TFIIH subunit, none of these two mutated rIIH6 complexes exhibit any transcriptional activity (data not shown).
cyclin H subunit to bind to a cobalt affinity column and was added in the reaction to stimulate the RNA synthesis. The amounts of rIIH6 were adjusted on the basis of p62 and p52 content, according to Western blot analysis. The transcriptional activity of the rIIH6 complexes was not modified by the additional factors, in addition to RNA pol II. The 309-nucleotide length transcription run-off assay (lanes 1–9) containing the MLP, the basal transcription factors, in addition to RNA pol II. The 309-nucleotide length transcript is indicated by an arrow.

We have expressed XPB, XPD, p62, or p34 and either wild type or mutated p44 (as indicated at the top of each panel), plus XPD, including mutated p44 (as indicated at the top of each panel), and further purified. Western blots of rTFIIH containing p44 subunit mutated in the C4 zinc finger motif (A) or in the RING finger motif (B) are presented. rIIH6/p44 (wt, C345A, C360A, C363A, H376A, H380A, C360A/C363A, and H376A/H380A) were tested in an in vitro transcription run-off assay (lanes 1–9) containing the MLP, the basal transcription factors, in addition to RNA pol II. The 309-nucleotide length transcript is indicated by an arrow.

FIG. 2. p44 mutations are detrimental for TFIIH subunits composition and transcription activity. Sf9 insect cells were coinjected with baculoviruses overexpressing TFIIH subunits of the core TFIIH, plus XPD, including mutated p44 (as indicated at the top of each panel), and further purified. Western blots of TFIIH containing p44 subunit mutated in the C4 zinc finger motif (A) or in the RING finger motif (B) are presented. rIIH6/p44 (wt, C345A, C360A, C363A, H376A, H380A, C360A/C363A, and H376A/H380A) were tested in an in vitro transcription run-off assay (lanes 1–9) containing the MLP, the basal transcription factors, in addition to RNA pol II. The 309-nucleotide length transcript is indicated by an arrow.

The N-terminal part of p44 interacts with and regulates the XPD helicase within TFIIH, giving rise to its optimum transcriptional activity. To investigate whether p44 C-terminal mutations do not affect the interaction with the XPD helicase, we carried out immunoprecipitation on Sf9 extracts coinjected with baculovirus overexpressing XPD and either p44/C291A, p44/C308A, p44/C360A/C363A, or p44/H376A/H380A complexes were analyzed for their ATPase activity. B, the four mutated p44, in addition to XPDwt, were overexpressed in Sf9 insect cells and immunoprecipitated using antibodies directed toward XPD; the precipitated proteins were analyzed for their protein content (WB) and for their helicase activity. C, increasing amounts of rIIH6/p44 wt, rIIH6/p44/C308A/C363A, and rIIH6/p44/H376A/H380A complexes were analyzed for the first phosphodiester bond formation (lower panel, synthesis of CpaP) and promoter escape (upper panels, synthesis of 17 and 31 nucleotides). In A and C HeLa TFIIH (lane 11 and 8, respectively) are used as a control.

Each p44 mutated rIIH6 complex with [α-32P]ATP as a substrate. All of the complexes exhibit an ATPase activity similar to that of the wild type (Fig. 3A, compare lanes 1–2 with lanes 3–10).

The C-terminal Moiety of p44 Is Involved in Promoter Escape—p44 is a central protein in the core TFIIH complex and has been shown to interact with XPB, XPD, p62, and p34 (20, 21). We have expressed XPB, XPD, p62, or p34 and either wild type or mutated p44 and subsequently immunopurified the binary complexes using p44 antibody, to demonstrate that the p44 double point mutations do not modify the interactions with the subunits of the core TFIIH (data not shown). Having shown that double mutations in the RING finger motif were detrimental for transcriptional activity of rTFIIH, we were then wondering if it could affect its proper enzymatic activities. ATPase activity was then assayed in the presence of DNA by incubating each p44 mutated rIIH6 complex with [α-32P]CTP and CAK. Both rIIH6
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mutated complexes, when compared with the wild type rIIH6, allow the association of CTP to the CpA dinucleotide to form a three-nucleotide-long product, meaning that the p44 RING finger mutations, detrimental for the run-off transcriptional activity of TFIIH, do not prevent the first phosphodiester bond formation (Fig. 3C, lower panel, compare lanes 2–3 with lanes 4–7). This reaction is highly specific, since in the absence of either TFIIH or TBP, which initiate the formation of the preinitiation complex on the TATA-box, no synthesis occurs (Fig. 3C, lower panel, compare lane 8 with lanes 9 and 1, respectively).

We then investigated the ability of rIIH6/p44-C360A/C363A and rIIH6/p44-H376A/H380A to allow the escape of RNA pol II from the promoter. This assay monitors the escape by using a premelted DNA template around the start site (from the position −8 to position +2), which circumvents the promoter opening step (5, 7) and consequently the requirement of a functional XPD helicase. To allow the accumulation of products that escape the promoter, we employed the chain terminating ATP analog cordycepin, which stops transcription elongation at thymidines at position +17 and +31 (see Ref. 25 and “Experimental Procedures”). The reaction is TFIIH-dependent and gives rise to 17- and 31-oligonucleotide length products (Fig. 3C, upper panel, compares lane 8 with lane 9). Both rIIH6/p44-C360A/C363A and rIIH6/p44-H376A/H380A do not allow the RNA pol II to escape from the promoter to synthesize longer transcripts (Fig. 3C, upper panel, compare lanes 2 and 3 with lanes 4 and 5 and lanes 6 and 7, respectively). Together these results demonstrate for the first time the implication of p44, and more particularly its RING finger motif in the promoter escape step.

DISCUSSION

It is now accepted that TFIIH joins the promoter late during the formation of the stable preinitiation complex. Once part of this closed and inactive preinitiation complex, and upon addition of ATP as a source of energy (27, 28), the XPD helicase of TFIIH will promote the opening of the promoter around the start site, the first phosphodiester bond formation, and further the promoter escape (5, 7, 29) and see also Refs. 30 and 31). XPD, the second helicase of TFIIH, does not play a role in the opening step, since present or not, mutated or not in its ATP binding site, RNA synthesis, although at a lower level, might occur. Rather it was suggested that it is its physical presence that is required for optimal RNA synthesis in addition to its ability to maintain the CAK complex within TFIIH. Indeed by binding on the one hand MAT1 and on the other hand p44, XPD likely bridges the CAK subcomplex to the core TFIIH (1, 32).

The role of p44, as an essential subunit of the core TFIIH, was further sustained by the discovery that a mutation in its yeast counterpart (SSL1) conferred UV sensitivity due to a nucleotide excision repair defect (18). It was then found that mutations either in the N-terminal moiety of p44 or in the C-terminal end of XPD, by preventing their interaction and thus optimal XPD helicase activity, lead to a destabilization of TFIIH. Besides a role as the regulatory subunit of XPD, p44 presents in its C-terminal moiety a highly conserved cysteine-rich domain, which was defined to contain a RING finger motif by NMR studies (26). The present study details the role of the C-terminal part of p44.

First, mutations in the C4 zinc finger of p44 completely decrease in the interaction between the mutated p44 and p62, since these proteins coimmunoprecipitate when they are coexpressed in insect cells. It is most likely due to a specific role of this C4 zinc finger motif in the structure of TFIIH, since first a complete depletion of p44, and second mutations in the RING finger motif, do not prevent the incorporation of p62 within TFIIH (this study and Ref. 23).

Second C360A/C363A and H376A/H380A double mutations in p44 inhibit TFIIH transcription activity. It is worthwhile to notice that the single mutation of one of these four residues has no effect on the basal transcription activity of TFIIH, indicating that chelation of a zinc atom by three amino acids (instead of four) is sufficient to maintain the RING finger motif of p44. Further investigations of the transcription reaction show that these double mutations in p44, although they allow the first phosphodiester bond formation, inhibit further RNA synthesis by RNA pol II, emphasizing the crucial role of the RING finger motif in the promoter escape process. Additional experiments have shown that this is not due to a defect in the XPD helicase activity within TFIIH (the present study) nor to the inability of cdk7 to phosphorylate the largest subunit of RNA pol II (data not shown). Indeed, CAK was still able to stimulate all the transcription reaction, when added to the rIIH6 complexes, showing that it is fully integrated within TFIIH upon XPD/p44 interaction. We can then assume that the defect of promoter clearance observed for the double RING finger mutants is not due to a defect of CAK activity, but rather to an inaccurate positioning of an other subunit of TFIIH, such as XPB. Indeed, we cannot exclude that p44 mutations might also prevent some protein-protein interaction in which the RING motif is required (26).

To conclude, we can hypothesize that p44 could allow the slipping of TFIIH on the DNA during the promoter escape knowing that TFIIH is associated with the RNA pol II during the synthesis of the first 40–50 nucleotides (see Ref. 33 and for a review, see Ref. 34). Whether or not the RING finger motif of p44 directly binds some transcription factors or the promoter, as suggested by cross-linking experiments3 or by gel shift assays4, remains to be further investigated. Preliminary experiments have illustrated interactions between p44 and other basal transcription factors.

Acknowledgments—We are grateful to F. Coin for fruitful discussions, to S. Fribourg and A. Poterszman for discussions on the RING finger structure, and to J. Bradsher for help in setting up the promoter escape and first phosphodiester bond assays. We also thank I. Kolb-Cheney and J. L. Weickert for providing baculovirus-infected cells.

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J. Biol. Chem. 2001, 276:27693-27697.
doi: 10.1074/jbc.M102457200 originally published online April 23, 2001

Access the most updated version of this article at doi: 10.1074/jbc.M102457200

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