The Xeroderma Pigmentosum Group C Protein Complex
XPC-HR23B Plays an Important Role in the Recruitment of Transcription Factor IIH to Damaged DNA*

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The xeroderma pigmentosum group C protein complex XPC-HR23B was first isolated as a factor that complemented nucleotide excision repair defects of XP-C cell extracts in vitro. Recent studies have revealed that this protein complex plays an important role in the early steps of global genome nucleotide excision repair, especially in damage recognition, open complex formation, and repair protein complex formation. However, the precise function of XPC-HR23B in global genome repair is still unclear. Here we demonstrate that XPC-HR23B interacts with general transcription factor IIIH (TFIIH) both in vivo and in vitro. This interaction is thought to be mediated through the specific affinity for XPC for the TFIIH subunits XPB and/or p62, which are essential for both basal transcription and nucleotide excision repair. Interestingly, association of TFIIH with DNA was observed in both wild-type and XP-A cell extracts but not in XP-C cell extracts, and XPC-HR23B could restore the association of TFIIH with DNA in XP-C cell extracts. Moreover, we found that XPC-HR23B was necessary for efficient association of TFIIH with damaged DNA in cell-free extracts. We conclude that the XPC-HR23B protein complex plays a crucial role in the recruitment of TFIIH to damaged DNA in global genome repair.

Nucleotide excision repair (NER) is the primary pathway for removal of lesions from DNA and is conserved across a wide range of species and from prokaryotes to eukaryotes. Studies of prokaryotic NER have shown that the NER pathway is controlled by phased enzymatic reactions (1, 2). Recently, analyses of NER-deficient yeast, rodent, and human mutant cells have been shown to be regulated by phased enzymatic reactions (1, 2). Recently, analyses of prokaryotic NER have shown that the NER pathway is conserved across a wide range of species and from prokaryotes to eukaryotes. XPC-HR23B was first isolated as a factor that complemented nucleotide excision repair defects of XP-C cell extracts in vitro. Recent studies have revealed that this protein complex plays an important role in the early steps of global genome nucleotide excision repair, especially in damage recognition, open complex formation, and repair protein complex formation. However, the precise function of XPC-HR23B in global genome repair is still unclear. Here we demonstrate that XPC-HR23B interacts with general transcription factor IIIH (TFIIH) both in vivo and in vitro. This interaction is thought to be mediated through the specific affinity for XPC for the TFIIH subunits XPB and/or p62, which are essential for both basal transcription and nucleotide excision repair. Interestingly, association of TFIIH with DNA was observed in both wild-type and XP-A cell extracts but not in XP-C cell extracts, and XPC-HR23B could restore the association of TFIIH with DNA in XP-C cell extracts. Moreover, we found that XPC-HR23B was necessary for efficient association of TFIIH with damaged DNA in cell-free extracts. We conclude that the XPC-HR23B protein complex plays a crucial role in the recruitment of TFIIH to damaged DNA in global genome repair.

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1 The abbreviations used are: NER, nucleotide excision repair; RPA, replication protein A; TFIIH, transcription factor IIIH; PCR, polymerase chain reaction; PCNA, proliferating cell nuclear antigen; PAGE, polyacrylamide gel electrophoresis; GST, glutathione S-transferase; ss, single-stranded; ds, double-stranded; N-AAA, N-acetoxy-2-acetyl-2-aminofluorene; r, recombinant; AMP-PNP, adenosine 5'-[β,γ-imino] triphosphate.
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EXPERIMENTAL PROCEDURES

Cell Culture and Preparation of Whole Cell Extracts—Human 293, XP7CASV (group A), and XP4PASV (group C) cells were grown at 37 °C in Dulbecco’s modified Eagle’s medium (Nissui) supplemented with 10% fetal bovine serum. HeLa cells were grown in suspension at 37 °C in Dulbecco’s modified Eagle’s medium supplemented with 10% bovine serum. Whole cell extracts were prepared as described previously (21).

Direct Interaction between XPC-HR23B—Untagged (HR23B), His-tagged (HR23B-His₃), and GST-tagged (GST-HR23B) HR23B constructs were expressed in Escherichia coli and purified as described previously (22, 23). Recombinant XPC (rXPC) was expressed using a baculovirus expression vector pGEX-6P-4 (28) subcloned into the E coli site of pB8 as template. PCR reactions (35 cycles) were carried out in a total volume of 50 μl containing 10 ng of the template DNA, 10 μM KCl, 20 μM Tris-HCl (pH 8.8), and 10 μM MgSO₄, 10 μM dNTPs, 0.1% Triton X-100, 100 μg/ml bovine serum albumin, 200 μM dNTPs, 20 μM of each oligonucleotide primer, and 2.5 units of Pfu DNA polymerase (Stratagene). For immunoblotting, proteins separated on SDS gels were electrotransferred onto polyvinylidene difluoride membranes (Immobilon-P, Millipore) at 8 V/cm 15 h in ice-cold transfer buffer (50 mM Tris, 38.4 mM glycine, 0.1% SDS, and 15% methanol). The membranes were successively incubated in blocking buffer (1% blocking reagent (Roche Molecular Biochemicals) in 0.1% maleic acid (pH 7.5), 150 μM NaCl), antibody in blocking buffer, and finally anti-rabbit or anti-mouse F(ab′)₂ antibody conjugated with horseradish peroxidase (Amersham Pharmacia Biotech). Detection was carried out with SuperSignal Substrate (Pierce) according to the manufacturer’s instructions. Protein concentrations were measured as described previously (30) using the Bio-Rad Protein Assay reagent and bovine serum albumin as a standard.

RESULTS

Direct Interaction between XPC and TFIIH—Of the mammalian NER factors identified to date, only TFIIH has been suggested to interact with XPC (31–33), although conclusive evidence of such interaction has been lacking. We performed co-immunoprecipitation experiments with NER-proficient human cell extracts and an antibody raised against cyclin H, which is one of the components of TFIIH. As shown in Fig. 1, not only XBP and p62, but also multiple subunits of TFIIH were detected in the fraction precipitated by anti-cyclin H antibody (lanes 3 and 4) but not with the control antibody (lane 2). In the other hand, no clear evidence to imply the interaction between TFIIH and XPA nor RPA was obtained. This result indicated that XPC interacted with TFIIH in the whole cell extracts; to examine
whether the interaction was direct or mediated by other proteins or DNA present in the cell extracts, co-precipitation experiments were performed using purified proteins. TFIIH purified from HeLa nuclear extracts (Fig. 2C) was incubated with glutathione-Sepharose beads bound to GST-rHR23B fusion protein that had been preincubated with or without rXPC. Co-precipitation of TFIIH with the Sepharose beads was assessed by immunoblotting with anti-p62 antibody. As shown in Fig. 2A, p62 was detected in the precipitate fraction in an XPC-dependent manner (compare lane 4 with lanes 2 and 3), suggesting that XPC alone or the XPC-HR23B complex interacted directly with purified TFIIH. This XPC-dependent interaction was also observed using rHR23B-conjugated Sepharose (Fig. 2B, lane 3). Therefore, we conclude that the XPC-HR23B complex interacts directly with TFIIH in vitro and that XPC is indispensable for this specific interaction. Next, a series of pull-down experiments were performed using the GST-tagged TFIIH subunits to assess which subunit of TFIIH interacted with the XPC-HR23B complex and whether HR23B was necessary for this interaction. As shown in Fig. 3, both the purified XPC-HR23B complex (A) and free rXPC (B) were found to bind XPC and p62. In contrast, rHR23B, rRPA, and rPCNA were not co-precipitated with any of the TFIIH subunits (C–E). These results indicate that the interaction between the XPC-HR23B complex and TFIIH is mediated through specific protein-protein binding (XPC-XPB and/or XPC-p62) and that HR23B is not essential for this interaction.

**XPC-HR23B Is Necessary to Precipitate TFIIH Efficiently with dsDNA**—To examine the relevance of the interaction between XPC-HR23B and TFIIH, several precipitation experiments were performed. The XPC-HR23B complex has a high affinity for both ssDNA and dsDNA (14, 17, 23). Thus, not only XPC-HR23B but also those proteins that interact with this complex or which associate with DNA might be expected to be precipitated from whole cell extracts by DNA-cellulose. We therefore examined the presence of not only XPC and TFIIH but also XPA and RPA, which have been characterized as DNA binding NER factors, in DNA-cellulose bound fractions. Because it has been reported that ATP is necessary to form the repair protein complex on DNA (19, 20, 34), we also examined the ATP dependence of the precipitation of TFIIH by DNA-cellulose. XPC, p62, XPA, and RPA32 were detected in the dsDNA-cellulose bound fraction of NER-proficient 293 whole cell extracts, as expected (Fig. 4, lanes 3 and 5). Interestingly, ATP was not required for efficient precipitation of TFIIH by DNA-cellulose from whole cell extracts (lane 3). Moreover, the presence of the nonhydrolyzable ATP analog AMP-PNP did not alter the amount of any precipitates (lane 6). Evans et al. (19) reported that ATP hydrolysis is necessary for the open complex formation immediately around the lesion. We therefore propose that the initial NER protein complex on DNA may be formed without reliance on ATP, but the initial opening reaction re-
requires ATP. TFIIH has been shown to have little affinity for DNA by itself (20, 35). Therefore, the observed precipitation of TFIIH by dsDNA may be due to specific interaction with other protein(s) bound to DNA or dependent upon conformational changes in DNA induced by DNA-binding proteins. To test the possibility that other NER proteins mediate the interaction between TFIIH and DNA, several precipitation experiments were performed using whole cell extracts derived from NER-deficient cell lines (Fig. 5). XPC, p62, and RPA32 were precipitated from XP-A and 293 whole cell extracts (lanes 3 and 11) or dsDNA-cellulose (lanes 4 and 12). In contrast, p62 was not recovered in the precipitates from XP-C whole cell extracts, whereas binding of XPA and RPA32 to both ssDNA- and dsDNA-cellulose were unaffected (compare lanes 3 and 4 with 7 and 8, respectively). The same results were obtained with extracts from XP-B (GM2252A), XP-D (XP6BESV), XP-G (XP3BRSV), CS-A (CS2OSSV), and CS-B (GM1629SV) cells, all of which express the XPC protein, as with 293 whole cell extracts (data not shown). Intriguingly, ssDNA was insufficient to associate with TFIIH even though XPC binds to ssDNA as well as to dsDNA (Fig. 5A, compare lanes 3, 4, 11, and 12). Furthermore, when the XP-C cell extracts were supplemented with purified XPC-HR23B complex, TFIIH was detected in the precipitate fraction of dsDNA-cellulose (Fig. 5B). Thus TFIIH stably associated with dsDNA, either directly or indirectly, only in the presence of XPC. It should be noted that extra signals were observed above the bands corresponding to XPA. These extra bands probably represent modified forms of the target proteins, because specific antibodies were used. We should also mention here that the amounts of precipitated proteins by ssDNA- or dsDNA-cellulose in each experiments could be altered when the different batch, but the same cell line, of cell extracts was employed (data not shown).

**TFIIH Was Precipitated Efficiently by Damaged DNA as Well as XPC**—To assess the relevance of the XPC-dependent association of TFIIH with DNA, similar precipitation experiments were performed with dsDNA fragments that had been treated with N-AAAFT and immobilized on Sepharose beads. As shown in Fig. 6A, the amounts of XPC and TFIIH detected in the precipitates from extracts of repair-proficient 293 cells increased with increasing time of N-AAAFT treatment of the DNA (lanes 3-6). On the other hand, a slight increase was observed in the amount of XPA in damaged DNA bound fractions (compare lane 3 with 4-6), and no significant increase was observed in the amounts of RPA in any precipitates (lanes 3-6). Intriguingly, under the same conditions, TFIIH was found to co-precipitate with N-AAAFT-treated DNA, even though TFIIH displays little affinity for dsDNA, UV-irradiated or untreated, by itself (20, 35). Again, we examined whether this binding of TFIIH to damaged DNA was dependent on the presence of ATP or XPC as well as previous experiments. Damaged DNA was incubated with 293 whole cell extracts in the absence or presence of ATP or AMP-PNP, and the precipitates were examined by immunoblotting. As shown in Fig. 6B, the examined proteins were equally immunodetected in the DNA binding fraction in the absence of ATP (lane 3) or in the presence of ATP (lane 5) or AMP-PNP (lane 6), suggesting that neither ATP nor ATP hydrolysis was required for early NER complex formation on damaged DNA. In the XP-C cell extracts, little p62 was found in the precipitate with undamaged DNA (Fig. 6C, lane 3), whereas addition of the XPC-HR23B complex resulted in a slight increase in the amount of bound p62 (Fig. 6C, lane 4). On the other hand, despite the absence of XPC, the amount of p62 in the precipitate was greater with the damaged DNA substrate (compare lane 3 with 5). A slight increase was also observed in the amounts of XPA, but no significant increase was observed in the amounts of RPA in the same precipitates (compare lane 3 with 5). One can presume that part of TFIIH will be recruited on damaged DNA with XPA. On the other hand, it has been reported that conformational changes in DNA can alter the cellular requirement for XPC in repair complex formation and initiation of NER (36). The minor increase in the amount of p62 precipitated by damaged DNA may have been due to modification of DNA structure by N-AAAFT treatment. Importantly, the XPC-dependent binding of TFIIH was much more pronounced with the N-AAAFT-treated DNA (compare

### Fig. 4. Precipitation of NER factors with DNA-cellulose from 293 cell extracts.**

Whole cell extracts from human 293 cells were incubated with cellulose (lanes 2 and 4) or dsDNA-cellulose (lanes 3, 5, and 6) beads in the absence of ATP (lanes 2 and 3) or in the presence of 2 mM ATP (lanes 4 and 5) or 2 mM AMP-PNP (lane 6). The presence of XPC, TFIIH (p62), XPA, and RPA (p32) in the precipitates was examined by immunoblotting with specific antibodies. Lane 1, 10% of input 293 cell extracts.

### Fig. 5. XPC-dependent precipitation of TFIIH with DNA-cellulose.**

A. Whole cell extracts from 293, XP4PASV (group C), or XP7CASV (group A) cells were incubated with cellulose (lanes 2, 6, and 10), ssDNA-cellulose (lanes 3, 7, and 11), or dsDNA-cellulose (lanes 4, 8, and 12) beads. The bound protein fractions were examined for the presence of XPC, TFIIH (p62), XPA, and RPA (p32) by immunoblotting with specific antibodies. Lanes 1, 5, and 9 contain 10% of the extracts included in the binding reactions. B. Similar binding experiments were carried out with the XP-C cell extracts supplemented with purified XPC-HR23B complex (lanes 7-9). Lanes 1, 4, and 7, 10% of the input extracts; lanes 2, 5, and 8, cellulose-bound fractions; lanes 3, 6, and 9, dsDNA-cellulose-bound fractions.
we cannot exclude the possibility that HR23B may somehow affect the interaction of XPC and TFIH, because both HR23B and HR23A (another human homolog of RAD23) have been shown to stimulate the repair activity of XPC in cell-free NER reactions (22, 26).

**XPC-HR23B Recruits TFIH to Damaged DNA**—We recently reported that the XPC-HR23B complex specifically binds various NER lesions and functions as a damage detector initiating the repair reaction in the global genome repair pathway (18). Based on these findings, it is conceivable that XPC-HR23B bound to the lesion could recruit other NER factors, possibly via local conformational changes in the DNA as well as protein-protein interactions. Importantly, TFIH is the only NER factor that has been shown to interact directly with XPC; neither purified XPA nor RPA was co-precipitated with the XPC-HR23B beads (data not shown). We show in this report that TFIH, like XPA, XPC, and RPA, can be precipitated from crude cell extracts with dsDNA immobilized on cellulose beads (Fig. 4), even though TFIH exhibits little affinity for DNA by itself (20, 35). Our observations indicate that the discrimination between damaged and undamaged DNA is modest among XPA, XPC, and RPA. We should point out, however, that the commercially available DNA-cellulose was used in this experiment, and so we cannot exclude the possibility that conformational changes in the DNA that were generated during the production of DNA-cellulose caused the binding of these NER proteins. It is also interesting to note that these NER proteins could be precipitated by dsDNA-cellulose or the damaged DNA substrate immobilized on Sepharose beads in an ATP-independent manner (Figs. 4 and 6B). It has been reported that ATP is necessary for formation of the stable excision nuclease-damaged DNA complex and for the dual incision reaction (19, 20, 34, 36). However, some reports suggest that ATP hydrolysis is not required for formation of the DNA-NER protein complex (35, 42). Our method may be sufficiently sensitive to detect relatively weak protein-protein and/or protein-DNA interactions formed in the absence of ATP. Importantly, the association of TFIH with DNA was markedly stimulated by the presence of XPC (Fig. 5) but not by XPA. Previous studies have reported that recombinant XPA may also be able to interact with purified TFIH and possibly recruit it to damaged DNA (35, 43). In contrast, our results strongly indicate that the protein-protein interaction between XPC and TFIH is required for recruitment of TFIH to damaged DNA in whole cell extracts. Furthermore, introduction of bulky adducts to dsDNA by N-AAA treatment resulted in marked enhancement of both XPC and TFIH binding to DNA (Fig. 6, A and C). Taken together, these observations indicate that TFIH is recruited to the NER lesion via interaction with prebound XPC. These results, however, do not exclude the possibility that XPA is required to initiate the NER reaction. Wakasugi and Sancar (44) have proposed a model for the assembly of human NER factors on damaged DNA. Based on measurements of binding affinity to DNA containing a single (6–4) photoproduct and a series of kinetic experiments using a reconstituted DNA repair system, they concluded that an RPA-XPA-DNA ternary complex forms first at the site of DNA lesions to initiate NER. In fact, the incision reaction has never been observed without XPA in the reconstituted excision reaction, probably because of the incomplete open complex formation (19, 34, 36). In contrast, our finding is consistent with the model proposed by Evans et al. (19), in which initial opening of dsDNA around the lesion may be conducted by XPC-HR23B and TFIH. Li et al. (45) also demonstrated that XPC is an indispensable component of the initial step of NER that stabilizes the interaction of TFIH with damaged DNA. Moreover, using gel mobility shift assays,

**Interaction between the XPC-HR23B Complex and TFIH**

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