The Xrcc2 and Rad51D/Rad51L3 proteins, which belong to the Rad51 paralogs, are required for homologous recombinational repair (HRR) in vertebrates. The Xrcc2 and Rad51D/Rad51L3 genes, whose products interact with each other, have essential roles in ensuring normal embryonic development. In the present study, we coexpressed the human Xrcc2 and Rad51D/Rad51L3 proteins (Xrcc2 and Rad51D, respectively) in *Escherichia coli*, and purified the Xrcc2-Rad51D complex to homogeneity. The Xrcc2-Rad51D complex catalyzed homologous pairing between single-stranded and double-stranded DNA, similar to the function of the Xrcc3-Rad51C complex, which is another complex of the Rad51 paralogs. An electron microscopic analysis showed that Xrcc2-Rad51D formed a multimeric ring structure in the absence of DNA. In the presence of ssDNA, Xrcc2-Rad51D formed a filamentous structure, which is commonly observed among the human homologous pairing proteins, Rad51, Rad52, and Xrcc3-Rad51C.

Chromosomal DNA is vulnerable to attacks from the environment and sustains multiple types of damage, including the double strand break (DSB), which is a lethal DNA lesion for cells if it is not repaired. Homologous recombinational repair (HRR) is one of the major pathways for the repair of DSBs. When cells are defective in HRR, unrepaired DSBs accumulate in chromosomes (1).

In *Escherichia coli*, the RecA protein catalyzes homologous pairing, which is a key step in HRR (2, 3). In homologous pairing, RecA binds the single-stranded DNA (ssDNA) produced at DSB sites and forms nucleoprotein filaments. Then, the nucleoprotein filaments bind double-stranded DNA (dsDNA) and form a three-component complex that includes ssDNA, dsDNA, and RecA. In the three-component complex, the homology between ssDNA and dsDNA is searched, and joint molecules, in which the ssDNA invades into the homologous region of the dsDNA, are formed as products of homologous pairing.

In eukaryotes, the Rad51 protein has been identified as a homologue of RecA (4). The Rad51 protein, which is conserved from yeast to human (5), forms a nucleoprotein filament that is strikingly similar to that formed by RecA (6), suggesting their functional similarity in HRR. Actually, the *Saccharomyces cerevisiae* and human Rad51 proteins (ScRad51 and HsRad51, respectively) catalyze homologous pairing (7–9). We have found that the human Xrcc3 and Rad51C/Rad51L2 proteins (Xrcc3 and Rad51C, respectively) form a complex and catalyze homologous pairing (10) in addition to HsRad51. Both Xrcc3 and Rad51C are members of the Rad51 paralogs (Xrc2 (11, 12), Xrcc3 (12, 13), Rad51B/hREC2/Rad51L1 (14–16), Rad51C/Rad51L2 (17), and Rad51D/Rad51L3 (16, 18, 19), etc.) and share 20–30% amino acid identity with HsRad51.

The Xrcc2 and Xrcc3 genes were first identified as human genes that complement the DNA damage-sensitive hamster cell lines, irs1 and irs1SF, respectively (13, 20–23), and both genes were confirmed to be involved in HRR in *vivo* (24, 25). Cells lacking Xrcc2 or Xrcc3 show extreme sensitivity to DNA cross-linking reagents like cisplatin and ionizing radiation and have significantly increased missegregation of chromosomes (12, 13, 26). Knock-out experiments in the chicken DT40 cell lines showed that the Rad51B/Rad51L1, Rad51C/Rad51L2, and Rad51D/Rad51L3 genes, as well as the Xrcc2 and Xrcc3 genes, are involved in the HRR pathway (27, 28). The Xrcc2 protein also has homology to the *S. cerevisiae* Rad55 protein (29), which interacts with the *S. cerevisiae* Rad57 protein (30–32). Interestingly, the Xrcc2 knock-out mice exhibited embryonic lethality associated especially with a high frequency of apoptotic death of neurons in the developing brain (33). Xrcc2 interacts with the human Rad51D/Rad51L3 protein (Rad51D), another Rad51 paralog (34, 35). The Rad51D knock-out mice also showed embryonic lethality (36), indicating that both Xrcc2 and Rad51D are essential for embryonic development.

In the present study, we purified the Xrcc2-Rad51D complex and characterized it biochemically. The purified Xrcc2-Rad51D complex catalyzed homologous pairing between ssDNA and dsDNA, similar to that of the Xrcc3-Rad51C complex. The Xrcc2-Rad51D complex formed a multimeric ring structure in
the absence of DNA and formed nucleoprotein filaments in the presence of ssDNA. These biochemical characteristics of Xrcc2-Rad51D are consistent with the idea that these proteins act in an early step of the HRR pathway.

**EXPERIMENTAL PROCEDURES**

**Proteins**—The Xrcc2 and Rad51D genes were cloned from human brain cDNA (purchased from CLONTECH) by PCR. The overexpression plasmid for Xrcc2 and Rad51D was introduced into E. coli JM109(DE3) cells with the plasmid containing the genes for E. coli tRNA Arg-3 and tRNA Arg-4, which recognize the CGG and AGA/AGG codons, respectively. The cells were grown in 10 liters of LB medium containing 100 µg/ml ampicillin and 34 µg/ml chloramphenicol at 30 °C for 10 h, and then 200 µM isopropyl β-thiogalactopyranoside was added to induce protein expression. Xrcc2 and Rad51D were produced for 12 h at 18 °C in the presence of isopropyl β-thiogalactopyranoside, and the cells were harvested. Both Xrcc2 and Rad51D contain a His tag at the N terminus. The cells producing Xrcc2 and Rad51D were resuspended in 20 ml of buffer containing 20 mM Tris-HCl (pH 8.5), 2 mM dithiothreitol, and 0.5 M NaCl. Subsequently, the resin was washed with 150 ml of buffer containing 20 mM Tris-HCl (pH 8.5) and 30% glycerol. Xrcc2 and Rad51D were eluted by a gradient of imidazole from 30 to 400 mM in 20 mM Tris-HCl (pH 8.1), 2 mM dithiothreitol, and 10% glycerol. Xrcc2 and Rad51D were further purified by gel filtration chromatography on Superdex 200HR resin (Amersham Biosciences). The purified proteins were stored in 20 mM Tris-HCl (pH 8.1), 5 mM dithiothreitol, and 10% glycerol.

**Preparation of Closed Circular Double-stranded DNA**—To avoid irreversible denaturation of the dsDNA, we prepared plasmid DNA without any treatment that would potentially cause denaturation, such as alkaline treatment. The plasmid DNA (pGsat4; 3216 base pairs) was excised from a clone of human α-satellite DNA. The 5’ ends of the oligonucleotides were labeled with 74 polyuridine kinase (New England Biolabs) in the presence of [γ-32P]ATP. DNA concentrations are expressed in moles of nucleotides.

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preincubated by ethanol. The pellet was dissolved in 1 ml of TE buffer (10 mM Tris-HCl (pH 8.0), 1 mM EDTA) and treated with 0.15 mg/ml RNase A at 37 °C for 30 min. The pGsat4 DNA was purified by a 5-20% sucrose gradient centrifugation at 24,000 rpm for 18 h. DNA concentrations are expressed in moles of nucleotides.

Preparation of Circular Single-stranded DNA—The E. coli cells (JM109) containing pGsat4 DNA were cultured in LB medium until an A$_{600}$ of 0.3 was achieved and were infected with the helper phage ρ408 at a multiplicity of infection of ~20. After infection, the cells were continuously cultured for 6 h with vigorous agitation. Then, the supernatant was harvested and treated with DNase I (10 unit/ml) and RNase A (10 μg/ml) for 15 min at 37 °C. The phage containing pGsat4 ssDNA was precipitated with 2 volumes of ethanol in the presence of 0.5% sodium dodecyl sulfate (SDS). After precipitation, the aqueous phase was extracted by chloroform, and the remaining DNA was extracted by chloroform:isoamyl alcohol (24:1) followed by phenol:chloroform saturated with TE buffer. After several phenol:chloroform extractions, the aqueous phase was extracted by chloroform, and the pGsat4 ssDNA was precipitated with 2 volumes of ethanol in the presence of 3.25 M ammonium acetate. The resulting pellet was resuspended with TE buffer, and the pGsat4 ssDNA was extracted by chloroform:isoamyl alcohol (24:1) followed by phenol:chloroform saturated with TE buffer. After several phenol:chlorof orm extractions, the aqueous phase was extracted by chloroform, and the pGsat4 ssDNA was precipitated with 2 volumes of ethanol in the presence of 3.25 M ammonium acetate. The resulting pellet was resuspended with 70% ethanol and dried under vacuum. The pGsat4 ssDNA was dissolved in H$_2$O; its concentration is expressed in moles of nucleotides.

Single-stranded DNA Binding Assay—Single-stranded pGsat4 DNA (30 μM) was mixed with Xrcc2-Rad51D in 10 μl of standard reaction buffer containing 20 mM Tris-HCl (pH 8.1), 2 mM ATP, 1 mM dithiothreitol, 100 μg/ml BSA, 2 mM MgCl$_2$, and 6% glycerol. The reaction mixtures were incubated at 37 °C for 10 min and then analyzed by 0.8% agarose gel electrophoresis in 0.5× TBE buffer. The bands were visualized by ethidium bromide staining.

Double-stranded DNA Binding Assay—Superhelical pGsat4 DNA (18 μM, 3216 base pairs) was mixed with Xrcc2-Rad51D in 10 μl of standard reaction buffer containing 20 mM Tris-HCl (pH 8.1), 2 mM ATP, 20 mM creatine phosphate, 1 mM dithiothreitol, 100 μg/ml BSA, 12 units/ml creatine phosphokinas e, 2 mM MgCl$_2$, and 6% glycerol. The reaction mixtures were incubated at 37 °C for 10 min, and then the samples were electrophoresed on a 0.8% agarose gel for 4 h at 3 V/cm in 0.5× TBE buffer. The bands were visualized by ethidium bromide staining.
The Xrcc2-Rad51D Complex Catalyzes D-loop Formation—We have previously reported that the Xrcc3-Rad51C complex catalyzes homologous pairing between a single-stranded oligonucleotide and a superhelical dsDNA by the D-loop formation assay, which is a standard assay for homologous pairing by RecA (10). As shown in Fig. 2, the Xrcc2-Rad51D complex also catalyzes homologous pairing. The superhelical dsDNA used in this assay was prepared by a method without alkali treatment to avoid irreversible denaturation of the double helix of the dsDNA (37). The homologous pairing by Xrcc2-Rad51D was an ATP-independent reaction (Fig. 2A, lane 7), like those catalyzed by Xrcc3-Rad51C (10), the human Rad52 protein (38, 39), and the E. coli RecT protein (40), and was detected only with a homologous combination of ssDNA and dsDNA but not with a heterologous combination or with BSA instead of Xrcc2 in the absence of DNA. All observed with RecA at its optimum MgCl₂ condition (10 mM) was preferred in the homologous pairing process under the same conditions (Fig. 2B, lanes 3 and 4). These data clearly show that the products formed by Xrcc2-Rad51D were authentic D-loops. A low MgCl₂ condition (2 mM) was preferred in the homologous pairing promoted by Xrcc2-Rad51D as compared with a high MgCl₂ condition (10 mM) (Fig. 2B, lanes 4 and 5), and the activity observed with the low MgCl₂ condition was about 20% of that observed with RecA at its optimum MgCl₂ condition (10 mM) (Fig. 2C).

Xrcc2-Rad51D Forms a Multimeric Ring Structure—In the Superdex 200 HR gel filtration chromatography, Xrcc2-Rad51D was eluted near the void volume, and no proteins were detected in the fractions corresponding to the molecular size (71 kDa) of the Xrcc2-Rad51D heterodimers (Fig. 3). This result indicates that Xrcc2-Rad51D exists as a multimer rather than a heterodimer. Therefore, we analyzed the structure of the Xrcc2-Rad51D multimer by electron microscopy. As shown in Fig. 4, Xrcc2-Rad51D formed multimeric ring structures (Fig. 4, A–E). The ring structure formed by Xrcc2-Rad51D is similar to that of HsRad52 in its size and shape (39, 41).

Xrcc2-Rad51D Binds to DNA—we tested the DNA binding activity of Xrcc2-Rad51D. Xrcc2-Rad51D bound both ssDNA and dsDNA (Fig. 5, A and B). Although significant amounts of ssDNA and dsDNA were bound to Xrcc2-Rad51D in the absence of ATP, agarose gel electrophoresis revealed that the migration distances of the complexes between Xrcc2-Rad51D and DNA were drastically changed by the addition of ATP (Fig. 5, A, lane 5, and B, lane 5). These results suggest that less Xrcc2-Rad51D bound to ssDNA in the absence of ATP. Both Xrcc2 and Rad51D have an ATP-binding motif. It has been reported that disruptions of the ATP-binding motif of Xrcc2 did not affect the complementation of the phenotypes of the Xrcc2-deficient cell line, irs1 (42). The ATP may be important in regulating the DNA binding of Rad51D or may bind different sites from the ATP-binding motifs of Xrcc2 and Rad51D. Further studies are required to elucidate the roles of ATP binding by Xrcc2-Rad51D.

Xrcc2-Rad51D Forms a Filamentous Complex with ssDNA—In the absence of DNA, Xrcc2-Rad51D formed a multimeric ring structure (Fig. 4), and no filamentous structure was observed. However, an electron microscopic analysis showed that Xrcc2-Rad51D formed filamentous structures in the presence of ssDNA (Fig. 6A). To compare the Xrcc2-Rad51D filament with the HsRad51 filament, we visualized the HsRad51-ssDNA complex under the same conditions (Fig. 6B) used for the Xrcc2-Rad51D filament observation. As shown in Fig. 6B, HsRad51 formed short nucleoprotein filaments with ssDNA, but the length of the filaments were shorter than that of the Xrcc2-Rad51D filament, which fully covered circular ssDNA (Fig. 6A). The ssDNA circle (5386 bases) covered with Xrcc2-Rad51D was significantly smaller than the same length of the dsDNA circle (5386 base pairs) covered by HsRad51 (Fig. 6, A and C). Thus, the DNA bound to Xrcc2-Rad51D may be packaged differently from that bound to HsRad51, possibly wrapping around the Xrcc2-Rad51D ring as in the case of HsRad52 (43).

Although both HsRad51 and Xrcc2-Rad51D formed filamentous complexes in the presence of ssDNA, the appearance of those filaments was different. HsRad51 formed a nucleoprotein filament, which was clearly helical in appearance (Fig. 6, B and C). This helical filament is strikingly similar to that of RecA (44). In contrast, the filaments formed by Xrcc2-Rad51D did not have a RecA-like helical appearance (Fig. 6A) and interestingly, did have significant similarities with those formed by Xrcc3-Rad51C (10) or HsRad52 (39).

DISCUSSION

It has been reported that Xrcc2 is required for embryonic neurogenesis (33). Both Xrcc2 and Xrcc3 are expressed in the brain (12), where the expression of Rad51 is very low (5). Our present and previous two-hybrid analyses have shown that Xrcc2 and Xrcc3 interact with Rad51D and Rad51C, respectively, in the human brain cDNA library (10). These facts suggest that Xrcc2-Rad51D and Xrcc3-Rad51C may have a specific function in the brain. In nondividing neuronal cells, the homologous chromosomes, not the sister chromatids, must be the templates for homologous pairing, because the cells are arrested before DNA synthesis (the G₀ phase). Xrcc2-Rad51D and Xrcc3-Rad51C may have a preference for homologous pairing between homologous chromosomes rather than sister chromatids.

Both Xrcc2 and Xrcc3 are required independently for HRR in mammalian cells, because no other proteins can complement their mutant phenotypes. These facts suggest that Xrcc2-Rad51D and Xrcc3-Rad51C are functionally similar but have different roles in the HRR pathway. Schild et al. (35) detected an indirect interaction between Xrcc2 and Rad51C by the three-hybrid system in the presence of Rad51D, suggesting a
weak interaction between Rad51C and Xrcc2-Rad51D. This interaction may function to form a multiprotein complex containing all four proteins onto the DSB site. Recently, it was reported that Xrcc2/Rad51D interacts with Rad51B and Rad51C, forming a complex distinct from that of Xrcc3/Rad51C (45, 46). Xrcc2-Rad51D may be a part of the complex containing Xrc2, Rad51B, Rad51C, and Rad51D. Further analyses are required to elucidate the biological significance of these homologous pairing activities in the human system.

The Xrcc2, Xrcc3, Rad51C, and Rad51D genes are also expressed in dividing cells, where the Rad51 expression is observed (12, 17, 19). Interestingly, the mutant cell lines lacking one of the Xrcc2, Xrcc3, Rad51C, and Rad51D genes significantly attenuated the Rad51 assembly on chromosomes after exposure to γ-rays (28, 47). These results suggest that Xrcc2-Rad51D and Xrcc3-Rad51C are assembled on the damaged sites of chromosomes before Rad51 and that they initiate homologous pairing in cooperation with Rad51 in dividing cells. In addition to the similarity between Xrcc2-Rad51D and Xrcc3-Rad51C, we also found significant similarity between Xrcc2-Rad51D and HsRad52, an important protein in the HRR pathway. HsRad52 is known to form a multimeric ring struc-
ture similar to that of Xrcc2-Rad51D (41). Furthermore, we found that HsRad52 also formed nucleoprotein filaments with ssDNA and catalyzed homologous pairing as did Xrcc2-Rad51D and Xrcc3-Rad51C (10, 39). These results indicate that the characteristics of HsRad52 are similar to those of the Rad51 paralogs, although HsRad52 lacks obvious sequence homology with any of the Rad51 paralogs. The multiple homologous pairing activities may be necessary to support the HRR pathway in human chromosomes, which are more complex than those of bacteria and unicellular organisms such as yeast.

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