A Novel Human Rad54 Homologue, Rad54B, Associates with Rad51*

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Members of the SNF2/SWI2 family, characterized with sequence motifs similar to those found in DNA and RNA helicases, play roles in various aspects of cellular fundamental processes such as transcriptional regulation, chromosome stability, nucleotide excision repair, and recombination. We have isolated a novel member of the human SNF2/SWI2 family, RAD54B, which is highly homologous to mammalian RAD54. The RAD54 gene is a member of the RAD52 epistasis group which is involved in the recombinational repair of DNA damage. Here we demonstrate that human Rad54B (hRad54B), like human Rad54 (hRad54), associates with human Rad51 (hRad51). Both hRad54B and hRad54 associate with hRad51 through their NH2-terminal domains, but there are differences in their ways of association with hRad51. In contrast to Rad54, whose association with Rad51 is induced by ionizing radiation, Rad54B associates with Rad51 constitutively in immunoprecipitation experiments. Also, the failure to detect the interaction between hRad54B and hRad51 in the yeast two-hybrid assay suggests that their interaction, unlike that between hRad54 and hRad51, may be indirect. Immunofluorescence microscopy revealed that hRad54B formed nuclear foci that colocalized with hRad51, hRad54, and hRad51. Our data suggest that Rad54B constitutes a part of the RAD52 epistasis group.

The SNF2/SWI2 family is a still expanding superfamly of DNA-dependent ATPases. Members of this family are involved in various functions, such as transcriptional regulation (SNF2, MOT1, and BRM), chromosome stability (lodestar), nucleotide excision repair (ERCC6 and Rad16), and recombination (Rad54) (1). Recently, we cloned a novel gene of the SNF2/SWI2 family, RAD54B, which shares homology with mammalian and yeast RAD54 (2).

In Saccharomyces cerevisiae, the RAD52 epistasis group genes are involved in the recombinational repair of DNA damage, including DNA double-strand breaks as well as playing a role in meiotic recombination (3), and it has been demonstrated that the RAD52 epistasis group genes are well conserved structurally and functionally throughout evolution (4). RAD54 is a member of the RAD52 epistasis group. Yeast Rad54 stimulates homologous DNA pairing by Rad51 (5), and it was shown that homozygous RAD54 mutants in mouse and chicken are highly radiation- and methyl methanesulfonate-sensitive and have reduced levels of homologous recombination (6, 7).

Human RAD54B encodes a protein of 910 amino acids. The central part, which contains the seven helicase motifs found in members of the SNF2/SWI2 family, is well conserved between hRad54 and hRad54B, whereas the NH2-terminal region is less conserved except the first 10 amino acids (2). The expression pattern of RAD54B coincides with those of members of the RAD52 epistasis group, although the functions of Rad54B are unknown. It has been shown that in mammals, as well as in yeast, the proteins of the RAD52 epistasis group interact with each other and form a complex to effect the recombinational repair of double-strand breaks (8–11). Both yeast and human Rad54 (hRad54) are known to interact with Rad51 (12–14). To investigate whether hRad54B is involved in homology-directed DNA repair, we examined the association of hRad54B and hRad51. We demonstrate that hRad54B, like hRad54, associates with hRad51 although it associates in a manner distinct from hRad54. Our data suggest that Rad54B constitutes a part of a complex of recombinational repair of double-strand breaks.

EXPERIMENTAL PROCEDURES

Cell Culture and DNA Transfection—COS-7 cells, mouse embryonic stem (ES) cells, DU145 cells (human prostate cancer), and three human breast cancer cell lines, MCF-7, MDA-MB-231, and SkBr3, were grown in Dulbecco’s modified Eagle’s medium. HCT116 (human colon carcinoma) cells were grown in McCoy’s 5A medium. BALL-1 (human B cell leukemia) and CCRF-HSB-2 (human T cell leukemia) cells were provided by the Riken Gene Bank (Tsukuba, Japan) and grown in RPMI 1640 medium. All cells were maintained at 37 °C in a 5% CO2 environment, and all culture media were supplemented with penicillin/streptomycin and 10% fetal calf serum. Plasmids were transfected using Superfect Transfection Reagent (Qiagen) according to the manufacturer’s instructions.

Plasmid Constructions—Human RAD51, RAD54, and RAD54B cDNA were cloned from a human testis cDNA library (CLONTECH Laboratories). The part of human RAD54 encoding the NH2-terminal 751 amino acids was placed under the 5S promoter in the vector pME18S to yield plasmid pME-Rad54B(1–751). For tagging hRad54B fragments at the NH2 termini, the FLAG epitope (DYKDDDDK) was inserted after the first methionine codon by polymerase chain reaction. The fragments of the FLAG-tagged NH2-terminal 251 amino acids and the COOH-terminal 659 amino acids of hRad54B were cloned into pME18S to yield plasmids pME-FLAG-Rad54B(1–251) and pME-FLAG-Rad54B(252–910), respectively. Human RAD51 cDNA was cloned into pcDNA3.1 (Invitrogen) to yield plasmid pcDNA-Rad51. A glutathione S-transferase (GST) fusion of the NH2-terminal 200 amino acids of hRad54B (GST-Rad54B(1–200)) was constructed by ligating the fragment of the NH2-terminal 200 amino acids of hRad54B into the pGEX-3X vector (Amersham Pharmacia Biotech). GST-Rad54B(1–252) was constructed by ligating the fragment of the NH2-terminal 242 amino acids of hRad54B into the pGEX-1 vector (Amersham Pharmacia Biotech). For the yeast two-hybrid assay, human RAD51, RAD54, and RAD54B cDNA were cloned into two-hybrid vectors pGBT9 and pGBT9.

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1 The abbreviations used are: hRad54, human Rad54; hRad54B, human Rad54B; hRad51, human Rad51; ES, embryonic stem; GST, glutathione S-transferase; PAGE, polyacrylamide gel electrophoresis; Gy, grays; IR, ionizing radiation.
pGAD424 (CLONTECH Laboratories) as fusion with the Gal4 DNA binding domains and activation domains, respectively. The nucleotide sequences corresponding to the NH2-terminal 142 amino acids of hRad54 and the NH2-terminal 200 amino acids of hRad54B were amplified by polymerase chain reaction and also cloned into pGBT9 and pGAD424.

Antibodies—A rabbit polyclonal antiserum against hRad51 was kindly provided by Dr. T. Ogawa (National Institute for Genetics, Shizuoka, Japan) (15). A goat polyclonal antiserum against hRad51 (Rad51(I-20)) was purchased from Santa Cruz Biotechnology. A rabbit polyclonal antiserum against chicken Rad54 was a gift from Dr. A. Shinohara (University of Chicago). To obtain polyclonal antibodies against Rad54B, GST-Rad54B(1–200) was expressed in Escherichia coli JM105 cells, purified, and used to immunize a rabbit and hamsters as described previously (16). A mouse monoclonal antibody to human BRCA1 (Ab-1) was purchased from Oncogene Research Products. Anti-FLAG M2 monoclonal antibody was purchased from Sigma.

Western Blotting—Cell extracts were prepared in radioimmune precipitation buffer (50 mM Tris-HCl (pH 7.5), 150 mM NaCl, 0.05% SDS, 1% sodium deoxycholate, 1% Triton X-100, and 1 mM phenylmethylsulfonyl fluoride). Equal amounts were subjected to SDS-polyacrylamide gel electrophoresis (SDS-PAGE) and electrotransferred onto polyvinylidene difluoride filters (Millipore), then reacted with rabbit Rad54B antiserum (1:200 in dilution), rabbit Rad51 antiserum (1:200 in dilution), or anti-FLAG M2 antibody (1:400 in dilution). The blots were visualized by ECL blotting system (Amersham Pharmacia Biotech).

Immunoprecipitation of Lysates—COS-7 cells cultured for 40 h after transfection were harvested in 250 μl of radioimmune precipitation buffer/100-mm-diameter culture dish. 1 mg of cell lysates/lane was mixed with 5 μl of rabbit or goat Rad51 antiserum, rabbit Rad54B antiserum, or anti-FLAG M2 antibody and rotated for 1 h at 4 °C. Then samples were incubated with protein A-Sepharose (Sigma; for rabbit antibodies) or protein G PLUS-agarose (Santa Cruz Biotechnology; for goat and mouse antibodies) for 1 h at 4 °C, followed by washing three times with radioimmune precipitation buffer. Immunoprecipitates were subjected to SDS-PAGE and Western blotting.

In Vitro Binding Assays—The GST, GST-Rad54(1–242), or GST-Rad54B(1–200) protein was expressed in E. coli JM105 cells and purified as described previously (16). Approximately 5 μg of each protein immobilized on glutathione-Sepharose 4B (Amersham Pharmacia Biotech) was incubated with the lysates from COS-7 cells overexpressing hRad51 in radioimmune precipitation buffer for 1 h at 4 °C with gentle rotation. The protein-GST beads were washed five times with radioimmune precipitation buffer and subjected to SDS-PAGE and Western blotting.

Yeast Two-hybrid Experiments—Interactions were analyzed in yeast reporter strain SPY926 by measuring the levels of β-galactosidase.
produced in a liquid assay, using O-nitrophenyl β-D-galactopyranoside as substrate (17). Yeast transformants were selected at 30 °C on plates with synthetic complete medium lacking leucine and tryptophan. For the β-galactosidase assay, cells from individual colonies were grown to saturation in the same medium. Three colonies were assayed in each case.

**Immunofluorescent Cell Staining—**DU145, MCF-7, MDA-MB-231, and SKBr3 cells were grown on coverslips. BALL-1 and CCRF-HSB-2 cells were centrifuged onto glass slides at 500 rpm for 5 min. The cells were fixed in –20 °C methanol for 30 min and then immersed in ice-cold acetone for a few seconds to permeabilize cells for antibody staining. After three washes with phosphate-buffered saline, the preparations were incubated at room temperature with a 1:200 dilution of rabbit antiserum against Rad51, Rad54, Rad54B, mouse BRCA1 monoclonal antibody (Ab-1), or hamster Rad54B antiserum for 1 h, followed by incubation with 1:100 dilution of fluorescein isothiocyanate-conjugated donkey anti-rabbit IgG (Chemicon International Inc.), rhodamine-conjugated donkey anti-mouse IgG (Chemicon International Inc.), or Texas Red-conjugated goat anti-hamster IgG (EY Laboratories Inc.) for 1 h. For double labeling, the mixture of rabbit Rad54B antiserum and Ab-1, Rad51 antiserum and hamster Rad54B antiserum, or Rad54 antiserum and hamster Rad54B antiserum was used as the primary antiserum, and the appropriate combination of labeled antibodies was used as the secondary antiserum. The cells were visualized under TCS NT (Leica) and LSM510 (Carl Zeiss) confocal laser scanning microscopy.

**RESULTS**

**Interaction of Overexpressed hRad54B and hRad51—**First, we investigated the association of hRad54B with hRad51 in COS-7 cells expressing the first 751 amino acids of hRad54B (Rad54B(1–751)) together with hRad51. For this purpose, we raised a rabbit antiserum against the GST fusion of the first 200 amino acids of hRad54B (GST-Rad54B(1–200)). A band of the expected molecular mass was only detected in the COS-7 cells transfected with the Rad54B(1–751) expression vector (Fig. 1A). After immunoprecipitation with Rad54B antiserum or rabbit Rad51 antiserum, these immunoprecipitates were analyzed by immunoblotting using Rad54B antiserum and Rad51 antiserum. hRad51 was coimmunoprecipitated with Rad54B(1–751), showing the association between hRad54B and hRad51 (Fig. 1A). Rad54B(1–751) was coimmunoprecipitated with hRad51 when hRad51 was overexpressed and even when not overexpressed in a lower amount, probably because of the high expression level of endogenous Rad51.

**Interaction of Endogenous Rad54B and Rad51—**Next we examined the association of endogenous Rad54B and Rad51 in various cell lines. To investigate the role for Rad54B in the DNA damage response, we treated the cells with IR or left them untreated. It was reported that genotoxic stress induced the interaction between Rad51 and Rad54 without affecting the expression levels of Rad54 (18). The Rad54B antiserum, but not preimmune serum, recognized a band of about 100 kDa corresponding to endogenous Rad54B in the extracts from ES cells, BALL-1, CCRF-HSB-2, HCT116, and DU145 (Fig. 1B and data not shown). This antiserum did not cross-react with Rad54, a protein with a molecular mass of about 85 kDa (data not shown). The expression levels of Rad54B did not change after treatment of the cells with IR in all the cell lines examined.
Rad54B Associates with Rad51

Interactions between hRad51, hRad54, and hRad54B proteins in the yeast two-hybrid system

β-Galactosidase activities (in Miller units) were measured in liquid assay using O-nitrophenyl β-D-galactopyranoside as a substrate. The activities were based on the average values of three colonies. Rad54B(1–242) and Rad54B(1–200) contain the first 142 amino acids of hRad54 and the first 200 amino acids of hRad54B, respectively.

| DNA binding domain fusion | Rad51 | Rad54 | Rad54B | Rad54B(1–142) | Rad54B(1–200) |
|--------------------------|------|------|-------|-------------|-------------|
| None                     | <0.1 | <0.1 | <0.1  | <0.1        | <0.1        |
| Rad51                    | 0.11 ± 0.00 | 0.12 ± 0.01 | 0.14 ± 0.03 | 0.15 ± 0.02 | 0.11 ± 0.00 |
| Rad54                    | <0.1 | 0.17 ± 1.0 | <0.1  | <0.1        | <0.1        |
| Rad54B                   | <0.1 | 0.12 ± 0.02 | <0.1  | <0.1        | <0.1        |
| Rad54B(1–142)            | <0.1 | 0.14 ± 1.4 | <0.1  | <0.1        | <0.1        |
| Rad54B(1–200)            | <0.1 | 0.12 ± 0.03 | <0.1  | <0.1        | <0.1        |



Rad54B Associates with Rad51

Figure 1B and data not shown). In contrast to Rad54, communoprecipitated Rad54B with Rad51 was detected in both irradiated and nonirradiated cells in all of the cell lines examined, suggesting that the association is constitutive (Fig. 1B and data not shown). We detected Rad54B communoprecipitated with Rad51 after long exposure of chemiluminescence, probably because of the low expression level of endogenous Rad54B and the weak association of Rad54B with Rad51. We could hardly find Rad51 coimmunoprecipitated with Rad54B (data not shown).

Association with hRad51 Is Mediated by the NH2-terminal Region of hRad54B—Both the yeast and human Rad54 proteins are known to interact with Rad51 through the NH2-terminal domains (12–14). We therefore performed an in vitro binding assay to examine the interaction of the NH2-terminal domain of hRad54B and hRad51. GST-Rad54B(1–200) immobilized on glutathione-Sepharose beads was incubated with the lysates from COS-7 cells overexpressing hRad51. GST fusion with the NH2-terminal 242 amino acids of hRad54 (GST-Rad54(1–242)) was used as a positive control. As shown in Fig. 2, the hRad51 protein bound to GST-Rad54B(1–200) as well as GST-Rad54(1–242), indicating that hRad54B interacts with hRad51 through the NH2 terminus of hRad54B. The amount of GST-Rad54(1–242) used was smaller than that of GST-Rad54B(1–200), but the amount of hRad51 bound to GST-Rad54(1–242) was comparable to that of hRad51 bound to GST-Rad54B(1–200), suggesting that the association between hRad54B and hRad51 may be weaker than the association between hRad54 and hRad51.

In an effort to confirm the existence of a physical interaction between the NH2-terminal region of hRad54B and hRad51 in vivo, we asked whether hRad51 would form a complex with epitope-tagged hRad54B in COS-7 cells expressing the FLAG-tagged NH2-terminal 251 amino acids (FLAG-Rad54B(1–251)) or COOH-terminal 659 amino acids (FLAG-Rad54B(252–910)) of hRad54B together with hRad51. Fig. 3 shows that hRad51 was detected in an anti-FLAG communoprecipitate only when expressed with FLAG-Rad54B(1–251), but not with FLAG-Rad54B(252–910). In a reciprocal experiment, goat Rad51 antisemurn immunoprecipitation of a FLAG-Rad54B(1–251)-transfected COS-7 lysate communoprecipitated FLAG-Rad54B(1–251) (Fig. 3). In contrast, FLAG-Rad54B(252–910) was not communoprecipitated with hRad51. Thus, further evidence of the specific complex formation between hRad54B and hRad51 through the NH2-terminal region of hRad54B was obtained.

Analysis of Interactions of hRad54B in the Yeast Two-Hybrid System—To investigate the direct association of hRad54B with hRad51, we performed the yeast two-hybrid assay. The Gal4 DNA binding domain and the activation domain were fused to hRad51, hRad54, and hRad54B. These constructs were transformed into yeast reporter strain SPY526, and the β-galactosidase activities were measured. As shown in Table I, we confirmed the previous finding that hRad51 associates with itself and the NH2-terminal 142 residues of hRad54B (Rad54(1–142)) (14). The interaction of hRad54B with hRad51 was detected only when hRad51 was fused to the activation domain of Gal4, which was consistent with the reported pattern. In contrast, we could not find convincing evidence that hRad54B interacts with hRad51. In addition, we could detect neither the interaction of hRad54B with hRad54 nor itself. These findings suggest that hRad54B interacts with hRad51 indirectly and that there may be other proteins mediating the complex formation. Alternatively, the fusion of the Gal4 DNA binding domain or activation domain to the NH2 terminus of hRad54B protein may interfere with the binding of hRad54B to other proteins, or the interaction between hRad54B and hRad51 is below the level of detection.

hRad54B Colocalizes with hRad51, hRad54, and BRCA1 in the Nucleus—hRad51 is known to form nuclear foci that increase by irradiation (19). Both BRCA1 and Rad54 colocalize with Rad51 (18, 20). We investigated the colocalization of hRad54B with these proteins. The rabbit Rad54B antiserumn was used to visualize the distribution of hRad54B in various cell lines. We found that hRad54B is concentrated focally in small and discrete sites throughout the nucleoplasm in all of the cell lines examined (BALL-1, CCRF-HSB-2, DU145, MCF-7, MDA-MB-231, and SkBr3 cells (data not shown)). Use of preimmune serum, as well as omission of either the primary or secondary antibody, resulted in the absence of focally concentrated nuclear immunofluorescence (data not shown). The cells with hRad54B foci increased after IR treatment in these cell lines (Table II and data not shown). Two-color confocal immunostaining with rabbit Rad54B antiserum and mouse BRCA1 monoclonal antibody revealed significant, albeit not complete, colocalization of the hRad54B and the BRCA1 nuclear dot patterns in DU145 cells (Fig. 4, top panels). Similar staining patterns were observed in other cell lines (data not shown). To demonstrate the colocalization of hRad54B and hRad51, we raised a hamster antiserum against GST-Rad54B(1–200) and performed two-color immunostaining with rabbit Rad51 antiserum and hamster Rad54B antiserum. hRad51 foci detected by rabbit Rad51 antiserum showed sig-

Table II

| Focus formation of hRad51, hRad54, hRad54B, and BRCA1 after γ-irradiation in DU145 cells |
|-----------------------------------------------|
| Foci containing cells | Rad51 | Rad54 | Rad54B | BRCA1 |
|-----------------------|------|------|-------|-------|
| 0 Gy                  | 38 ± 3 | 36 ± 4 | 71 ± 4 | 32 ± 1 |
| 20 Gy                 | 98 ± 2 | 87 ± 1 | 89 ± 4 | 46 ± 6 |
significant colocalization with hRad54B foci detected by hamster Rad54B antiserum (Fig. 4, middle panels). We found the colocalization of hRad54B and hRad51 even in nonirradiated cells as well as in irradiated cells (data not shown). We also examined the colocalization of hRad54B and hRad54 using a rabbit antiserum against chicken Rad54. This antiserum was raised against the central, well conserved region of chicken Rad54 and cross-reacted with hRad54 but not with hRad54B in a Western blotting (data not shown). The immunostaining with rabbit Rad54 antiserum and hamster Rad54B antiserum showed the substantial colocalization of hRad54 and hRad54B (Fig. 4, bottom panels). Thus, hRad54B formed nuclear foci that colocalized with BRCA1, hRad51, and hRad54. hRad51, hRad54, and BRCA1 nuclear foci were dramatically induced by IR, consistent with previous findings (Table II). In contrast, we observed a slight increase in hRad54B foci formation after IR.

**DISCUSSION**

**Rad54B Associates with Rad51 in a Manner Distinct from Rad54**—Although both Rad54 and Rad54B interact with Rad51, there are differences between them in their ways of interacting with Rad51. First, the interaction between Rad54 and Rad51 is induced by IR, whereas that between Rad54B and Rad51 seems to be constitutive. Second, the results of the yeast two-hybrid assay suggest that the association of hRad54B with hRad51 may be indirect. These differences, probably originating from the diversity in NH2-terminal regions, imply that Rad54B has functions distinct from those of Rad54. Biochemically, Rad54 is considered to be a protein that promotes Rad51-mediated homologous DNA pairing through its double-stranded DNA-dependent ATPase activity (5, 22, 23). It is probable that Rad54B also has a double-stranded DNA-dependent ATPase activity because both Rad54 and Rad54B have Walker A and B motifs involved in ATP hydrolysis. Further study is needed to elucidate the functional relationship between Rad54B and Rad51.

**Alterations in Recombination Genes and Oncogenic Transformation**—Several lines of evidence support the idea that defects in homologous recombination are responsible for tumor formation. (i) Breast tumor susceptibility gene products BRCA1 and BRCA2 associate with Rad51 (20, 24). (ii) Nbs1, which is associated with the Mre11-Rad50 complex, is mutated in the...
Nijmegen breakage syndrome characterized by increased cancer incidence and IR sensitivity (25–27). (iii) RAD51B-HMG1C fusions are generated in (12;14) uterine leiomyomas (28). (iv) BRCA1-deficient ES cells have impaired repair of DNA double-strand breaks by homologous recombination (29). We have reported mutations in RAD54 and RAD54B in primary tumors, suggesting that some cancers arise through the alterations of members of RAD52 epistasis group (2, 30). Our present finding that Rad54B forms a complex with Rad51 strengthens the idea of a role for recombination proteins in tumor formation.

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