JOURNAL OF BACTERIOLOGY, Nov. 2000, p. 6287–6291 Vol. 182, No. 22
0021-9193/00/$04.00+0

radC102 of *Escherichia coli* Is an Allele of *recG*

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Received 22 May 2000/Accepted 21 August 2000

The *radC102* mutation causes mild UV and X-ray sensitivity and was mapped previously to near *pyrE* and *recG* at 82 min on the *Escherichia coli* chromosome (I. Felzenszwalb, N. J. Sargentini, and K. C. Smith, Radiat. Res. 97:615–625, 1984). We report that *radC102* has two striking phenotypes characteristic of *recG* mutations. First, it causes dramatically increased RecA-dependent mutation in a stationary-phase mutation assay. Second, it causes extreme UV sensitivity in combination with *ruv* mutations affecting the RuvABC Holliday junction resolution system. DNA sequencing of the *radC* and *recG* genes in *radC102* strains revealed that the *radC102* mutation creates a stop codon in *recG* that is predicted to truncate the RecG protein at 410 of 603 amino acids. A low-copy-number plasmid carrying the *radC*$^+$ gene did not affect the UV sensitivity of a wild-type strain, a *radC102* strain, or a *recG258*::Tn*10*mini-*kan* strain. We conclude that *radC102* is an allele of *recG* and that the function of the RadC protein remains to be determined.

The *radC102* mutation causes a mild UV and X-ray sensitivity and was mapped by transduction previously to the *pyrE recG* region at 82 min on the *Escherichia coli* chromosome (11). Further work identified a novel open reading frame (ORF), designated *radC*, as the site of the *radC102* mutation (9, 10). We present here several lines of evidence that *radC102* is an allele of *recG*. The *recG* gene was identified originally by mutations that cause mild UV sensitivity and slight defects in transductional and conjugational recombination (20, 22, 35). *recG* encodes a helicase capable of binding and unwinding strand exchange recombination intermediates (such as Holliday junctions) in vitro (39, 40) and probably carries out branch migration of recombination substrates in vivo (reviewed in reference 21). The importance of the RecG protein in recombination became clear with the discovery that cells lacking both RecG and RuvA, -B, or -C are extremely UV sensitive and recombination defective (18). Because the absence of either RecG or RuvABC has only slight effects on transductional and conjugational recombination (in the RecBCD pathway of recombination [21]), they were thought to play functionally redundant roles in recombination of linear substrates. However, they do not play identical roles, because their substrate specificities and directions of branch migration differ in vitro (1, 38, 39) and their effects on stationary-phase mutation (13, 17) and on recombination in some assays (19, 20, 25; M. Motamedi and S. M. Rosenberg, unpublished results) differ in vivo.

We began this work to ask whether *radC102* might affect *lac* frameshift mutation in stationary-phase *E. coli* cells, a mutational process dependent upon the recombination proteins RecA, RecBC, and RuvABC (13, 16, 17). In this stationary-phase (or adaptive) Lac$^+$ mutation process, recombination intermediates are proposed to promote DNA replication and mutation (16, 23, 30). In the course of these experiments we found that the phenotypes of *radC102* strains mimicked those of *recG* mutations in two assays, stationary-phase mutation and UV sensitivity. Subsequently, sequencing revealed that *radC102* strains carry a mutation in the *recG* gene and that the *radC* gene is wild type in *radC102* strains. We conclude that *radC102* is an allele of *recG*. The function of the *E. coli radC* gene (and its many bacterial homologs) remains to be determined.

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MATERIALS AND METHODS

Bacterial strains. Strains used in this work are shown in Table 1. All strains were constructed by standard transformation or P1 transduction techniques (28). Antibiotics were used as necessary at the following concentrations: tetracycline, 15 μg/ml; ampicillin, 100 μg/ml. The *radC102* allele was transduced from SR1187 (11) with selection for nearby *pyrE*$^+$ into SMR5426. Eleven of 12 Pyr$^+$ transductants tested were mildly UV sensitive on YENB medium (11), consistent with the linkage and UV sensitivity described previously (11). One of these transductants (SMR5441) was used for strain constructions and experiments. The *radC102 ruvC53* strain was constructed and grown at 30°C. Initial constructions at 37°C gave widely varying colony sizes, UV sensitivities, and stationary-phase mutation phenotypes. Instability of such phenotypes in *ruv recG* strains was observed previously (13, 17, 24).

Mutation and UV sensitivity assays. Stationary-phase mutation assays were as described previously (17). Briefly, multiple independent cultures of each strain were grown to saturation in minimal glycerol medium, washed twice in minimal medium with no carbon source, and plated on minimal lactose medium. Lac$^-$ scavenger cells, incapable of reverting to Lac$^+$ (FC29 [4]), were plated along with each strain at approximately a 20-fold excess cell number to prevent growth on any contaminating nonlactose carbon sources (4). Plates were incubated at 37°C, and Lac$^+$ colonies were counted each day for 5 days. *recA* strains were concentrated 10-fold prior to plating to obtain enough Lac$^+$ colonies. Viability of the Lac$^-$ frameshift-bearing cells on the selection plates was monitored each day as described previously (16, 17). There was no net change in the total number of Lac$^-$ frameshift-bearing cells on the plates during the course of these experiments (data not shown).

UV sensitivity was determined using saturated LBH (36) cultures. When plasmid-bearing strains were tested, ampicillin was included in the broth and in plates. Cultures were diluted and plated on LBH or LBH-ampicillin, irradiated or not, incubated at 37°C for approximately 24 h, and then counted. The fraction surviving was calculated as cells surviving/cells plated. All cultures were grown at 37°C, except when *radC102 ruv* and *recG ruv* strains were involved (see Fig. 1), in which case all cultures were incubated at 32°C for all steps of the experiment to prevent faster-growing suppressor mutants from accumulating (17).

Construction of a *radC*$^+$ plasmid and DNA sequence analysis. The entire *radC*$^+$ gene (as annotated in the *E. coli* genome sequence [Swiss-Prot no. P25531,3]) and its promoter region (14) were amplified by PCR from *E. coli* SMR4562 cells using primers 5′CGTAGTGGTATAGAAGTGACCAGTA3′ and 5′ACCAGAAACCGCCTGCAAGCTAAGT3′. This product was cut at an *Aat*II site flanking the *radC* gene and ligated as a 1,489-bp *Aat*II blunt fragment into *Aat*II-*Sma*I digested pLG338-30, a pSC101-derived low-copy-number plasmid (5). The resulting *radC*$^+$ plasmid was designated pMJ10. DNA sequencing confirmed that the entire fragment cloned is identical to the sequence in the published *E. coli* genome (3). Cloning of the same PCR product into *Aat*II-*Sca*I-digested pBR322 (a higher-copy-number plasmid) gave small sickly transformants, suggesting that *radC*$^+$ is toxic in high copy number. Similar toxicity in high copy was reported for a plasmid carrying the complete *radC*$^+$ gene and flanking sequences (10). One *radC* plasmid previously reported to complement the UV sensitivity of the *radC102* mutation does not contain the entire ORF (a site

TABLE 1. *E. coli* K-12 strains

Strain	Genotype	Source and/or reference
BW229	*pyrE70 rfa-209*::Tn*10 lac rpsL gltS metB thi*	*E. coli* Genetic Stock Center (Yale University)
CS85	*ruvC53 eda-51*::Tn*10 his-4 argE3 leuB6 proA2 thr-1 thi-1 rpsL31 galK2 lacY1 ara-14 xyl-5 mtl-1 kdgK51 supE44 tsx-33*	34
FC29	Δ(*lac-proB*)$_{XIII}$ *ara thi*/F′ Δ*lacIZ proAB*$^+$	4
GY8322	Δ(*srlR-recA*)*306*::Tn*10 sfiA11* (*sulA*) *thr-1 ara-14 leuB6* Δ(*gpt-proA*)*62 lacY1 tsx-33 qsr′ supE44 galK2 hisG4 rfbD1 mgl-51 rpsL31 kdgK51 xylA5 mtl-1 argE3 thi-1*/mini-F K5353 (*recA*$^+$)	S. Sommer; ENZ280 (6) carrying the K5353 mini-F plasmid (7)
RDK2641	*ruvA59*::Tn*10 recB21 recC22 sbcB15 sbcC201 hsdR*(r_K^- m_K^+)[a]	R. Kolodner
RSH154	SMR4562 *ruvA59*::Tn*10*	17
RSH316	SMR4562 *recG258*::Tn*10*mini-*kan*	17
SMR624	SMR4562 Δ(*srlR-recA*)*306*::Tn*10*	16
SMR2041	SMR4562 *recG258*::Tn*10*mini-*kan ruvC53 eda-51*::Tn*10*	15
SMR4562	Δ(*lac-proB*)$_{XIII}$ *ara thi* Rif^r/F′ *lacI33*Ω*lacZ proAB*$^+$	Identical in genotype to FC40 (4)
SMR5426	SMR4562 *pyrE70 rfa-209*::Tn*10*	Tet^r Pyr$^-$ transductant of P1(BW229) × SMR4562
SMR5441	SMR4562 *radC102*	Pyr$^+$ Tet^s UV^s transductant of P1(SR1187) × SMR5426
SMR5447	SMR4562 *radC102* Δ(*srlR-recA*)*306*::Tn*10*	Tet^r UV^s transductant of P1(GY8322) × SMR5441
SMR5509	SMR4562 *radC102 ruvA59*::Tn*10*	Tet^r UV^s transductant of P1(RDK2641) × SMR5441
SMR5517	SMR4562 *ruvC53 eda-51*::Tn*10*	Tet^r UV^s transductant of P1(CS85) × SMR4562
SMR5518	SMR4562 *radC102 ruvC53 eda-51*::Tn*10*	Tet^r UV^s transductant of P1(CS85) × SMR5441
SMR5677	SMR4562/pLG338-30 (Amp^r)	Amp^r transformant of pLG338-30 into SDMR4562
SMR5678	SMR4562/pMJ10 (*radC*$^+$ Amp^r)	Amp^r transformant of pMJ10 into SMR4562
SMR5681	SMR5441/pLG338-30	Amp^r transformant of pLG338-30 into SMR5441
SMR5682	SMR5441/pMJ10	Amp^r transformant of pMJ10 into SMR5441
SMR5685	RSH316/pLG338-30	Amp^r transformant of pLG338-30 into RSH316
SMR5686	RSH316/pMJ10	Amp^r transformant of pMJ10 into RSH316
SR1187	*radC102 thr-1 leu-6 proA2 his-4 thi-1 lacY1 galK2 ara-14 xyl-5 gpt mtl-1 txs-33 strA31 supE44*	N. Sargentini (11)

[a] This genotype may not be complete.

within the *radC* ORF was used as the cloning site) (10). A predicted RadC protein of 99 amino acids was proposed to be expressed from that plasmid (8, 10).)

The chromosomal *radC* and *recG* genes of several strains (see Results and Discussion) were sequenced using PCR-generated templates (Lone Star Labs, Inc., Houston, Tex.). Primers for *radC* amplification were the same as those used for cloning (see above). Primers for *recG* amplification were 5′AGCAACAAC GCCTGTTGTTTGAAG3′ and 5′GTGATGAATCGCATCCGGCAGGAA3′. Additional primers were designed for sequencing. The absence of the reported frameshift mutation in the *radC102* strain SR1187 (9) was confirmed by sequencing across the mutation site in five independent PCR products.

RESULTS AND DISCUSSION

***radC102* greatly elevates stationary-phase mutation of a *lac* frameshift allele.** Stationary-phase (or adaptive) mutation in the *lac* frameshift assay system requires the recombination proteins RecA, RecBC, and RuvABC (13, 16, 17). In this mutation assay (4), cells carrying a *lac* frameshift allele on an F′ plasmid are placed on lactose minimal medium and Lac$^+$ mutant colonies are scored each day for several days. New colonies appear each day due to mutations that occur on the selective plate and not prior to plating (27). The recombination gene dependence (13, 16, 17), sequence spectrum (12, 31), and other features of Lac$^+$ stationary-phase mutation support the hypothesis that the mutations are formed by DNA polymerase errors during synthesis primed from recombination intermediates (16, 30). We wondered whether *radC102* might affect stationary-phase mutation, given its effects on UV sensitivity (11) and recombination between tandem repeats (33). We find that *radC102* dramatically increases the frequency of Lac$^+$ mutations in this assay (Table 2). The increase is completely dependent on the RecA protein (Table 2). These phenotypes of *radC102* are very similar to those of *recG* mutations, which also stimulate RecA-dependent Lac$^+$ stationary-phase mutation strongly (13, 17).

***radC102 ruv* mutants are extremely UV sensitive.** We constructed strains carrying *radC102* and mutations in *ruvA* or *ruvC*. Both of these strains are extremely UV sensitive, as

TABLE 2. *radC102* elevates RecA-dependent stationary-phase mutation of a *lac* frameshift allele

Relevant genotype[b]	Lac$^+$ colonies per 10^8 cells plated[a]					
	Expt 1			Expt 2		
	Day 2	Day 2–5	Fold difference	Day 2	Day 2–5	Fold difference
rec$^+$	9.4 ± 2.4	200 ± 35	1	13 ± 2.5	220 ± 30	1
radC102	10 ± 7	5,000 ± 850	25	64 ± 19	6,400 ± 610	29
recA	0.95 ± 0.30	5.2 ± 0.52	0.026	3.0 ± 0.77	14 ± 1.8	0.064
radC102 recA	1.2 ± 0.37	4.3 ± 0.61	0.022	4.3 ± 1.9	27 ± 7	0.12

[a] See Materials and Methods. The values given are the average ± one standard error of the mean from experiments with six independent cultures of each strain. The effects of *radC102* correspond closely to those obtained by Harris et al. (17) and Foster et al. (13) with the *recG258*::Tn*10*mini-*kan* allele. Values for days 2 to 5 are the total number of Lac$^+$ colonies on day 5, and the fold differences of the values for days 2 to 5 relative to *rec*$^+$ are also given.

[b] From top to bottom, these strains are SMR4562, SMR5441, SMR624, and SMR5447.

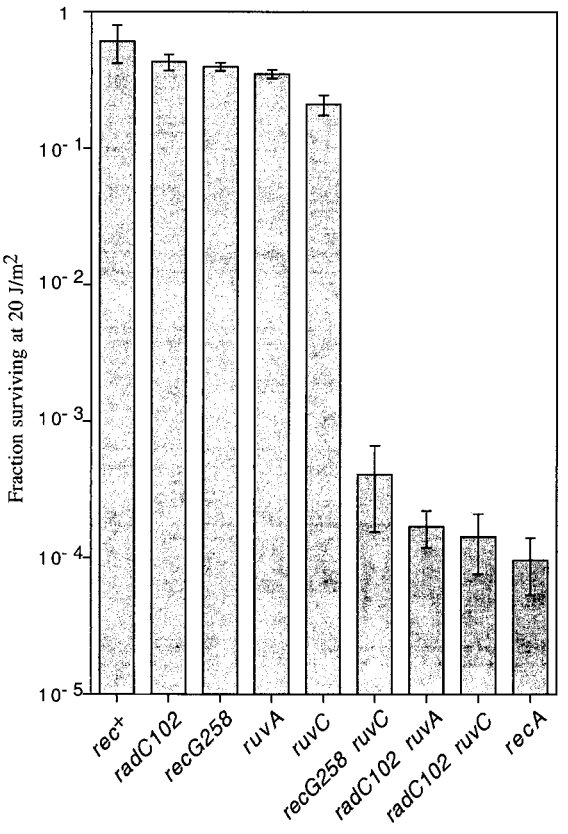

FIG. 1. *radC102* confers extreme UV sensitivity in combination with *ruv* mutations. Values are averages from one experiment with three independent cultures of each strain. The error bars represent one standard deviation. Similar results were obtained in another experiment (data not shown). All strains were grown at 32°C to prevent accumulation of suppressor mutants or revertants (detected as heterogeneity of colony size and UV resistance) in the double *ruv recG* mutants (17). The *eda-51*::Tn*10* transposon linked with *ruvC53* has no phenotype on its own (data not shown). The strains are (from left to right): SMR4562, SMR5441, RSH316, RSH154, SMR5517, SMR2041, SMR5509, SMR5518, and SMR624.

shown in Fig. 1. *radC102* strains carrying *ruvA* or *ruvC* mutations are as sensitive as the *recA* deletion strain and as a *recG258 ruvC53* strain at this dose. In contrast, *radC102* or the single *ruv* mutations alone cause little or no UV sensitivity at this dose. Extreme UV sensitivity is a phenotype of *recG258 ruv* strains (18). Thus, *radC102* also behaves like a *recG* mutation in combination with *ruv* mutations. In addition, the *radC102 ruv* strains show an absence of stationary-phase mutation (data not shown), as do the *recG258 ruv* combinations (13, 17).

The *radC102* mutation is in the *recG* gene. The striking similarity between *radC102* and *recG* in stationary-phase mutation and in UV sensitivity led us to consider that, contrary to published work (9, 10), *radC102* might be an allele of the nearby *recG* gene. We sequenced the entire *radC* coding region (Swiss-Prot accession no. P25531) in two isolates of the *radC102* strain SR1187 (obtained on separate occasions from N. Sargentini [9]) in SMR5441 (*radC102*) and in SMR4562 (*radC*$^+$). SR1187 is the source of the *radC102* allele for the experiments reported here and those of Saveson and Lovett (33). All of these *radC* sequences were identical to that in the genome sequence. None displayed the frameshift mutation in the *radC* ORF that was reported to be the *radC102* mutation in SR1187 (9). The *recG* genes of the two *radC102* strains (SR1187 and SMR5441) and the *radC*$^+$ strain SMR4562 were also sequenced completely. The *radC102* strains both contain a substitution mutation in *recG* (TGG to TGA) that changes Trp411 to a stop codon, predicting translation of a truncated RecG of 410 amino acids rather than 603 amino acids. Based on these data, we conclude that *radC102* is an allele of *recG*.

The *radC102* allele and *recG258* alleles of *recG* have similar UV sensitivity phenotypes. Because *radC102* had not been compared directly with *recG* alleles previously, we assayed the UV sensitivities of a set of isogenic *radC102*, *recG258*::Tn*10*mini-*kan*, and *recG*$^+$ strains (Fig. 2). The *radC102* strain is as sensitive to UV as the strain carrying *recG258*::Tn*10*mini-*kan*, a commonly used null allele of *recG* (20).

Effect of *radC* overexpression on UV sensitivity. With the above findings, the evidence that *E. coli radC* encodes a DNA repair gene becomes limited to the report that expression of the complete *radC*$^+$ gene (and of a fragment of *radC*) from a low-copy-number vector complements the UV sensitivity of the *radC102* (*recG*) mutation (10). The plasmid carrying the complete *radC*$^+$ gene was also reported to confer UV sensitivity to a wild-type strain (10). We cloned the complete *radC*$^+$ gene on a related low-copy-number vector and found that this *radC*$^+$ plasmid does not alter the UV sensitivity of either a *radC102* strain, a *recG258*::Tn*10*mini-*kan* strain, or a *recG*$^+$ strain (Fig. 3). This result is consistent with our finding that the *radC102* mutation affects the *recG* gene. The apparent conflict between these overexpression results and those reported pre-

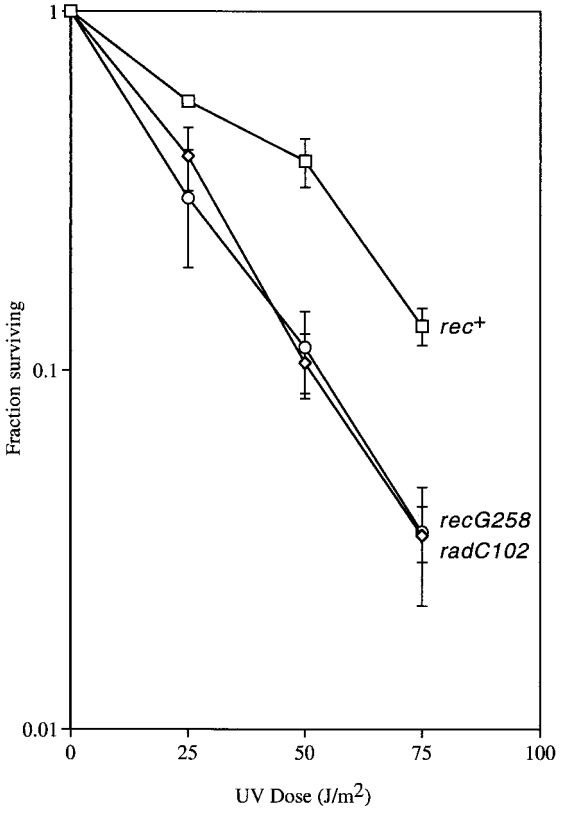

FIG. 2. *radC102* and *recG258* confer similar UV sensitivities. Values are the averages from one experiment with three independent cultures. The error bars represent one standard deviation. A similar result was obtained in a second experiment (data not shown). The strains are as follows: *recG*$^+$ (*rec*$^+$), SMR4562; *radC102*, SMR5441; *recG258*::Tn*10*mini-*kan* (*recG258*), RSH316.

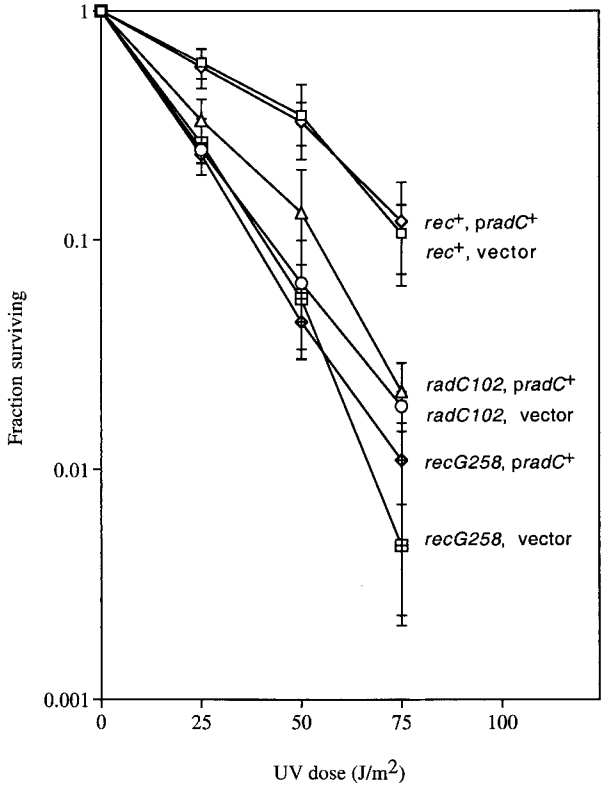

FIG. 3. Effect of p*radC*$^+$ on UV sensitivity. Values are averages from one experiment with three independent cultures of each strain. The error bars represent one standard deviation. Within each set of strains (*rec*$^+$, *radC*, and *recG258*), the error bars are overlapping, with the exception of the *radC102* set at the 25 J/m^2 dose. Similar results were obtained in a second experiment (data not shown). The strains used are (from top to bottom) SMR5678, SMR5677, SMR5682, SMR5681, SMR5686, and SMR5685.

viously (10) might be due to differences between the plasmid constructs. An observed high-copy toxicity of the *radC*$^+$ gene (see Materials and Methods), similar to that seen previously (10), suggests that *radC*$^+$ is expressed.

Implications for RecG and RadC function. The *radC102* mutation causes a large decrease in tandem repeat recombination stimulated by a *dnaB107* mutation (33). Our results demonstrating that *radC102* is an allele of *recG* indicate that RecG is required for that RecA-dependent process. The *dnaB107*-stimulated recombination assay (33) provides an example in which the RuvABC and RecG systems appear to play dramatically different roles in vivo, in contrast to their overlapping functions in recombination of linear DNA substrates (18). The *ruv dnaB107* combination is inviable, suggesting that RecG cannot process some lethal recombination substrate(s) created in the *dnaB107* strains or that RecG processes them to lethal intermediates (33). In contrast, the *radC102 dnaB107* combination appears to be viable (33), indicating that RuvABC deals efficiently with the potentially lethal recombination substrates produced in a *dnaB107* strain lacking RecG.

There are several other recombinational assay systems in which RecG and RuvABC appear to play different roles, including recombination in the RecF pathway (19, 20), recombination-dependent stationary-phase Lac$^+$ mutation (13, 17), homeologous recombination (25), λ Red-mediated recombination (29), and double-strand break repair (M. Motamedi and S. M. Rosenberg, unpublished results). In stationary-phase Lac$^+$ mutation, RuvA, -B, and -C are required for mutation and RecG is inhibitory (13, 17). In that system, these opposing roles are proposed to reflect different effects of RuvABC and RecG on initiation of DNA synthesis from 3′ strand invasion intermediates, with RuvABC facilitating and RecG inhibiting due to opposite polarities of branch migration (17). This proposal is supported by work with the λ Red system, a known 3′ end invasion system inhibited by RecG and requiring RuvC (29).

The distinct functions of the Ruv and RecG systems in vivo are likely to be dictated by two classes of factors. First, their intrinsic properties, such as different polarities of branch migration (37, 40) or different affinities for particular recombination substrates such as D-loops and three-stranded junctions (26, 37), will govern which substrates can be bound and whether branch migration will facilitate or abort the reaction. Second, competition with other proteins for recombination intermediates will help define which substrates will be accessible. For example, genetic and biochemical evidence indicates that RecG and PriA, a primosome assembly protein with important roles in replication restart and double-strand break repair (reviewed in reference 32), probably compete for D-loop recombination intermediates in vivo (1, 26). Further investigation of assay systems in which RuvABC and RecG have differential effects will help to reveal their functions and substrates in vivo.

With the finding that the *radC102* mutation affects *recG*, the evidence that *E. coli* RadC is involved in DNA repair becomes very limited, as discussed above. Construction and characterization of *radC* mutations will be necessary to reveal its function and to provide clues to the function of the multiple bacterial *radC* homologs. These include three *E. coli* homologs (*ykfG*, *yfiY*, and *yeeS*), which lack the putative helix-hairpin-helix DNA binding motif in the N terminus of RadC (2).

ACKNOWLEDGMENTS

We thank N. Sargentini for *radC102* strains, R. Montelaro for pLG338-30, the *E. coli* Genetic Stock Center for bacterial strains, and M. Price for medium preparation.

This work was supported by National Institutes of Health grants F32-GM19909 (to M.-J. L.), R01-GM53158, and R01-AI43917.

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