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Conjugational Recombination in Resolvase-Deficient *ruvC* Mutants of *Escherichia coli* K-12 Depends on *recG*

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***ruvC* mutants of *Escherichia coli* appear to lack an activity that resolves Holliday intermediates into recombinant products. Yet, these strains produce close to normal numbers of recombinants in genetic crosses. This recombination proficiency was found to be a function of *recG*. A "mini-kan" insertion in *recG* was introduced into *ruvA*, *ruvB*, and *ruvC* strains. Conjugational recombination was reduced by more than 100-fold in *recG ruvA*::Tn*10*, *recG ruvB*, and *recG ruvC* strains and by about 30-fold in a *recG ruvA* strain carrying a *ruvA* mutation that is not polar on *ruvB*. The double mutants also proved very deficient in P1 transduction and are much more sensitive to UV light than *ruv* single mutants. Since mutation of *recG* alone has very modest effects on recombination and sensitivity to UV, it is concluded that there is a functional overlap between the RecG and Ruv proteins. However, this overlap does not extend to circular plasmid recombination. The possibility that RecG provides a second resolvase that can substitute for Ruv is discussed in light of these findings.**

Genetic recombination in *Escherichia coli* K-12 depends normally on RecA protein to promote the synaptic stage when homologous DNA molecules pair and exchange strands (7, 30). In vitro, RecA catalyzes the formation of an intermediate, commonly referred to as a Holliday junction, in which the two interacting duplexes are held together at the point of strand crossover (11). Resolution of this intermediate into recombinant products in vivo is generally assumed to involve cleavage of the DNA by a junction-specific nuclease. Connolly and West (9) succeeded in identifying such an activity in *E. coli* cell extracts. Genetic and biochemical studies indicate that this activity is the product of the *ruv* genes (2, 8).

Earlier studies had linked the *ruv* locus with DNA repair and recombination. Mutations in any of the three closely linked genes designated *ruvA*, *ruvB*, and *ruvC* (32) increase sensitivity to UV light, ionizing radiation, and mitomycin C (13, 16, 29, 31). They also reduce recombination in *recBC sbcA* and *recBC sbcB sbcC* genetic backgrounds but not to any great extent in rec^+ sbc^+ strains (15, 16, 20, 29). *ruvA* and *ruvB* form a LexA-regulated operon (3, 34, 36) and are components of the inducible SOS system of DNA repair (41). The *ruvC* gene is located just upstream of *ruvAB* in a separate operon that is not regulated by LexA (32, 33). All three *ruv* gene products have been overexpressed and purified. RuvC is a nuclease that acts specifically to cleave Holliday intermediates (8). RuvA binds to DNA and especially to molecules containing synthetic Holliday junctions (10, 35). RuvB is an ATPase that interacts with RuvA (10, 12). Given the very similar phenotypes of *ruvA*, *ruvB*, and *ruvC* mutants, it is tempting to think that RuvAB might assist RuvC in cleaving Holliday junctions in vivo. However, there is no direct evidence to support this view.

The fact that *ruv* single mutants are reasonably proficient in recombination is therefore highly significant. It suggests that *E. coli* has an alternative activity capable of resolving Holliday junctions. To try to identify this activity, mutations in other genes known to be involved in recombination and DNA repair were introduced into *ruv* mutants and the strains constructed were examined for their ability to produce recombinants in genetic crosses. The results reported here show that conjugational and transductional recombination in *ruv* mutants depends on *recG*.

MATERIALS AND METHODS

Strains and plasmids. The *E. coli* K-12 strains used are listed in Table 1. *ruvA60*::Tn*10* has a polar effect on *ruvB*. Strains carrying this insertion are deficient therefore in both RuvA and RuvB (32). pRDK41 is a pBR322 dimer carrying two mutant copies of the Tc^r gene and confers resistance to ampicillin (14).

Media and general methods. Luria-Bertani (LB) broth and 56/2 salts media have been described previously (22). The LB media contained 0.5 g of NaCl per liter except for matings, for which the salt concentration was increased to 10 g/liter. Broth and agar media were supplemented with 20 μg of tetracycline per ml, 40 μg of kanamycin per ml, or 50 μg of ampicillin per ml, as required for strains carrying antibiotic-resistant plasmids or transposons. Plasmid transformations and methods for measuring sensitivity to UV light have been described before (17, 23, 32). UV irradiation was at a dose rate of 1 $J/m^2/s$.

Genetic crosses and measures of recombination. F′ and Hfr donors were mated with F^- recipients in high-salt LB broth at 37°C by procedures described in detail elsewhere (21, 23). Measures of cell viability relate to the number of CFU in the recipient cultures at an A_{650} of 0.4. Transconjugants were selected on 56/2 or LB agar, as appropriate, supplemented with 100 μg of streptomycin per ml to counterselect donor cells. Plasmid recombination was measured by scoring the formation of Ap^r Tc^r cells in strains transformed with pRDK41 (14). For transductions with phage P1*vir*, the recipes and protocols described by Miller (28) were followed.

RESULTS

A "mini-kan" insertion in *recG* (*recG258*) was introduced into a series of closely related strains carrying mutations in

TABLE 1. *Escherichia coli* K-12 strains used in this study

Strain	Relevant genotype[a]	Source or reference
AB1157	*rec*$^+$ *ruv*$^+$[b]	1
AB2463	*recA13*[b]	1
W3110	IN(*rrnD-rrnE*)*1*	1
SR2210	*ruvA200 eda-51*::Tn*10*[b]	31
HI24	*ruvB4*[b]	29
CS85	*ruvC53 eda-51*[b]	37
CS140	*ruvC53 eda*? (Tc^s)[b]	37
N1373	F$^-$ *ruvC51 hisG4 argE3 thi-1 thrB1007 mtl-1 xyl-5 rpsL31 gyrA262 supE44*? *tsx-33*?	37
N2057	*ruvA60*::Tn*10*[b]	37
N2731	*recG258*::Tn*10* mini-kan[b]	18
N3395	*recG258 ruvB4*[b]	P1.N2731 × HI24 to Km^r
N3396	*recG258 ruvA60*[b]	P1.N2731 × N2057 to Km^r
N3397	*recG258 ruvA200 eda-51*[b]	P1.N2731 × SR2210 to Km^r
N3398	*recG258 ruvC53 eda-51*[b]	P1.N2731 × CS85 to Km^r
N3475	F$^-$ *recG258 ruvC51 hisG4 argE3 thi-1 thrB1007 mtl-1 xyl-5 rpsL31 gyrA262 supE44*? *tsx-33*?	P1.N2731 × N1373 to Km^r
N3476	*recG258 ruvC53*	P1.N2731 × CS140 to Km^r
KL548	F′ (F128) *lacI3 lacZ118 proAB*$^+$	K. B. Low, 18
KL226	Hfr (Cavalli, PO2A) *relA1 tonA22*	K. B. Low
GY2200	Hfr (Hayes, PO1) (λind$^-$)$^+$ *thi-1 relA1*	R. Devoret

[a] After the first full listing, transposon insertions are abbreviated to the allele number.

[b] These strains are also F$^-$ *thi-1 his-4* Δ(*gpt-proA*)*62 argE3 thr-1 leuB6 kdgK51 rfbD1*(?) *ara-14 lacY1 galK2 xyl-5 mtl-1 tsx-33 supE44 rpsL31*.

ruvA, *ruvB*, or *ruvC* (Table 1). The constructs made are very sensitive to UV light, much more so than the corresponding *ruv* single mutants (Fig. 1). At low UV doses in the range of 1 to 5 J/m^2, the *recG* insertion reduced the survival of *ruvA60*, *ruvB4*, and *ruvC53* strains by 50- to 500-fold. The effect is particularly striking since *recG258* alone confers very little sensitivity in this dose range, as was reported previously (18). *recG258* was also introduced into the *ruvC51* strain, N1373. The double mutant proved to be very much more sensitive than the *ruvC51* parent (data not shown). The *recG* insertion also increases the sensitivity of a *ruvA200* strain, but the effect is not as great as with the *ruvA60*::Tn*10* strain. *ruvA200* differs from *ruvA60* in that it does not have a polar effect on *ruvB* (31). From these data, it appears that RecG is not quite as vital for repair of UV damage in the absence of RuvA as it is in the absence of RuvB or RuvC. However, we cannot exclude the possibility that *ruvA200* is a leaky mutation.

The same *recG ruv* double mutants were also tested for conjugational and transductional recombination. From the data shown in Table 2, it is clear that the yield of recombinants from Hfr crosses with *recG ruvA60*, *recG ruvB4*, and *recG ruvC53* strains is reduced by more than 100-fold, even after allowing for reduced viability. Zygotic induction of prophage λ in the Hfr GY2200 control crosses was nearly as efficient with these strains as with the *rec*$^+$ *ruv*$^+$ control, while the yield of F′ Pro$^+$ transconjugants in crosses with strain KL548 was reduced by no more than expected from the reduced viability. From these data, it seems quite clear that the *recG ruv* strains are very deficient in conjugational recombination. This contrasts sharply with the results for single mutants, in which recombination is reduced by 2.5-fold at most. Again, the *recG ruvA200* strain has a less extreme phenotype in that the yield of recombinants in Hfr crosses is reduced by no more than about 30-fold. The particular construct studied also shows a deficiency in zygotic induction of λ. The reason for this deficiency is

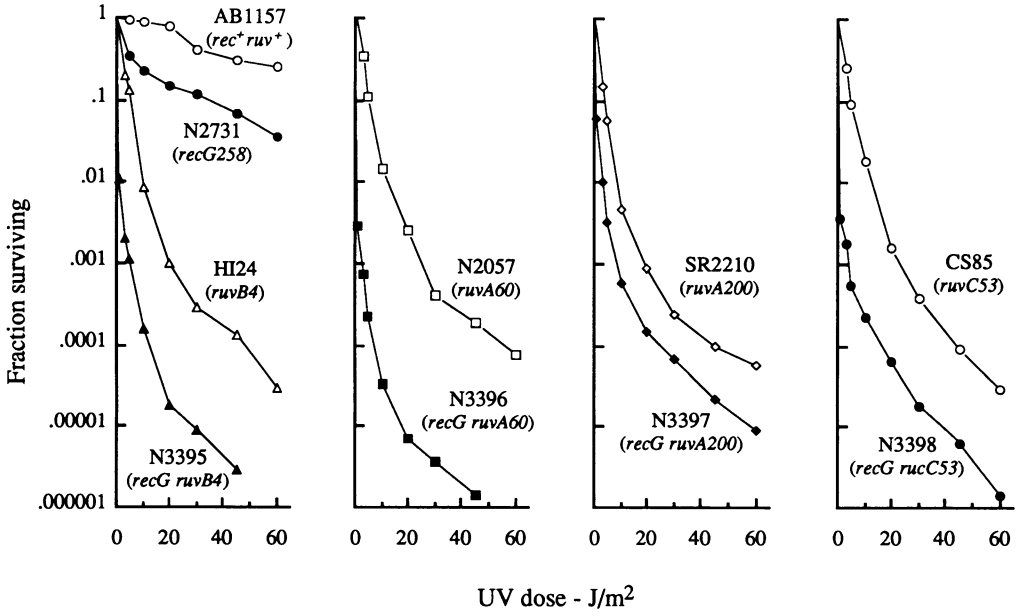

FIG. 1. UV irradiation survival of strains carrying combinations of *recG* and *ruv* mutations. The strains used and relevant genotypes are identified within each graph.

TABLE 2. Effect of *recG* and *ruv* on conjugational and transductional recombination

Strain and relevant genotype	Viability[a]	Relative yield of transconjugants or transductants per cross with donor strain				
		KL548 (F′ Pro$^+$)	Hfr GY2200[b]		Hfr KL226 (Pro$^+$)	P1.W3110 (Leu$^+$)
			(λ plaques)	(Thr$^+$ Leu$^+$)		
AB1157 (*rec*$^+$ *ruv*$^+$)	1.0 (1.8×10^8)	1.0 (1.9×10^7)	1.0 (9.6×10^6)	1.0 (1.4×10^7)	1.0 (1.3×10^7)	1.0 (8.2×10^3)
N2731 (*recG*)	0.86	0.78	0.99	0.37	0.32	0.11
HI24 (*ruvB4*)	0.61	0.27	1.17	0.28	0.31	0.28
N3395 (*recG ruvB4*)	0.22	0.11	0.68	0.0015	0.002	<0.00074
N2057 (*ruvA60*)	0.51	0.48	0.87	0.38	0.33	0.13
N3396 (*recG ruvA60*)	0.23	0.14	0.67	0.0018	0.0016	0.00059
SR2210 (*ruvA200*)	0.56	0.53	0.17	0.39	0.36	0.13
N3397 (*recG ruvA200*)	0.43	0.27	0.061	0.011	0.014	<0.0012
CS85 (*ruvC53*)	0.51	0.49	0.98	0.24	0.17	0.043
N3398 (*recG ruvC53*)	0.23	0.19	0.67	0.0025	0.00089	0.00052

[a] Mating was for 30 min (KL548), 40 min (KL226), or 60 min (GY2200); the transconjugant or transductant class selected is shown in parentheses. The standard errors for the values shown (means of two to four experiments) vary from 2 to 35% of the mean. Actual values for AB1157 (in parentheses) are per milliliter of recipient culture (viability) or mating mixture (transconjugants) or per 10^9 P1 phage adsorbed (transductants).

[b] λ plaques arise from zygotic induction of the Hfr prophage.

uncertain since the results of the F′ cross indicate that DNA transfer is not seriously affected.

P1 transductional crosses (Table 2) showed a similar trend. The *recG ruv* strains gave very few or no transductants. Indeed, from the control platings with uninfected cells (data not shown), the few Leu$^+$ colonies scored could well have been revertants. Transduction was also reduced to some extent in the single mutants, particularly with the *recG* (8-fold) and *ruvC* (12-fold) strains. The effect of *ruvC* on transduction was noted previously (40) in a strain (R7061) subsequently discovered to carry the *ruvC51* allele (37).

The involvement of *recG* and *ruv* mutations in circular plasmid recombination was measured by using pRDK41, a pBR322 dimer that carries two mutant copies of the tetracycline resistance gene (14). Recombination between these two genes in vivo gives rise to Tc^r cells. From the data presented in Table 3, it is clear that mutations in *ruv* reduce the frequency of recombination in this system. Recombination in the *ruvB* strain, HI24, was reduced by a rather modest threefold. However, this effect is greater than was reported previously for a *ruvB9* strain (14). The greatest reduction (20-fold) was observed with the *ruvC51* strain, N1373. In comparison, recombination in a *recA* strain was reduced by about 70-fold. *recG258* does not appear to affect recombination in this system, as was reported previously (18). Furthermore, inactivation of *recG* made very little difference in the frequency of recombination in the *ruv* mutants tested.

DISCUSSION

The genes involved with homologous recombination in *E. coli* have been defined as acting in the RecBCD, RecE, or RecF pathway depending on whether they are needed for the formation of recombinants in conjugational crosses with wild-type, *recBC sbcA*, or *recBC sbcB sbcC* strains (7, 27). According to this concept, the *ruv* genes would be classified as components of the RecF pathway and would be expected therefore to play a minor role in the formation of recombinants in the wild type, in which the RecBCD pathway predominates (4–6).

The results presented here suggest otherwise. They show that the recombination-proficient phenotype of *ruv* mutants is a function of *recG*. Since recombination proceeds reasonably efficiently in both *recG* and *ruv* strains, this observation suggests a functional overlap between the gene products and a much more critical role for these proteins in the formation of recombinants in the wild type than can be deduced from the properties of the single mutants. To continue with the notion that the *ruv* genes are components of the RecF pathway would require the addition that mutation of *recG* blocks the RecBCD pathway at a stage at which the DNA substrates can be diverted into the RecF pathway. This assumption would be difficult to reconcile with the facts that mutation of *recG* causes a substantial reduction in recombination in a *recBC sbcBC* genetic background and that

TABLE 3. Effect of *recG* and *ruv* on plasmid (pRDK41) recombination

Strain and relevant genotype	No. of transformants tested[a]	Mean no. of viable Ap^r cells per ml	Mean % Tc^r cells (relative yield)
AB1157 (*rec*$^+$ *ruv*$^+$)	8	1.88×10^8	0.097 ± 0.035 (1.0)
AB2463 (*recA13*)	6	0.82×10^8	0.0014 ± 0.00019 (0.014)
N2731 (*recG*)	9	1.20×10^8	0.11 ± 0.03 (1.1)
HI24 (*ruvB4*)	9	0.95×10^8	0.029 ± 0.01 (0.30)
N3395 (*recG ruvB4*)	9	0.40×10^8	0.015 ± 0.0038 (0.15)
CS140 (*ruvC53*)	11	1.42×10^8	0.014 ± 0.0028 (0.14)
N3476 (*recG ruvC53*)	10	0.62×10^8	0.021 ± 0.0023 (0.22)
N1373 (*ruvC51*)	9	0.71×10^8	0.0054 ± 0.0018 (0.055)
N3475 (*recG ruvC51*)	8	0.29×10^8	0.0077 ± 0.0015 (0.079)

[a] Transformant colonies were inoculated into 8.0 ml of LB broth supplemented with ampicillin and grown to an A_{650} of 0.4 (approximately 2×10^8 total cells per ml as determined microscopically) before being assayed for Ap^r cells and for Ap^r Tc^r cells.

mutations in other genes (*recF*, *recJ*, *recN*, *recQ*) of the RecF pathway do not prevent recombination in *recG* strains (18). The functional overlaps described here are reminiscent of others that have been detected between *recD* and *recJ* (23, 26) and between these two genes and *recN* (19). Perhaps the concept of pathways should continue to guide the study of *E. coli* recombination (38, 39) no more.

The fact that *recG* is needed for recombination in *ruvA*, *ruvB*, and *ruvC* mutants is consistent with the hypothesis that all three *ruv* genes are involved in the resolution of Holliday intermediates in vivo (8). If RuvA and RuvB have other activities, then RecG must be able to substitute for these as well. The functional overlap with RuvC raises the intriguing possibility that *recG* specifies an alternative resolvase or at least a nuclease that can function as such in the absence of RuvC. *recG* has been cloned (18) and sequenced (15). It encodes a poorly expressed protein of 76 kDa, not 90 kDa as was incorrectly reported previously (18). Whatever the activities of this protein, it is clear that they do not overlap perfectly with those of the Ruv proteins. This is apparent from the slight deficiencies in recombination seen in the single mutants and also from the sensitivity of these strains to UV damage. The Ruv proteins in particular seem to have a role in the repair of UV damage that cannot be compensated for by RecG. It also appears that RecG is not needed for circular plasmid recombination, even in *ruv* mutants. In the assays conducted, substantial numbers of Tc^r (recombinant) plasmids are formed in the absence of RecA. It is therefore not too surprising that *recG ruv* double mutants produce a considerable number of recombinant plasmids in these assays.

The idea that the RecG and Ruv proteins have somewhat different roles and compensate rather imperfectly for one another may explain why *recG* and *ruv* mutations have more extreme phenotypes in *recBC sbcB* strains. The progress of recombination appears to be slower than normal in this genetic background (24), and the formation of recombinants is generally more sensitive to changes in the availability of recombination enzymes (24, 25).

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