GENETIC ANALYSIS OF MUTATIONS INDIRECTLY SUPPRESSING recB AND recC MUTATIONS

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ABSTRACT

Mutations in sbcB inactivate exonuclease I and suppress the UV-sensitive, mitomycin-sensitive, recombination-deficient phenotypes associated with recB and recC mutations. Mapping experiments have located sbcB about 0.4 minutes from the his operon at 38.0 on the standard map of E. coli. This places sbcB between supD and his. A four-point cross shows that sbcB lies between P2 attH and his. P2 eduction deleting the his operon beginning with P2 attH also deletes sbcB and produces the expected exonuclease I deficiency and suppression of recB-. The occurrence of chemical-mutagen-induced and spontaneous mutations indirectly suppressing recB- and recC- is examined. Three lines of strains produce only sbcA mutations while only sbcB mutations occur in a fourth line. Explanations for this behavior are proposed in light of the ability of the first three lines to express sbcB mutations which they inherit by transduction.

MUTATIONS in the recA gene of Escherichia coli K12 virtually abolish the ability to perform recombination following conjugation or transduction (CLARK 1967). In crosses with various Hfr strains progeny formed by recombination arise from zygotes to which the recA+ allele has been transferred (CLARK 1967; WILLETTS, CLARK and Low 1969). Progeny formed from zygotes not receiving the recA+ allele are most often formed by replication; i.e., by inheriting autonomous F' elements (Low 1968). recB or recC mutants, on the other hand, perform substantial amounts of residual recombination even though the conjugal zygotes have not received the recB+ or recC+ alleles (CLARK 1967; Low 1968).

Since this residual recombination might result from a pathway of recombination alternative to that involving recB and recC, revertants of recB and recC mutants were examined for indirect suppression. Two different kinds of indirect suppression were found. The first appeared to result from the appearance of an ATP-independent DNAase produced by mutations called sbcA- (BARBOUR et al. 1970). The second resulted from the loss of exonuclease I activity produced by mutations called sbcB- (KUSHNER et al. 1971). In a previous publication (KUSH-
### TABLE 1

**Characteristics of bacterial strains***

<table>
<thead>
<tr>
<th>Strain Number</th>
<th>recB</th>
<th>recC</th>
<th>sbcB</th>
<th>str</th>
<th>arg</th>
<th>his</th>
<th>thr</th>
<th>leu</th>
<th>trp</th>
<th>ilv</th>
<th>pro</th>
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<td>+</td>
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<td>+</td>
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2. **Hfr strains**

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<th>Strain Number</th>
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<th>arg</th>
<th>his</th>
<th>thr</th>
<th>leu</th>
<th>trp</th>
<th>ilv</th>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>supD-, PO44</td>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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3. **F' strain**

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<th>thr</th>
<th>leu</th>
<th>trp</th>
<th>ilv</th>
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<td>A2</td>
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<td>1</td>
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* Gene symbols are those used by Taylor (1970). Point of origin designations (PO53 etc.) were obtained from B. Bachmann.
NER et al. 1971) we indicated that sbcB mutations were 30–50% cotransducible with his and that sbcB+ was carried by the F30 episome. In this work we provide data which indicate the location of sbcB relative to his and flanking markers and a method of producing sbcB deletion mutations. We also discuss the occurrence of sbcA mutations.

MATERIALS AND METHODS

Bacterial strains: The strains used in this study are all derivatives of E. coli K12 and are listed in Table 1. Map positions of the relevant genetic markers are shown in Figure 1. P2 phage was obtained from Dr. Melvin Sunshine.

Nomenclature conforms to the recommendations of Demerec et al. (1966) except that the minus sign is used with the gene symbol to indicate a general mutant allele when the specific allele number is not required. The gene symbols are those recorded by Taylor (1970) with the exception of sbc, which stands for a set of genes involved in the indirect suppression of recB21 or recC22. Phenotypic abbreviations are as follows: Rec, recombination; UV, ultraviolet irradiation; Mit, mitomycin; His, histidine; Trp, tryptophan; Leu, leucine; Pro, proline; TL, threonine leucine; Arg, arginine; IV, isoleucine-valine; DES, diethylsulfate; “-”, requiring when used with abbreviations of amino acids and deficient when used with Rec; “+”, independence when

**Figure 1.**—Linkage map of Escherichia coli K12 showing genetic markers relevant to this work. The map is according to Taylor (1970) and gene symbols conform to Demerec et al. (1960). Points of origin of Hfr strains are as cited by Barbara Bachmann (personal communication).
used with abbreviations of amino acids and proficient when used with Rec; R, resistance; S, sensitivity.

Methods: The procedures and media for conjugational and transductional crosses and for the discrimination of Rec- from Rec+ strains have been described previously (Clark 1967; Clark and Margulies 1965; Willetts, Clark and Low 1969). Acridine curing was performed as described by Bastarachea and Willetts (1968). Enzyme assays for exonuclease I were performed as described by Kushner et al. (1971) and for both exonuclease V, and the ATP-independent nuclease appearing in sbcA- strains as described by Barbour and Clark (1970). Thymine-requiring mutants were isolated by the use of trimethoprim (Stacey and Simson 1965).

Diethysulfate (DES) treatment: A saturated solution of DES was made by shaking 0.1 ml DES in 5 ml of minimal medium lacking a carbon source and allowing the undissolved DES to settle. 0.1 ml of a fully grown L broth culture of the strain to be mutagenized was inoculated into the mixture without removing the DES. After 15 min incubation at 37°C an aliquot of 0.1 ml was removed, added to 10 ml of L broth and shaken overnight at 37°C. Appropriate dilutions were plated on supplemented minimal media.

Eduction: P2 treatment to obtain His- eductants was performed as described by Sunshine and Kelley (1971). When sbcB- eductants were desired the method was modified by plating appropriate dilutions on Luria agar containing mitomycin C at a concentration of 1.0 microgram per ml rather than on the low histidine media used to detect the His- eductants.

Detection of sbcB- and recB- mutations: The presence of sbcB- mutations in a rec+ strain was tested for by growing P1 on the strain in question and using the lysate to transduce a his- recB- recC- sbcB- recipient to His+. Cotransduction of sbcB- with his+ caused no change in the UV8Mit8Rec+ phenotype of the recipient. The presence of recB- in a strain containing sbcB- was tested for by growing P1 on the strain and using the lysate to transduce an argA- recB- sbcB+ recipient to Arg+. Cotransduction of recB- with argA+ yielded a UV8Mit8Rec- phenotype.

RESULTS

Location of sbcB: The strains which carry the sbcB mutations are multiply marked and are of genotype recB- recC- sbcB-. Phenotypically these strains are Rec+ and give rise to Rec- progeny by inheritance of the sbcB+ allele. Since the recB and recC cistrons are near thyA (Emmerson and Howard-Flanders 1967; Willetts and Mount 1969), we can locate the sbcB gene on the chromosome by crossing the triple mutant with an appropriate Hfr, selecting for inheritance of widely spaced markers and screening for those which become Rec- because of their unselected inheritance of sbcB+. The Hfr JC5491 is appropriate because it carries the recB21 and recC22 mutations and transfers them one to two min from its point of origin PO45 (Figure 1) and because it is Rec- by virtue of its nonsuppressed (i.e. sbc+) genotype. When His+ [IV+] recombinants sired by JC5491 were selected about 70% were found to have inherited the Rec- phenotype. Since the recipients were Rec+ phenotypically but recB-recC-sbcB- genotypically, the only way they could inherit the Rec- phenotype was by inheriting sbcB+ from the donor. In parallel crosses of the Hfr KL98, which is recB+recC+ but transfers these cistrons late in conjugation, and our recB-recC-sbcB- strains we found between 65% and 82% coinheritance of sbcB+ with his+.

The strong linkage between his and sbcB observed in conjugational crosses led us to suspect that cotransduction of the two might be observed. AB259 was assumed to be a suitable wild-type, i.e. his+ sbcB+, strain. P1 grown on this donor strain were used to transduce four his-sbcB-recB-recC- strains to His+ and Pro+. As shown in Table 2 between 51% and 62% of the His+ transductants had
Table 2

Cotransduction* of sbcB+ with his+

<table>
<thead>
<tr>
<th>Recipient</th>
<th>Genotype</th>
<th>Transduction frequency†</th>
<th>Number tested</th>
<th>Number UV8 Mit8 Recc</th>
<th>Percent sbcB+ cotransduction</th>
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</thead>
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<tr>
<td></td>
<td></td>
<td>His+ Pro+</td>
<td>His+ Pro+</td>
<td>His+      Pro+</td>
<td>His+ Pro+</td>
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<td>- -</td>
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<td>100 95</td>
<td>62 0</td>
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</table>

* P1 virus was grown on AB259 (his+ sbcB+); the transduction was performed as described in Willetts, Clark and Low (1969).
† Number of transductants per 10⁶ input phage.

inherited the sbcB+ allele as judged by their Rec- Mit8 and UV8 behavior. No Rec- UV8 could be found among the approximately 100 Pro+ transductants per strain tested indicating no detectable cotransduction with pro and ruling out various hypotheses to explain the Rec-His+ transductants which do not assume cotransduction.

To determine whether sbcB lies to the right or left of his, we resorted to a series of transductional crosses using the supD marker already mapped on the right side of his (Hoffman and Wilhelm 1970). supD suppresses certain amber mutations, among which are a trp amber mutation and a leu amber mutation present in a Su-SmR strain JC4693. The leu amber mutation in JC4693 was detectably suppressed by supD- only in the presence of streptomycin but this did not prevent our selection for supD- by selecting for Trp+Leu+ transductants. JC4693 was recB+ sbcB+ however so we first constructed a recB+sbcB- derivative in three steps: (1) selection of a thyA- spontaneous mutant by the trimethoprim method (Stacey and Simson 1965), (2) cotransduction of sbcB15 with his+ from a his+sbcB15 donor, JC7644, made by transduction of his+ into our primary sbcB15 mutant strain, and (3) cotransduction of recB2I with thyA+. The resulting strain, JC7722, had all the desired properties for use in our mapping experiments except that it was his+. Consequently a his- mutant was obtained by the use of DES and tests were made to ensure that the his mutation derived was not suppressible by supD- and that the strain retained both sbcBI5 and recB2I.

The resulting strain, JC7729, was used as a recipient in a transductional cross with the his+sbcB+supD- strain KL96b as a donor. his+ inheritance was detected by the His+ phenotype, supD- inheritance by the Trp+Leu+ phenotype and sbcB+ inheritance by the Rec- phenotype. When His+ transductants were selected 35% inherited sbcB+ and 10% inherited supD- indicating that sbcB is closer to his than supD is to his but not that sbcB and supD are on the same side of his. When Trp+Leu+ (i.e. supD-) transductants were selected an average of 5.5% (102/1869) inherited sbcB+ and 3.9% (74/1869) inherited his+ indicating that sbcB is closer to supD than his is to supD and the order is his-sbcB-supD.

To measure more closely the location of sbcB between his and supD we used
TABLE 3

Cotransduction frequencies obtained in four-point transductional crosses

<table>
<thead>
<tr>
<th>Selected marker*</th>
<th>Selected phenotype</th>
<th>his+</th>
<th>Unselected donor markers</th>
<th>Percent sbcB+ among (P2)+ recombinants</th>
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<td>his+</td>
<td>His+</td>
<td>—</td>
<td>51% 48% 2%</td>
<td>81%</td>
</tr>
<tr>
<td>supD−</td>
<td>Trp+ Leu+</td>
<td>2%</td>
<td>6% 26% —</td>
<td>16%</td>
</tr>
</tbody>
</table>

* P1 was grown on JC7756 which is his+ sbcB+ (P2)+ supD+. JC7729 was the recipient and was his− sbcB− (P2)− supD+. In addition JC7729 carried recB21 and an amber mutation in both trpE and leu. Forty-nine transductants of each genotype were tested.

one of the P2 attachment sites (P2 attH) which lies between his and supD. A P2 lysogen of KL96b was isolated and the location of the prophage near his was verified. This strain JC7756 was used as a transductional donor with JC7729, the same recipient we used above. Both His+ and Trp+Leu+ transductants were selected. Forty-nine of each were streaked and single colonies were tested for unselected characters. Table 3 shows that the results are in agreement with the order his−sbcB−(P2)−supD. The four-point cross when analyzed as a series of two-point crosses shows sbcB+ more closely linked to his than the P2 prophage and less closely linked to supD than the P2 prophage. When analyzed as three-point crosses then 81% of his+ (P2)+ transductants inherit sbcB+ while only 16% of (P2)+ supD− transductants inherit sbcB+. Thus it is clear that sbcB lies between his and the P2 attachment site (Figure 1).

Deletion of sbcB: The location of sbcB has been confirmed by making use of the phenomenon of P2 eduction. SUNSHINE and KELLY (1971) have found that strains of E. coli K12 lysogenic for P2 at P2 attH spontaneously give rise to a small fraction of non-lysogenic, histidine-requiring cells. The deleted portion of the chromosome always starts with P2 prophage on one end while the other end is located beyond the gnd locus. The process is termed eduction and the resulting deletion-containing strains are called eductants. If sbcB+ does lie between the P2 attachment site and his as indicated by our transductional studies then eductants should have lost the sbcB+ gene.

To test this prediction we made P2 eductants from JC7735 which has the genotype his+sbcB+recB21 supD−leu− amber trp− amber and the phenotype His+ Rec−MitRLeu+Trp+. Advantage was taken of the fact that one does not need to start with P2 lysogens to obtain eductants but can isolate them from the bacterial growth in the center of P2 plaques. P2 old− was used since the old+ product kills recB− or recC− strains (LINDAHLL et al. 1970).

The results show that 14 of 4500 colonies tested were His− eductants; all 14 were MitR as expected if the sbcB+ gene had also been deleted. At the same time MitR (i.e. sbcB−) eductants were selected directly by plating on complex media containing mitomycin C. Of 392 MitR colonies picked all were His−MitRUVTrp+Leu+. Thus P2 eductants have lost his and sbcB but not supD.

Enzyme assays for exonuclease I were performed on two eductants, JC7749 selected as His− and JC7750 selected as MitR and both were found to be exonuclease I deficient. These two strains were also tested for their recombination
ability in crosses with the Hfr JC158. Both produced as many Lac+ [Ser+SmR] 
recombinants as did their rec+ sbcB+ ancestor, JC4693, and its recB21 sbcB15 
derivative, JC7729.

Effect of episome carrying sbcB: Because sbcB is so closely linked to his it 
seemed likely that the sbcB gene might be present on Fhis plasmids. F30 carries 
hisA323 which complements the his-4 mutation in the AB1157 series of strains. 
F30 was transferred into the rec+ and rec- strains and into a series of suppressed 
rec- strains by crossing with an appropriate F30 donor, JC2429. The presence of 
another sbc+ gene does not affect the phenotype of either the recB+recC+sbcB+ 
or recB-recC-sbcB+ strains. However, recB-recC-sbcB- strains are converted to a 
UVsMitSRec- phenotype indicating both that sbcB+ is present on the episome and 
that it is dominant. sbcB9, sbcB11, sbcB12 and sbcB15 were all found to be 
recessive by this method.

Nonspecificity of suppression: Since sbcB mutations have been obtained only in 
strains which are recB21 recC+ or recB21 recC22 it is possible that these combi-
nations of rec alleles are the only ones which can be suppressed by sbcB-. This 
possibility was tested by constructing strains which contain other rec alleles and 
an sbcB- gene. P1 lysates on strains which were recB58 or recB60 were used to 
transduce a thyA+his-sbcB15 strain which also contained the F30 episome. The 
resulting Thy+ transductants which had received recB- were phenotypically 
UVsMitSRec- because of the presence of the dominant sbcB+ gene on the episome. 
When the episome was eliminated by use of acridine orange, the phenotypes 
became UVsMitRRec+ showing that recB58 and recB60 are suppressed by sbcB15. 
Transfer of F30 back into the cured recB- sbcB15 strains converts them to the 
UVsMitSRec- phenotype. In the same fashion a recB+recC22 sbcB15 strain was 
constructed. Its phenotype is UVsMitSRec+ indicating that recC22 is suppressed 
by sbcB15.

Occurrence of suppressor mutations: Mutations causing indirect suppression of 
the recB and recC genes have been obtained in strains of at least four different 
genetic backgrounds. We can characterize the strains and their backgrounds as 
follows: (1) Endo I- to stand for a line carrying an endA mutation and conse-
quently endonuclease I deficient (DURWALD and HOFFMANN-BERLING 1968), 
(2) Su- to stand for a line carrying amber leu and trp mutations in the absence 
of amber-suppressing alleles, (3) Hfr to stand for a line derived from the Hfr 
JC5029, and (4) 1157 to stand for a line derived from the multiply auxotrophic 
F- AB1157. Table 4 summarizes the data concerning the occurrence of back-
mutations and suppressor mutations among Mit+ revertants selected from these 
strains with or without exposure to a mutagen. Single recB21 or recC22 deriva-
tives of the Endo I genetic background were previously shown to revert to Mit+ 
by indirect suppression (BARBOUR et al. 1970), and here we indicate that the same 
is true for recB-recC+ derivatives in the Su- and Hfr background. By contrast 
single recB21 or recC22 derivatives with the 1157 background revert chiefly by 
backmutation. When Mit+ revertants of a recB21 recC22 derivative with the 1157 
background are selected only suppressed revertants occur and all of these carry 
an sbcB mutation which cotransduces with his and cause exonuclease I deficiency
TABLE 4
Occurrence of suppressor mutations and backmutations in MitR revertants of recB21 and recC22 derivatives of four different genetic backgrounds

<table>
<thead>
<tr>
<th>Genetic background</th>
<th>rec allele</th>
<th>Number of revertants tested</th>
<th>Genotype of MitR revertants*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>recB</td>
<td>recC</td>
<td></td>
</tr>
<tr>
<td>Endo 1-</td>
<td>-+</td>
<td>2+</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>+</td>
<td>-+</td>
<td>2+</td>
</tr>
<tr>
<td>Su-</td>
<td>-+</td>
<td>2+</td>
<td>0</td>
</tr>
<tr>
<td>Hfr</td>
<td>-+</td>
<td>6+</td>
<td>1</td>
</tr>
<tr>
<td>1157</td>
<td>-+</td>
<td>1+</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>-+</td>
<td>7+</td>
</tr>
<tr>
<td></td>
<td>-+</td>
<td>3+</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>+</td>
<td>-+</td>
<td>3+</td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>-+</td>
<td>7+</td>
</tr>
</tbody>
</table>

* The presence of recB, recC, sbcA and sbcB mutations was tested for genetically and enzymatically.
† One of each pair of revertants was induced by EMS; the other, by NTG.
‡ All of these mutants were induced by EMS.
§ Spontaneous mutant.
¶ One revertant was spontaneous; the others were induced by EMS.
• One revertant was spontaneous; one was induced by EMS and one by NTG.

(KUSHNER et al. 1971). By contrast all indirectly suppressed MitR revertants isolated from strains with the Endo I-, Su- or Hfr backgrounds carry an sbcA mutation which does not cotransduce with his and results in high levels of ATP-independent nuclease activity. Thus there is a clear difference between the 1157 strains and the strains with the three other genetic backgrounds both in the type of suppressor which occurs and in the frequency of suppressor mutations as opposed to backmutations.

DISCUSSIONS

Three- and four-point transductional crosses place the sbcB gene between his and the E. coli K12 P2 attH. The distance from his to sbcB can be calculated by means of a formula relating cotransductional frequency and distance as follows: c.f. = (1 - d/l)^3 where c.f. is the cotransductional frequency expressed as a fraction, l is the length of the transducing fragment expressed in whatever units are convenient and d is the distance between selected and unselected marker (WU 1966). We assume that l is 6.5 x 10^7 daltons (the size of the P1 chromosome) or 2.3 min of distance as the E. coli map is generally related to its chromosome size of 2.5 x 10^9 daltons. Then by averaging the cotransduction frequencies of sbcB to his shown in Table 2 (0.56) we calculate that the distance between these two markers is about 0.4 min.

This location has been confirmed by the isolation of eductants in which the chromosomal region from P2 attH through gnd is deleted. This deletion invariably takes out both the his operon and sbcB thus making eductants both His- and Exo I-. The deletions also invariably suppress the UV8Mit8 phenotypes conferred by recB21. Two of the eductants were tested and found to be Rec+ indicating
suppression of the Rec\(^{-}\) phenotype as well. This result confirms the hypothesis of Kushner et al. (1971) that the absence of exonuclease I rather than the presence of a mutationally altered enzyme is responsible for the suppression of the recB mutation. Recently we reported the existence of xonA mutations inactivating exonuclease I and lying very close to sbcB (Kushner, Nagaishi and Clark 1972). The phenotype of recB\(^{+}\)recC\(^{+}\)xonA\(^{-}\) strains is UV\(^{+}\)Mit\(^{+}\)Rec\(^{-}\) indicating that xonA mutations suppress the UV\(^{+}\) and Mit\(^{+}\) of recB\(^{+}\)recC\(^{-}\) but not their Rec\(^{-}\) property. By contrast sbcB mutations suppress all three phenotypes. Since theeductants tested were Rec\(^{+}\) as well as Mit\(^{+}\)UV\(^{+}\) they must genetically be sbcB\(^{-}\)xonA\(^{-}\) or sbcB\(^{-}\)xonA\(^{+}\). At present we can not determine which.

Considerable evidence has been adduced to demonstrate indirect suppression of recB and recC mutations by both sbcA and sbcB mutations (Barbour et al. 1970; Kushner et al. 1971; Templin and Clark, unpublished results). Intragenic suppression is ruled out by the occurrence of both types of suppressors in recB\(^{+}\)recC\(^{-}\)double mutants and by the lack of cotransduction between the original mutations and their suppressors. Informational suppression is ruled out by the lack of detectable suppression of UAG, UAA or UGA mutant phages. Furthermore exonuclease V activity determined by recB and recC is not restored in either sbcA or sbcB mutants, a fact incompatible with either intragenic or informational suppression. In this paper we show that one recC and three recB mutations are suppressed by sbcB15 demonstrating the nonallele-specific behavior of the indirect suppression.

sbcA and sbcB mutations appear to suppress recB and recC mutations by different means. sbcA\(^{-}\) strains show higher levels of an ATP-independent nuclease than shown by sbcA\(^{+}\) strains (Barbour et al. 1970); in addition sbcA\(^{-}\) strains contain exonuclease I (Kushner et al. 1971). sbcB\(^{-}\) strains do not show higher levels of ATP-independent nuclease activity than sbcB\(^{+}\) strains; in addition sbcB\(^{-}\) strains do not contain exonuclease I (Kushner et al. 1971). This difference led us to expect that the suppressed mutants isolated from any given strain would fall into both sbcA and sbcB groups. Instead when we have looked for spontaneously occurring or mutagen-induced revertants, we have found no sbcA mutants from strains of the 1157 genetic background and no sbcB mutants from strains of Endo I\(^{-}\), Su\(^{-}\), and Hfr background. One possibility to explain this is that sbcB mutations would be unexpressed in the three genetic backgrounds in which they do not appear to occur as primary mutants. We have, however, shown previously (Kushner et al. 1971) that sbcB15 transduced to an Endo I\(^{-}\), a Su\(^{-}\) and an Hfr strain confers exonuclease I deficiency on these three strains. Another possibility is that the absence of exonuclease I in these three genetic backgrounds would not indirectly suppress recB or recC mutations. In this paper we have shown however that sbcB15 when transduced into the Su\(^{-}\) background can suppress recB21 and we have used this suppression in order to map the sbcB mutation relative to the his operon and P2 attH. Furthermore we have shown that sbcB deletion mutations which suppress recB21 occur in the Su\(^{-}\) background by a process known as P2 eduction.

It is still possible that the Endo I\(^{-}\), Su\(^{-}\), and Hfr strains we have used all carry
a tandem duplication of \textit{sbcB} which is lost by transduction from the 1157 strains which do not carry this duplication. This, however, does not explain why \textit{sbcA} mutations apparently fail to occur in the 1157 background. Since the \textit{sbcA} mutation may be looked upon as a regulatory mutation derepressing a structural gene, \textit{recE}, which determines an ATP-independent nuclease activity (CLARK 1971a,b), the lack of occurrence of \textit{sbcA} mutations in the 1157 line of strains may therefore indicate that these strains are \textit{recE}.

In fact there is a highly speculative line of reasoning which indicates that the 1157 series of strains may be \textit{sbcA} as well as \textit{recE}. This speculation depends upon the behavior of the gene \textit{rac} which is located near \textit{trp} (TAYLOR 1970) and was discovered by Low (personal communication). \textit{rac} when transferred to zygotcs of \textit{recB} or \textit{recC} derivatives of the 1157 line suppressed the recombination deficiency of these zygotes and permitted nearly wild-type frequencies of recombinants for distal markers to be produced. When \textit{rac} is transferred to zygotcs of \textit{recB} or \textit{recC} mutants of other genetic backgrounds the activation of recombination is not observed (Low, personal communication). This suggests that the \textit{rac} gene can be zygotically derepressed in the 1157 line only, and not in other lines of strains. These other lines may be \textit{sbcA} like the Endo I-, Su-, and Hfr lines we have used. If we hypothesize that \textit{rac} and \textit{recE} are two symbols for the same gene, then we expect that \textit{rac} would be repressed by \textit{sbcA}. Since only the 1157 lines allows zygotic derepression of \textit{rac} we would then expect the 1157 line to be \textit{sbcA}.

Thus we feel that the 1157 line of strains may be both \textit{sbcA} and \textit{recE} (i.e. \textit{rac}) while other strains may be \textit{sbcA}+\textit{recE}+. If true this would fit quite well with Low's hypothesis (personal communication) that \textit{rac} occurs on an active or cryptic prophage. Presumably the regulator gene for \textit{rac} would also occur on the same prophage; hence we can explain the genotype of 1157 strains by hypothesizing that they lack this crucial prophage. Since lambdoid phages carry a \textit{redX} gene which determines an ATP-independent exonuclease involved in phage recombination (SHULMAN \textit{et al.} 1970) it is therefore attractive to suspect that the \textit{recE} (i.e. \textit{rac}) nuclease presumably derepressed by \textit{sbcA} mutations will be found to possess substrate specificities and activities similar to those of the lambda exonuclease. This is even more attractive when it is recognized that the lambda exonuclease will catalyze bacterial recombination (Low, personal communication) and that the \textit{recE} enzyme will apparently catalyze lambda recombination (UNGER, ECHOLS and CLARK 1972).

We are very grateful to \textsc{Meiz Sunshine} for his advice and close attention to the P2 eduction experiments as well as for many discussions on all aspects of this work. We also are grateful to \textsc{Haruko Nagaishi} for her help in performing some of the enzyme assays.

\textbf{LITERATURE CITED}


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