Recombinational DNA repair: the ignored repair systems

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Summary
The recent finding of a role for the *recA* gene in DNA replication restart does not negate previous data showing the existence of *recA*-dependent recombinational DNA repair, which occurs when there are two DNA duplexes present, as in the case for *recA*-dependent excision repair, for postreplication repair (i.e., the repair of DNA daughter-strand gaps), and for the repair of DNA double-strand breaks. Recombinational DNA repair is critical for the survival of damaged cells. *BioEssays* 26:1322–1326, 2004. © 2004 Wiley Periodicals, Inc.

Introduction

Currently there is much interest in the repair of damaged DNA replication forks, because of recent observations that some of the bacterial genes that participate in homologous recombination may also be involved in the reinitiation of DNA synthesis after ultraviolet (UV) irradiation (e.g. 1,2). Finding a new function for an old protein (i.e. RecA) is exciting, however, it does not negate the original observations that RecA functions in recombinational repair processes that require two DNA duplexes to complete DNA repair, and where strand exchanges occur (see below).

Such statements as "It has recently become clear that the recombinational repair of stalled replication forks is the primary function of homologous recombination systems in bacteria" (1), totally ignore the problems that a cell faces when its DNA that was replicated prior to UV irradiation is damaged, where two DNA duplexes exist, and where replication restart has little or no relevance (Fig. 1).

A cell has many different DNA-repair systems, but the sheer volume of publications on "cut and patch" nucleotide excision repair (e.g. 3) has seemingly generated the impression that cells possess only this repair system. Furthermore, an essay has been published denying the existence of recombination repair, but the authors were very selective with their literature citations.[4]

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There are many incorrect statements in the literature about recombinational repair. For example, "We now know that several of the processes that interact with or are controlled by *recA*, such as excision repair and translesion synthesis, operate to ensure that DNA replication occurs processively without strand exchanges."[4] Yet, a publication from the same laboratory,[5] and by others,[6–8] have all demonstrated that strand exchanges occur after UV irradiation.

"From a practical point of view, these results demonstrate that, in the absence of nucleotide excision repair, *recA* function does not contribute significantly to cellular viability."[4] This statement is inconsistent with the data of Howard-Flanders and Boyce,[9] which these authors even reproduce in their paper.[4] If the statement by these authors[4] were true, one would expect that a mutation blocking *recA* function would have no effect on the survival of UV-irradiated *uvr*-deficient cells. However, the data of Howard-Flanders and Boyce[9] show that the additional presence of a *recA* mutation has a very significant sensitizing effect on the survival of UV-irradiated *uvr*-deficient cells. In view of the results of Howard-Flanders and Boyce,[9] the statement that "...the ability of recA to promote recombination is virtually useless for cellular survival..."[10] is without merit.

Recombination repair requires many more gene products than does excision repair; it also requires two DNA duplexes, not just one, and there are multiple pathways of recombination repair (see below). The sheer complexity of recombination repair has apparently resulted in it being largely ignored by the general scientific community. However, recombination repair[11] is an important set of repair systems that should not be ignored.

Multiple pathways of DNA repair

The first indication that nucleotide excision repair ("cut and patch") is NOT the only mechanism by which cells repair damage to their DNA, was the observation that bacterial cells deficient in nucleotide excision repair (i.e. *uvrA*) or in genetic recombination (i.e. *recA*) are very sensitive to UV radiation, and show a similar level of survival after UV irradiation. A double mutant (*uvrA recA*) is very much more sensitive to UV irradiation.[9] From the most fundamental principles of radiation biology and genetics, these data argue that, (a) these two systems, i.e. coded by the *uvrA* and the *recA* genes, function largely independently of each other, and (b) they are of about

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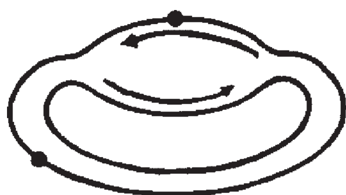

Figure 1. Schematic of DNA replication with lesions (●) both in the DNA that was replicated prior to UV irradiation, where two DNA duplexes exist, and in that portion of the chromosome prior to replication, where only one DNA duplex exists. The problems and opportunity for recombinational DNA repair and replication restart in these two regions of the chromosome are markedly different.

equal importance to the survival of UV-irradiated cells of *E. coli* K-12.

Second, although ignored in most reviews on DNA repair, there is a pathway of nucleotide excision repair that is dependent upon recombination (see below). This occurs when lesions are produced in the portion of the chromosome that was replicated PRIOR to UV irradiation, and therefore, two DNA duplexes are present in the region of the lesion (Fig. 1).

Third, statements such as, "...replication also fails to recover in *uvr* mutants...",[12] ignore the fact that photoproducts such as pyrimidine dimers do NOT permanently stop DNA synthesis in cells that are deficient in nucleotide excision repair.[13,14] Therefore, cells must have repair system(s) in addition to excision repair, otherwise excision-repair-deficient cells would not survive UV irradiation. One such system is postreplication repair, and it requires homologous recombination.

Postreplication repair

The DNA synthesized immediately after UV irradiation in excision-repair-deficient cells (and also wild-type cells; see below) of *E. coli* K-12 has discontinuities when assayed in alkaline sucrose gradients. The mean length of newly synthesized DNA approximates the distance between pyrimidine dimers in the parental strand. With further incubation of the cells, however, these discontinuities disappear, and the DNA approximates the molecular size of that from unirradiated control cells.[13,15] The exchanges envisioned by this type of repair resemble those involved in genetic recombination.[6,13] This prediction has been verified by demonstrating that *recA* cells are deficient in the production of normal-length DNA from the small pieces synthesized immediately after UV irradiation.[16,17]

When DNA synthesis proceeds along a damaged template, synthesis halts at the site of a non-coding lesion, and then resumes downstream from the lesion, leaving gaps in the newly synthesized daughter strand opposite the UV radiation-induced lesion in the parental strand.[13] The fact that photoreactivation after UV irradiation in a *uvrA* strain stimulated gap filling is taken as further evidence that a large proportion of the DNA daughter-strand gaps are opposite pyrimidine dimers.[18]

The dimers that are opposite DNA daughter-strand gaps are no longer subject to excision, since this process requires an intact complementary strand.[19,20] Only after the gaps are filled by sister-strand exchanges will the dimers again be subject to excision repair.

These gaps in the daughter strands, which average 1000 nucleotides in length,[21] are subsequently repaired in recombination-proficient strains by transferring the appropriate sections of DNA from the parental strands into the daughter strands. This transfer of parental strands into daughter strands has been confirmed by direct measurement.[5–8] Although most studies on postreplication repair have been performed in excision-repair-deficient cells, this type of repair is fully operative in wild-type cells.[16,22,23]

Although postreplication repair (i.e. the repair of DNA daughter-strand gaps) is completely dependent upon the *recA* gene, mutations in the *recB* and *recC* genes do NOT cause a deficiency in the repair of DNA daughter-strand gaps.[16] However, the *recB* gene is known to function in the repair of DNA double-strand breaks that are formed metabolically after UV irradiation in *E. coli*.[24] In fact, unrepaired DNA double-strand breaks appear to be the major cause of lethality in UV-irradiated wild-type bacteria.[25,26] The repair of metabolically produced DNA double-strand breaks constitutes a second type of recombination repair that is distinct from the repair of DNA daughter-strand gaps, i.e. it is *recBC*-dependent.[24,27]

Multiple pathways of postreplication repair

Three pathways are known for the repair of DNA daughter-strand gaps, i.e. the *recF*-dependent, the *recF*-independent and the *umuCD*-dependent pathways. Much of postreplication repair is constitutive,[17,28] but a portion (i.e. *umuCD*) is inducible by UV radiation and is responsible for UV radiation mutagenesis (see below). Each of these pathways is *recBC*-independent.[16]

RecF pathway

About half of the DNA daughter-strand gaps are repaired by a *recF*-dependent process.[24,29–31] The involvement of the *recF* gene suggests that the *recF* pathway of homologous recombination may be involved in this repair process. The RecF protein is one of at least three single-strand DNA-binding proteins, along with the RecA and Ssb proteins.[32]

The repair of daughter-strand gaps by the *recF*-dependent and the *recF*-independent process (see below) is accompanied by the transfer of DNA lesions from the parental strand to the daughter strand.[5,8] This occurs about 50% of the time in *E. coli*,[5] and appears to be due to the random resolution of the Holliday junction (e.g. 33), an intermediate in recombination.

RecF-independent pathway

The fact that a *uvrB recF* stain is not as deficient in the repair of daughter-strand gaps as is a *uvrB recA* strain suggested that a second pathway must exist for the repair of daughter-strand gaps.[24] This conclusion was supported by studies using an insertion mutation of *recF* (*recF332*:Tn3) to ensure that the earlier results were not due to leakiness in the original *recF143* mutation. The *recF*-independent pathway is also independent of the *recBC* genes and is constitutive.[34] Studies using Δ*polA* mutants indicate that the *polA* gene (DNA polymerase I) plays a major role in the *recF*-independent repair of daughter-strand gaps. Studies on different *polA* mutants (i.e. *polA1, polAex2*, Δ*polA*, etc.) suggest that it is the $5' \rightarrow 3'$ exonuclease activity of DNA polymerase I that plays a major role in the repair of daughter-strand gaps.[35]

Furthermore, since DNA polymerase is known to be involved in the joining of Okazaki fragments synthesized in the lagging strand of unirradiated cells, this raises the possibility that the daughter-strand gaps formed in the lagging strand of UV-irradiated cells may be selectively repaired by the *recF*-independent, *polA*-dependent pathway, while the daughter-strand gaps formed in the leading strand (i.e. presumably longer gaps) may be repaired by the *recF*-dependent pathway.[36]

UmuC pathway

Since a *uvrA* Δ*polA recF* strain is not quite as deficient in the repair of daughter-strand gaps as is a *uvrA recA* strain,[35] it suggests that a third pathway must exist for the repair of daughter-strand gaps. Consistent with this observation, a small fraction of the repair of daughter-strand gaps is dependent upon the *umuC* gene, but is independent of the *recF* and *recBC* genes.[37] A *uvrA* Δ*polA recF umuC* strain has not yet been tested to see if it is as deficient as a *uvrA recA* strain in the repair of daughter-strand gaps.

The UmuC and UmuD proteins combine, after the selective cleavage of the UmuD protein by RecA, to form an error-prone polymerase ($UmuD'_2UmuC$), polV[38,39] which can synthesize past lesions in DNA. This is consistent with the fact that umuC controls all of UV radiation mutagenesis.[40] A *umuC* mutation, however, has only a partial effect on spontaneous mutagenesis,[41] and on X-ray mutagenesis.[42]

Some authors (e.g. 43) think only in terms of polV as assisting replication restart by synthesizing past a pyrimidine dimer. An additional function for polV may be the repair of rare lesions such as overlapping daughter-strand gaps, perhaps facilitating translesion DNA synthesis to repair one of the daughter-strand gaps, after which the other daughter-strand gap could be repaired by the recombination pathways described above.

It is interesting to note that UV radiation mutagenesis is largely a two-hit process, i.e. two lesions are required.[44,45] Replication restart using polV to bypass a pyrimidine dimer could account for one-hit mutagenesis, but the repair of overlapping DNA daughter-strand gaps would seem a more probable explanation for two-hit mutagenesis.

Nucleotide excision repair

There are two pathways of nucleotide excision repair. One pathway is DNA polymerase I dependent, growth medium independent (i.e., macromolecular synthesis is not required), and produces short repair patches (about 20 nucleotides long). This pathway requires only one DNA duplex.[3]

The second excision-repair process, long-patch excision repair, which requires two DNA duplexes, is largely ignored by reviewers (e.g. 46). Nevertheless, this excision-repair pathway does exist, and it has been confirmed by other authors (e.g. 47). It is dependent upon the recA gene, it is growth medium dependent (i.e. macromolecular synthesis is required) and it produces long repair patches (1500–9000 nucleotides long).[48–50] Long-patch excision repair also requires the *recF* gene,[51] but does NOT require the *recBC* genes.[52]

When wild-type cells are allowed to repair their DNA after UV irradiation in the presence of chloramphenicol to inhibit the synthesis of induced proteins, only about 80% of the dimers are excised.[53] Similarly, a *recA* mutant, which is deficient in the induction of proteins after UV irradiation, only excises about 80% of the dimers compared to a wild-type strain.[54] The early repair seems to be short-patch excision repair, which occurs immediately after UV irradiation and is controlled by DNA polymerase I,[48] while the induced repair appears to be the long-patch system that is controlled by *recA*.[50] Additional copies of the UvrA protein[55] and the UvrB protein[56] are synthesized after UV irradiation, and may be relevant to the inducible long-patch excision-repair process.

The excision repair that occurs in cells that contain completely replicated chromosomes, i.e. where only one DNA duplex is present per chromosome, is not dependent upon *recA*. In this situation, classical nucleotide excision repair occurs, i.e. without strand exchanges. The excision repair that functions in the part of the chromosome that was replicated before UV irradiation (i.e. where two DNA duplexes exist, Fig. 1), is *recA* dependent.[57]

The similarities between the genetic requirements for long-patch excision repair and the repair of DNA daughter-strand gaps, i.e. the requirement for *recA* and *recF*, but not *recBC*, and the requirement for sister DNA duplexes, suggests that the mechanisms for these two repair processes are similar, i.e. requiring strand exchanges. The only significant difference between these two processes is the manner in which the gaps in the sister duplexes are formed, i.e. by excision or by replication bypass.[57]

Summary and conclusions

It is unfortunate that the older DNA-repair literature, which clearly shows the importance of recombinational DNA repair,

is being ignored. Furthermore, most reviewers make no distinction between the repair events that take place in the two different parts of the chromosome, i.e. the part of the chromosome that was replicated before UV irradiation, where two DNA duplexes exist, and the part of the chromosome that contains only one DNA duplex, and is replicated after UV irradiation. Clearly the problems and the opportunities for recombination repair and replication restart are different in these two regions of the chromosome.

It is exciting to find a new use for an old protein, i.e. the involvement of the RecA protein in translesion synthesis for replication restart, but this does not mean that *recA*-dependent recombination repair of DNA damage no longer exists. As documented by data from a number of laboratories (see above), it does exist and includes the *recA*-dependent branch of excision repair, the *recA*-dependent repair of DNA daughter-strand gaps (i.e. postreplication repair), and the *recA recB*-dependent repair of DNA double-strand breaks.

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References
1. Cox MM. 2001. Recombinational DNA repair of damaged replication forks in *Escherichia coli*: questions. Annu Rev Genet 35:53–82.
2. Rangarajan S, Woodgate R, Goodman MF. 2002. Replication restart in UV-irradiated *Escherichia coli* involving pols II, III, V, PriA, RecA and RecFOR proteins. Molec Microbiol 42:617–628.
3. Petit C, Sancar A. 1999. Nucleotide excision repair: from *E. coli* to man. Biochimie 81:15–25.
4. Courcelle J, Ganesan AK, Hanawalt PC. 2001. Therefore, what are recombination proteins there for? BioEssays 23:463–470.
5. Ganesan AK. 1974. Persistence of pyrimidine dimers during post-replication repair in ultraviolet light-irradiated *Escherichia coli* K-12. J Mol Biol 87:103–119.
6. Rupp WD, Wilde CE III, Reno DL, Howard-Flanders P. 1971. Exchanges between DNA strands in ultraviolet-irradiated *Escherichia coli*. J Mol Biol 61:25–44.
7. Howard-Flanders P, Rupp WD. 1972. Recombination repair in UV-irradiated *Escherichia coli*. In: Beers RF Jr, Herriott RM, Tilghman RC, editors. Molecular and Cellular Repair Processes. Baltimore, MD: Johns Hopkins Medical Journal, Suppl 1, pp 212–225.
8. Wang TV, Smith KC. 1984. *recF*-Dependent and *recF recB*-independent DNA gap-filling repair processes transfer dimer-containing parental strands to daughter strands in *Escherichia coli* K-12 uvrB. J Bacteriol 158:727–729.
9. Howard-Flanders P, Boyce RP. 1966. DNA repair and genetic recombination: studies on mutants of *Escherichia coli* defective in these processes. Radiat Res Suppl 6:156–184.
10. Courcelle J, Hanawalt PC. 2001. Participation of recombination proteins in rescue of arrested replication forks in UV-irradiated *Escherichia coli* need not involve recombination. Proc Natl Acad Sci USA 98:8196–8202.
11. Kuzminov A. 1999. Recombinational repair of DNA damage in *Escherichia coli* and bacteriophage λ. Microbiol Mol Biol Rev 63:751–813.
12. Courcelle J, Crowley DJ, Hanawalt PC. 1999. Recovery of DNA replication in UV-Irradiated *Escherichia coli* requires both excision repair and RecF protein function. J Bacteriol 181:916–922.
13. Rupp WD, Howard-Flanders P. 1968. Discontinuities in the DNA synthesized in an excision-defective strain of *Escherichia coli* following ultraviolet irradiation. J Mol Biol 31:291–304.
14. Smith KC. 1969. DNA synthesis in sensitive and resistant mutants of *Escherichia coli* B after ultraviolet irradiation. Mutat Res 8:481–495.
15. Howard-Flanders P, Rupp WD, Wilkins BM, Cole RS. 1968. DNA replication and recombination after UV irradiation. Cold Spring Harbor Symp Quant Biol 33:195–207.
16. Smith KC, Meun DHC. 1970. Repair of radiation-induced damage in *Escherichia coli*. I. Effect of *rec* mutations on post-replication repair of damage due to ultraviolet radiation. J Mol Biol 51:459–472.
17. Sedgwick SG. 1975. Genetic and kinetic evidence for different types of postreplication repair in *Escherichia coli* B. J Bacteriol 123:154–161.
18. Bridges BA, Sedgwick SG. 1974. Effect of photoreactivation on the filling of gaps in deoxyribonucleic acid synthesized after exposure of *Escherichia coli* to ultraviolet light. J Bacteriol 117:1077–1081.
19. Jansz HS, Pouwels PH, Van Rotterdam C. 1963. Sensitivity to ultraviolet light of single- and double-stranded DNA. Biochim Biophys Acta 76: 655–657.
20. Yarus M, Sinsheimer RL. 1964. The U.V.-resistance of double-stranded ΦX174 DNA. J Mol Biol 8:614–615.
21. Iyer VN, Rupp WD. 1971. Usefulness of benzoylated naphthoylated DEAE-cellulose to distinguish and fractionate double-stranded DNA bearing different extents of single-stranded regions. Biochim Biophys Acta 228:117–126.
22. Rupp WD, Iyer VN, Zipser E. 1973. The reconstitution of chromosomal DNA in irradiated cells by post-replication recombinational repair. In: Duplan JF, Chapiro A, editors. Advances in Radiation Research: Physics and Chemistry (Proceedings of the 4th Congress of Radiation Research, Evian-les-Bains, France, 1970) New York: Gordon and Breach; pp 39–50.
23. Howard-Flanders P, Rupp WD. 1981. Measurement of postreplication repair in prokaryotes. In: Friedberg EC, Hanawalt PC, editors. DNA Repair: A Laboratory Manual of Research Procedures, Vol. 1, Part B New York: Marcel Dekker; pp 459–470.
24. Wang TV, Smith KC. 1983. Mechanisms for *recF*-dependent and *recB*-dependent pathways of postreplication repair in UV-irradiated *Escherichia coli uvrB*. J Bacteriol 156:1093–1098.
25. Bonura T, Smith KC. 1975. Enzymatic production of deoxyribonucleic acid double-strand breaks after ultraviolet irradiation of *Escherichia coli* K-12. J Bacteriol 121:511–517.
26. Bonura T, Smith KC. 1975. Quantitative evidence for enzymatically-induced DNA double-strand breaks as lethal lesions in UV-irradiated *pol*$^{+}$ and *polA1* strains of *E. coli* K-12. Photochem Photobiol 22:243–248.
27. Wang TV, Smith KC. 1986. Postreplicational formation and repair of DNA double-strand breaks in UV-irradiated *Escherichia coli uvrB* cells. Mutat Res 165:39–44.
28. Ganesan AK, Smith KC. 1972. Requirement for protein synthesis in *rec*-dependent repair of deoxyribonucleic acid in *Escherichia coli* after ultraviolet or X irradiation. J Bacteriol 111:575–585.
29. Ganesan AK, Seawell PC. 1975. The effect of *lexA* and *recF* mutations on post-replication repair and DNA synthesis in *Escherichia coli* K-12. Mol Gen Genet 141:189–205.
30. Kato T. 1977. Effects of chloramphenicol and caffeine on postreplication repair in *uvrAumuC* and *uvrArecF* strains of *Escherichia coli* K-12. Mol Gen Genet 156:115–120.
31. Tseng YC, Hung JL, Wang TC. 1994. Involvement of RecF pathway recombination genes in postrepliction repair in UV-irradiated *Escherichia coli* cells. Mutat Res 315:1–9.
32. Madiraju MV, Clark AJ. 1991. Effect of RecF protein on reactions catalyzed by RecA protein. Nucleic Acids Res 19:6295–6300.
33. Sigal N, Alberts B. 1972. Genetic recombination: The nature of a crossed strand-exchange between two homologous DNA molecules. J Mol Biol 71:789–793.
34. Sharma RC, Smith KC. 1985. A minor pathway of postreplication repair in *Escherichia coli* is independent of the *recB*, *recC* and *recF* genes. Mutat Res 146:169–176.
35. Sharma RC, Smith KC. 1987. Role of DNA polymerase I in postreplication repair: A reexamination with *Escherichia coli* Δ*polA*. J Bacteriol 169: 4559–4564.
36. Liu H, Cheng A, Wang TV. 1998. Involvement of *recF*, *recO*, and *recR* genes in UV-radiation mutagenesis of *Escherichia coli*. J Bacteriol 180: 1766–1770.

37. Wang TV, Smith KC. 1985. Role of the *umuC* gene in postreplication repair in UV-irradiated *Escherichia coli* K-12 *uvrB*. Mutat Res 145:107–112.
38. Tang M, Shen X, Frank EG, O'Donnell M, Woodgate R. 1999. UmuD′$_2$C is an error-prone DNA polymerase, *Escherichia coli* pol V. Proc Natl Acad Sci USA 96:8919–8924.
39. Ferentz AE, Walker GC, Wagner G. 2001. Converting a DNA damage checkpoint effector (UmuD$_2$C) into a lesion bypass polymerase (UmuD′$_2$C). The EMBO J 20:4287–4298.
40. Kato T, Shinoura Y. 1977. Isolation and characterization of mutants of *Escherichia coli* deficient in induction of mutations by ultraviolet light. Mol Gen Genet 14:121–131.
41. Sargentini NJ, Smith KC. 1981. Much of spontaneous mutagenesis in *Escherichia coli* is due to error-prone DNA repair: Implications for spontaneous carcinogenesis. Carcinogenesis 2:863–872.
42. Sargentini NJ, Smith KC. 1989. Mutational spectrum analysis of *umuC*-independent and *umuC*-dependent gamma-radiation mutagenesis in *Escherichia coli*. Mutat Res 211:193–203.
43. Pham P, Rangarajan S, Woodgate R, Goodman MF. 2001. Roles of DNA polymerases V and II in SOS-induced error-prone and error-free repair in *Escherichia coli*. Proc Nat Acad Sci USA 98:8350–8353.
44. Doudney CO. 1976. Complexity of the ultraviolet mutation frequency response curve in *Escherichia coli* B/r: SOS induction, one-lesion and two-lesion mutagenesis. J Bacteriol 128:815–826.
45. Sargentini NJ, Smith KC. 1979. Multiple independent components of ultraviolet radiation mutagenesis in *Escherichia coli* K-12 *uvrB5*. J Bacteriol 140:436–444.
46. Hanawalt PC. 2001. Controlling the efficiency of excision repair. Mutat Res 485:3–13.
47. Youngs DA, van der Schueren E, Smith KC. 1974. Separate branches of the *uvr* gene-dependent excision repair process in ultraviolet-irradiated *Escherichia coli* K-12 cells; their dependence upon growth medium and the *polA*, *recA*, *recB*, and *exrA* genes. J Bacteriol 117:717–725.
48. Cooper PK, Hanawalt PC. 1972. Role of DNA polymerase I and the *rec* system in excision-repair in *Escherichia coli*. Proc Natl Acad Sci USA 69: 1156–1160.
49. Cooper PK, Hanawalt PC. 1972. Heterogeneity of patch size in repair replicated DNA in *Escherichia coli*. J Mol Biol 67:1–10.
50. Cooper PK. 1982. Characterization of long patch excision repair of DNA in ultraviolet-irradiated *Escherichia coli*: an inducible function under Rec-Lex control. Mol Gen Genet 185:189–197.
51. Hanawalt PC, Cooper PK, Ganesan AK, Lloyd RS, Smith CA, et al. 1982. Repair responses to DNA damage: enzymatic pathways in *E. coli* and human cells. J Cell Biochem 18:271–283.
52. Hanawalt PC, Cooper PK, Smith CA. 1981. Repair replication schemes in bacteria and human cells. Prog Nucleic Acid Res Mol Biol 26:181–196.
53. Lin CG, Kovalsky O, Grossman L. 1997. DNA damage-dependent recruitment of nucleotide excision repair and transcription proteins to *Escherichia coli* inner membranes. Nucleic Acids Res 25:3151–3158.
54. Shlaes DM, Anderson JA, Barbour SD. 1972. Excision repair properties of isogenic *rec*-mutants of *Escherichia coli* K-12. J Bacteriol 111:723–730.
55. Kenyon CJ, Walker GC. 1981. Expression of the *E. coli uvrA* gene is inducible. Nature 289:808–810.
56. Schendel PF, Fogliano M, Strausbaugh LD. 1982. Regulation of the *Escherichia coli* K-12 *uvrB* operon. J Bacteriol 150:676–685.
57. Smith KC, Sharma RC. 1987. A model for the *recA*-dependent repair of excision gaps in UV-irradiated *Escherichia coli*. Mutat Res 183:1–9.