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MINIREVIEW

Derepression of Specific Genes Promotes DNA Repair and Mutagenesis in *Escherichia coli*

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INTRODUCTION

Many of the DNA damage-inducible genes encoding DNA repair and recombination proteins are expressed as part of the SOS response (39, 40). LexA protein represses the genes of the SOS regulon (Table 1); when DNA damage occurs, RecA protein inactivates the LexA repressor, leading to induction of the SOS response. The SOS response controls the expression of genes involved in recombination repair (daughter-strand gap repair, double-strand break repair), excision repair (both short and long patch), mutagenesis (error-prone repair), and mismatch repair of DNA. This review will focus on recent data pertaining to DNA repair and mutagenesis associated with expression of the SOS response.

The presence of DNA damage may be lethal unless the repair and recombination potential of the cell is increased. Many lesions resulting from DNA-damaging treatments or stalled DNA replication require derepression of the SOS response for their repair or bypass. The damage sites themselves and/or the products of preinduction processing may serve as signals to activate RecA so that SOS functions may be derepressed (34). The various repair systems under control of the SOS regulon then act directly to repair the original lesions or allow the cell to tolerate the lesions until repair can be effected by other mechanisms.

REGULATION OF THE SOS RESPONSE

The SOS response is induced in *Escherichia coli* after treatments that cause DNA damage or stalled DNA replication (26, 39, 40). DNA damage is thought to lead to the production of an intracellular inducing signal(s) that presumably interacts with RecA protein. RecA protein is reversibly altered to an activated form (RecA*) that can mediate LexA repressor cleavage, thus derepressing the SOS regulon. The nature of the in vivo signal has not yet been established, but evidence gathered in vitro, as well as evidence from mutants with metabolic defects that cause increased expression of the SOS response, suggests that the signal is derived from nucleic acids.

A number of mutants have been isolated that exhibit constitutively induced or subinduced phenotypes (Table 2). All of the mutations found to date affect some aspect of DNA metabolism or replication. Mutations in *dam*, *dnaB*, *dnaE*, *dnaG*, *lig*, *polA*, *recF*, *ruv*, *ssb*, and *uvrD* all derepress the SOS regulon to various extents (29; N. Ossanna, K. R. Peterson, B. L. Fisher, and D. W. Mount, unpublished data). The variety of biochemical activities affected emphasizes the diversity of perturbations which generate the signal(s).

In some cases, increased SOS gene expression may compensate for the detrimental effects of the original mutation by providing the cell with a mode of repair or tolerance. Two classes of defects have been observed, those which produce mutants that remain viable when SOS expression is blocked and those which produce mutants that are inviable when SOS expression is blocked. In the case of *dam* mutants, the products of two SOS genes, *recA* and *ruv*, are required for viability (29). In contrast, *uvrD* mutants that constitutively express the SOS response are viable when expression of SOS genes is blocked by *recA*(Def) or *lexA*(Ind$^-$) mutations.

The signal(s) generated by metabolic defects is sensed by RecA protein. The best evidence for this function of RecA comes from the existence of mutants in which RecA protein is always activated for LexA repressor cleavage without the usual need for DNA damage. Alleles of this class, termed *recA* protease-constitutive (Prt^c), have been isolated and characterized (35, 36, 43; N. Ossanna and D. W. Mount, unpublished data). Further analysis of these mutants and isolation of mutations in other genes causing derepression of SOS genes should provide further clues as to the molecular nature of signals and their generation.

INDUCTION OF RECOMBINATION REPAIR

The RecF recombination pathway becomes more active in cells exposed to DNA-damaging agents (20, 21). Recombinational repair of DNA is probably accomplished by promoting exchanges that allow damage to be repaired or bypassed during replication of the chromosome. Mutations in the RecF pathway genes have little effect on recombination in otherwise wild-type cells, suggesting that the major role of these recombination proteins is in DNA repair.

Eight genes, including *recA*, *recF*, *recJ*, *recN*, *recO*, *recQ*, *ruv*, and *uvrD* (formerly *recL*), have been identified as members of the RecF pathway (12, 16, 17, 19, 22, 25; Table 1). *recN*, *recQ*, *ruv*, and *uvrD* are part of the SOS regulon; *recF* is not, and the status of the remaining genes is unknown (4, 14, 19, 33). The functions of most of the RecF pathway gene products in recombination and repair have not been elucidated. The direct role of RecA protein in strand exchange reactions has been well characterized and is necessary for the function of all recombination pathways. The *recN* gene product is a 60-kilodalton (kDa) protein and is a major constituent of the cell after induction of the SOS response (9, 31). Recently, *recJ* and *recQ* were shown to encode 53- and 68-kDa proteins, respectively (14, 23). The

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TABLE 1. SOS-associated recombination and DNA repair genes in *E. coli*

Gene	Map location (min)	Function	SOS gene	Molecular mass (kDa)
lexA	91	SOS repressor	Yes	22.7
phr	16	Photolyase	No	40–46
recA	58	General recombination, LexA repressor cleavage, SOS mutagenesis, RecF pathway of recombination and DNA repair	Yes	37.8
recB	61	Exonuclease V, RecBC-dependent recombination, SOS-inducing signal generation	No	134
recC	61	Exonuclease V, RecBC-dependent recombination, SOS-inducing signal generation	No	129
recD	61	Exonuclease V, RecBC-dependent recombination, SOS-inducing signal generation	No	67
recF	82	RecF-dependent recombination, daughter-strand gap repair, double-strand break repair	No	40
recJ	62	RecF-dependent recombination	Unknown	53
recN	58	RecF-dependent recombination, double-strand break repair	Yes	60
recO	55	RecF-dependent recombination	Unknown	Unknown
recQ	85	RecF-dependent recombination, resistance to thymineless death	Yes	68
ruv	41	RecF-dependent recombination, daughter-strand gap repair	Yes	41
sbcB	44	Exonuclease I, suppressor of *recBC*	No	53
sbcC	9	Suppressor of *recBC*	Unknown	Unknown
sulA (*sfiA*)	22	Cell division inhibitor	Yes	17–18
umuC	25	SOS mutagenesis	Yes	48
umuD	25	SOS mutagenesis	Yes	15
uvrA	92	Excision repair	Yes	103
uvrB	17	Excision repair	Yes	76
uvrC	42	Excision repair	Unknown	66
uvrD	85	DNA helicase II, RecF-dependent recombination, excision repair, methyl-directed mismatch repair, DNA replication	Yes	75

ruv gene has recently been cloned, and the gene product was identified as a 41-kDa protein, but the role of Ruv protein in repair is not yet known (2). DNA helicase II, encoded by *uvrD*, may be required to unwind DNA during recombination (12).

Cells mutant in only one of the RecF recombination pathway genes display variable phenotypes when treated with different DNA-damaging agents. *recA*, *recF*, *ruv*, and *uvrD* mutants are sensitive to both UV light and mitomycin C (40). Mutations in *recN* make cells mitomycin C sensitive but not UV sensitive; *recO* mutants have the opposite phenotype (16, 19). *recJ* and *recQ* mutations do not confer sensitivity to either of these agents (26). This differential sensitivity of RecF pathway mutants may reflect variations in the proteins required to repair a given type of lesion in the chromosome when this pathway of recombination is active.

The RecF pathway is the exclusive route of recombination in *recB* or *recC* mutants which are constitutively activated for RecF recombination by the *sbcB sbcC* double mutation (18). *recBC sbcBC* backgrounds cause increased expression of the SOS genes *recA*, *recN*, and *sulA*, as if expression of certain SOS-inducible products were important (essential?) for the viability of this type of mutant (19; K. R. Peterson and D. W. Mount, unpublished data). Presumably, the inducible products are rate limiting in repairing DNA.

The predominant mechanism of daughter-strand gap repair appears to be mediated through the RecF pathway of recombination. RecA, RecF, and Ruv proteins function directly in daughter-strand gap repair (40). Mutations in the genes encoding these proteins or in the regulatory genes that block induction of the SOS response (*lexA* or *recA*) cause deficiencies in daughter-strand gap repair.

Inducible double-strand break repair is performed by components of the RecF pathway and requires a functional *recA* gene and a functional *recN* gene (30). A *recB*-dependent process has been described, but the relationship between the *recN*-dependent and *recB*-dependent double-strand break repair mechanisms remains unclear. Introduction of an *sbcB* mutation into a *recB recC uvrA* strain, deficient in double-strand break repair, restores this capability to the cell, presumably by derepressing the RecF recombination pathway genes (i.e., *recN*) necessary for double-strand break repair (41). Further addition of a *recF* mutation blocks this repair capacity, suggesting a role for *recF* in double-strand break repair in *recB recC uvrA sbcB* cells.

There is evidence that RecA protein plays a direct role in the inducible RecF recombination pathway. Suppressors of the DNA repair and recombination deficiencies associated with *recB recC sbcB recF* or *recB recC sbcB recF uvrA* have been found and were mapped in *recA* (37, 38, 42). These mutations include *recA* suppressors of recF (Srf) and the *recA441*(Prt^c) allele. One role for *recF* in DNA repair and recombination may be to modulate RecA activities to allow RecA to participate in these *recF*-dependent processes. The *recA*(Srf) mutations may allow the RecA protein to partici-

TABLE 2. Mutations that derepress the SOS regulon

Gene	Protein	Process affected
dam	DNA adenine methylase	Methyl-directed mismatch repair
dnaB	DnaB helicase	Initiation of DNA replication
dnaE	α subunit of DNA polymerase III	DNA replication
dnaG	Primase	Initiation of DNA replication
lig	DNA ligase	Ligation of Okazaki fragments and other nicks
polA	DNA polymerase I	DNA replication, DNA repair
recF	Unknown	RecF pathway of recombination and DNA repair
ruv	Unknown	RecF pathway of recombination and DNA repair
ssb	Single-strand DNA-binding protein	DNA replication, recombination
uvrD	DNA helicase II	DNA replication, methyl-directed mismatch repair, excision repair, RecF pathway of recombination and DNA repair

pate in the *recF*-dependent activities without the need for the RecF protein.

INDUCTION OF EXCISION REPAIR GENES

Excision repair in *E. coli* is an error-free repair mechanism (39, 40). This repair path removes bulky lesions in DNA, such as aflatoxin B_1 and UV-induced pyrimidine dimers. The gene products of the SOS-regulated *uvrA*, *uvrB*, *uvrC*(?), and *uvrD* genes are required, as are DNA polymerase I and DNA ligase. Recently, the nucleotide sequence of *uvrB* was determined; the UvrB protein has a predicted size of 76 kDa (1, 3). A comparison of the amino acid sequences of the UvrB and UvrC proteins revealed regions of sequence homology, and the *uvrB* nucleotide sequence showed a consensus ATP-binding site. Since UvrB protein does not hydrolyze ATP, it may serve to bind ATP which is then hydrolyzed by UvrA in the UvrABC protein complex (3). The binding of UvrC to the UvrAB proteins leads to endonucleolytic activity of the complex (45). Recent work shows that in vitro, DNA polymerase I and UvrD protein stimulate release of the 12- to 13-base damage-containing DNA fragment. The resulting gap is filled by DNA synthesis mediated by DNA polymerase I and requires the action of UvrD protein (6). Photolyase, encoded by the *phr* gene, has been shown to augment UvrABC-directed excision repair in vitro and in vivo (11, 32). This enzyme stimulates the removal of pyrimidine dimers by UvrABC excision nuclease.

Evidence for the functional significance of derepressing excision repair genes comes from studies of the *lexA41* mutant. In this strain excision repair is faster than in a *lexA*$^+$ strain, and the mutant has been shown to constitutively express the excision repair genes *uvrA*, *uvrB*, and *uvrD* (10, 28).

Two types of excision repair have been described based on the amount of resynthesis that occurs; these are short-patch repair (described above) and long-patch repair. The majority of lesions are repaired with small patches, ranging from 10 to 20 nucleotides in length. Fewer lesions are repaired with much longer patches, averaging 1,500 nucleotides in length. Long-patch repair is an SOS function since such repair does not occur in mutants defective in the SOS response. Recent studies have suggested that any one of the three DNA polymerases can perform long-patch repair (7). In wild-type strains, DNA polymerase I seems to be the major polymerase involved in long-patch repair.

SOS MUTAGENESIS

SOS mutagenesis (formerly called error-prone repair) is one of the cellular processes controlled by the derepression of the SOS regulon, requiring the SOS genes *recA* and *umuDC*. Mutagenesis may not be a form of DNA repair, but rather may be a mode of damage tolerance. The molecular mechanisms for this process and their relationship, if any, to other repair processes have yet to be determined. The types of mutagenesis and their genetic requirements are not yet fully understood and may differ for single-stranded and double-stranded DNA, for damaged and undamaged target DNA sequences in which the mutation is scored, and for uninduced and induced cells (39). Current models suggest that during SOS induction the fidelity of DNA polymerase is relaxed, permitting DNA synthesis and misincorporation of nucleotides opposite lesions in the template strand (13).

Some types of mutagenesis found after treatment with agents such as ionizing radiation, UV light, or certain chemical mutagens do not occur in cells lacking an inducible SOS system. The products of at least two *lexA*-regulated operons, RecA protein and UmuDC proteins, are known to be required for certain types of SOS mutagenesis (39; Table 1). The *umuDC* operon encodes proteins with calculated molecular masses of 15 and 48 kDa, respectively (15, 27). Activated RecA protein (RecA*) is required for derepression of the SOS regulon and has a second, direct role in mutagenesis that is independent of its regulatory function (8, 44). Biochemical evidence indicates that RecA protein binds to UV photoproducts in DNA, suggesting that this binding may assist DNA polymerase in bypassing lesions (24). The role of UmuDC proteins in mutagenesis is unknown, but they may act as fidelity-relaxing factors for DNA polymerase, allowing a stalled polymerase complex to continue synthesis past lesions (5, 24). UmuD protein shows homology to the carboxy-terminal domain of the LexA repressor, including the region corresponding to the LexA cleavage site (27). However, the Ala-Gly bond defining the site of proteolysis in the LexA repressor and other repressors cleaved in a RecA-promoted reaction is a Cys-Gly bond in UmuD. Homology between UmuD and LexA may define a site of interaction between UmuD and RecA that is necessary for mutagenesis. Recently, we found that the selective derepression of the *umuDC* operon was sufficient to permit high levels of reversion of an amber mutation in UV-damaged bacteriophage lambda (D. G. Ennis, K. R. Peterson, and D. W. Mount, unpublished data).

In summary, recent experiments clearly establish that several sets of genes influencing different modes of DNA repair and mutagenesis are components of the SOS response. This response is induced after DNA damage or by mutations affecting DNA replication of metabolism. Different types of damage are repaired by increased expression of specific gene products having a variety of effects. Mutagenesis appears to be a mode of lesion tolerance involving specific proteins that are part of the SOS response.

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