Expression of the *Bacillus subtilis* dinR and recA Genes after DNA Damage and during Competence

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The *Bacillus subtilis* dinR gene product is homologous to the LexA protein of *Escherichia coli* and regulates the expression of dinR and dinC. Using transcriptional fusions in the dinR and the recA genes, we have investigated the epistatic relationship between these two genes during the SOS response induced either by DNA damage or by competence. The results show that after DNA damage, induction of the expression of both recA and dinR is dependent on the activity of the DinR and RecA proteins. A RecA-dependent activity on DinR is proposed as the initial event in the induction of the SOS network. In contrast, the competence-related induction of dinR and recA appears to involve two distinct mechanisms. While one mechanism corresponds to the classical regulation of the SOS response, the other appears to involve an activating factor. Moreover, this factor is active in cells in which competence is prevented by a mutation in the regulatory gene comA.

In *Bacillus subtilis*, an SOS system is induced when cells are exposed to conditions that damage DNA or undergo the competence state. Several phenomena that occur during the SOS response caused by DNA damage have been described elsewhere: overexpression of the din and recA genes (13), induction of DNA damage reparation (12), increase in the rate of mutagenesis (26), Weigé reactivation of prophages (5), induction of the lytic cycle of prophages (24), induction of the SPB DNA methylase (25), and development of cell filamentation (12). To date, three damage-inducible genes (din) have been identified: dinA (uvrA), dinB, and dinC (10, 11), as well as a uvrB gene (2, 18). Recently, a new din gene, dinR, was identified for *B. subtilis*. The dinR1 mutant was found to be deficient in both recombination and DNA repair. The DinR protein shares significant amino acid sequence similarities (47.3%) with the LexA protein of *Escherichia coli* (19). LexA is the repressor of the SOS gene network, and like LexA, DinR appears to be a regulator of the expression of other din genes (19, 22). In *E. coli*, induction of the SOS system results from the reversible activation of RecA, leading to the cleavage of the repressor of the SOS genes (22). The amino acid sequences in the three regions known to be required for the cleavage of *E. coli* LexA are highly conserved in DinR, suggesting that DinR could undergo similar RecA-mediated cleavage. *E. coli* and *B. subtilis* RecA proteins have several regions of similarity (21), and *B. subtilis* RecA is immunoreactive with antibodies raised against *E. coli* RecA. Furthermore, the *B. subtilis* RecA protein can facilitate *E. coli* LexA cleavage in vitro (14), and the *E. coli* RecA protein expressed on a plasmid in *B. subtilis* complements, at least partially, the recA4 (previously recE4) mutant for recombination, induction of din genes, and Weigé reactivation (13, 16). However, the *E. coli* RecA protein is unable to restore prophage induction in the recA4 mutant of *B. subtilis* (13), and the *B. subtilis* RecA cannot promote the cleavage of the ACl repressor molecule in vitro (14).

An additional regulation of the SOS system occurs when *B. subtilis* cells develop competence, a physiological state in which the cells are able to bind and take up exogenous DNA. Competence is expressed after exponential growth and is subject to three types of regulation: nutritional, growth-stage specific, and cell-type specific (for a review, see reference 4). Several genes that regulate the development of competence have been identified. The comA and comP genes belong to the family of two-component regulators (23) and encode a response regulator and a histidine kinase protein, respectively. The regulatory com genes are expressed throughout the *B. subtilis* growth cycle. To express late competence genes in addition to the identified early com gene products, a limiting transcription factor is necessary. A DNA fragment upstream of the promoter region of the comDE, comC, and comG genes appears to bind this competence transcription factor (CTF) (4, 17). The SOS system appears to be partially derepressed during competence, as judged by overexpression of the din (including dinR) and recA genes (11, 15, 19). The induction of the dinA, dinB, and dinC genes is observed only in the presence of wild-type RecA activity. During competence, the increased synthesis of RecA does not require the presence of a functional RecA protein; in the recA4 mutant, the competence-related stimulation of recA expression is observed, whereas overexpression of recA following DNA damage is inhibited (15). Overexpression of dinR is also observed during competence, even in the presence of the mutant DinR1 protein (19). These results indicate that another factor might be involved in the specific induction of the SOS response.

Within the promoter regions of several din genes, there are conserved sequences that have been proposed as recognition elements for the binding of a presumably common SOS-specific regulatory protein. These sequences have also been detected upstream of the recA, recM, and dinR genes (3, 19). In addition, a sequence that resembles the putative CTF binding element was found upstream of the recA gene (4). This finding raised the possibility that, in addition to the classical SOS regulation, a dual regulation of recA involves the binding of CTF and the subsequent displacement of the repressor of the SOS genes, leading to induction of recA expression during competence (4).

We have investigated the epistatic relationship between dinR and recA by measuring (i) expression of recA in
TABLE 1. B. subtilis strains used in this study

| Strains         | Genotype and/or relevant characteristics | Reference or source |
|-----------------|------------------------------------------|---------------------|
| 1680*           | Wild type, prototrophic                   | Our collection      |
| MOS06           | trpF7 ura-1 thr-5 acf-1 rfm-486           | Our collection      |
| MOS34           | dinRI::Tn917lac                           | 19                  |
| M056            | 1680* [pB16]; Campbell recombinant* in dinR | 19                  |
| BD1626          | hisA1 metB leu coA124::pTV21Δ2; Lac- derivative | 9                   |
| YB886::recA::cat| metB5 trpC2 xin-1 SFP- recA2              | R. Yasbin           |
| QB4444          | trpC2 recA2 (recA::aphA3)                 | F. Kunst            |
| BG225           | (recA::xylE)                              | J. Alonso           |
| MO551           | coA124::pTV21Δ2 dinR::Tn917lac recA2      | This work           |
| MO569           | recA::xylE                                | This work           |
| MO579           | 1680* [pB16] recA2                       | This work           |
| MO572           | 1680* [recA::xylE]                       | This work           |
| MO573           | dinRI::Tn917lac recA::xylE               | This work           |
| MO574           | 1680* [pB16] recA::xylE                  | This work           |

* Brackets indicate an integrated plasmid molecule.

MATERIALS AND METHODS

Bacterial strains, plasmids, and growth media. B. subtilis strains were derivatives of wild-type strain 168 and are listed in Table 1. Construction of the dinR merodiploid strain that contains both the dinR-lacZ fusion and a wild-type copy of dinR has been previously reported (19). The recA::cat mutation from the strain YB886::recA::cat was transduced into the dinRI strain MO534 to create the strain MO569. The strain MO565 was constructed by Campbell-like recombination between the pB16 plasmid, which carries the dinR-lacZ fusion and the cat gene, and the chromosome of the wild-type strain. Because strain MO565 was resistant to chloramphenicol, we performed its transformation with a recA::aphA3 null allele (Km'); recently constructed by F. Kuntz. The resulting strain, MO579, contains the dinR-lacZ fusion along with an intact copy of the dinR gene and the recA::aphA3 mutation. Chromosomal DNA from QB4444, a strain which carries the recA::aphA3 mutation, was used to transform competent MO565 cells to kanamycin resistance (Km'), thereby generating the strain MO579. We renamed recA::cat and recA::aphA3 the recA2 allele of recA; in these constructions, the cat or aphA3 gene was inserted into the ClaI site of recA. To introduce the recA::xylE fusion into the wild-type and dinRI strains, we used the strain BG225, which carries the recA::xylE gene fusion ectopically integrated onto the chromosome. The chromosomal DNA purified from BG225 (recA::xylE) was mixed (1:1) with DNA purified from MOS06 (Rif'), Rif' recA::xylE recombinants were then obtained by co-transformation of these two markers into the wild-type strain and strain MO556. The Rif recombinants were selected in rich medium in the presence of rifampycin and then sprayed with a 0.5 M solution of carboxyl to reveal the activity of the xylE gene product, catabol 2,3-oxygenase (catOase) (27). By a similar method, the wild-type strain was also transformed for dinRI::Tn917lac and recA::xylE. In this case, DNA from BG225 (recA::xylE) was mixed with DNA purified from MO534 (dinRI:: Tn917lac Ery'), and recombinants were selected on medium containing erythromycin and cspayed with the carboxyl solution. The strains MO572 (recA+ recA::xylE rfm-486), MO573 (dinRI::Tn917lac recA+ recA::xylE), and MO574 (dinR' dinR::Tn917lac recA+ recA::xylE rfm-486) were subsequently purified by streaking them on rich medium in the presence of the appropriate antibiotic. The coA124 mutation from BD1626 (9) was moved into the strain MO534 by transformation.

Plasmid pB16 has been previously described (19) and contains the BgIII promoter proximal clone of the dinR::lacZ fusion from strain MO542. Erythromycin at 5 μg/ml, lincomycin at 20 μg/ml, mitomycin (MC) at 50 or 150 ng/ml, rifampicin at 5 μg/ml, kanamycin at 5 μg/ml, chloramphenicol at 5 μg/ml, and 5-bromo-4-chloro-3-indolyl-β-D-galactopyranoside (X-Gal) at 40 μg/ml were utilized.

Genetic and molecular procedures. Genetic transformation, transduction, development of competence, and chromosomal DNA purification were performed as described previously (6).

Enzymatic assays. β-Galactosidase assays were conducted by using bacteria grown in Luria-Bertani or Schaeffer’s medium; no significant difference in the β-galactosidase activity between bacteria grown in each of the two media was observed. For the catOase assay, strains were grown in Luria-Bertani medium because the fusion has no activity in Schaeffer’s medium. For measuring enzymatic activities during competence, a one-step protocol was used as described previously (1). In particular experiments, MC was added at 150 ng/ml during the exponential phase of growth. The level of β-galactosidase activity was determined as described previously (27) and was expressed as units per milligram of protein. The amount of protein was determined from standard curves by relating the turbidity (optical density at 570 nm) to the protein concentration of the bacterial cultures. Preparation of samples from culture and determination of catOase activity were performed as described elsewhere (27), with minor modifications: the cells were washed in 20 mM potassium phosphate buffer (pH 7.2) and resuspended in AP buffer (100 mM potassium phosphate buffer [pH 7.5] and 10% acetone [vol/vol]). Whole cells were disrupted by 30 min of incubation with 50 μg of lysozyme per ml at 37°C and then stored at 4°C. The catOase activity was determined by following the increase in A730 due to the accumulation of 2-hydroxy-3-monic semialdehyde. Milliunits of activity are defined as described by Sala-Trepas and Evans (20).

RESULTS

Expression of dinR after DNA damage. The previously characterized dinRI mutant was deficient in both recombination and DNA repair, and the expression of the dinRI allele was noninducible by DNA damage. In the dinR
merodiploid strain MO556, which carries an intact copy of the dinR gene coexistent with the dinRI::Tn917lac mutation, restored, and DNA repair capacities were restored, and the expression of the dinR gene became inducible by DNA-damaging agents (19). To investigate the role of RecA on the expression of dinR, a recA2 null allele (recA::cat or recA::aphA3) was introduced into strains MO534 (dinRI) and MO556 (dinR+/dinRI), each of which contains a dinR-lacZ fusion. The double mutant strain MO569 (dinRI recA2) had a lower growth rate than the parental dinR strain, which had a rate of growth equivalent to that of the wild-type strain. The doubling time of strain MO569 in rich medium was 35 min, whereas the doubling time of strain MO534 was 23 min. In addition, strain MO569 demonstrated a phenotype of extensive filamentation, which was not observed in the recA or dinR mutants.

The β-galactosidase activities of cultures of MO534 (dinRI recA+), MO569 (dinRI recA2), MO556 (dinR+/dinRI recA+), and MO579 (dinR+/dinRI recA2) were measured, in the presence or absence of MC (Table 2). Increased expression of the dinR gene in the presence of MC was seen only in strain MO556 (dinR+/dinRI recA+); β-galactosidase activity observed 3 h after the addition of MC was 4.5-fold greater than that observed in the absence of MC. In addition, the basal level of dinR-lacZ expression in this strain was three- to fourfold higher than that of the other strains. In contrast, no significant increase in the expression of the dinR-lacZ fusion in the presence versus the absence of MC was observed in strains MO534 (dinRI recA+), MO569 (dinRI recA2), and MO579 (dinR+/dinRI recA2). Comparison of dinR expression in strain MO556 (dinR+/dinRI recA+) with its expression in strain MO579 (dinR+/dinRI recA2) indicates that the absence of a functional RecA protein prevents the induction of the dinR gene after DNA damage. These results suggest that a RecA-dependent activity is necessary for the induction of dinR expression following DNA damage.

**Effect of DinR on recA expression following MC treatment.**

The DinR protein is a protein that regulates the expression of both dinR and dinC (19). Another important protein in the function of the SOS response in B. subtilis is RecA. To measure the effect of DinR on the transcription of the recA gene, a recA::xyIE gene fusion was introduced into the wild-type, dinR, and merodiploid dinR+/dinR1 strains. All of the resultant strains are merodiploid for the recA gene, with the wild-type copy of recA at its normal position on the B. subtilis chromosome and the recA::xyIE gene fusion integrated ectopically. Expression of the recA::xyIE fusion was measured in the following strains: MO572 (wild type), MO573 (dinRI), and MO574 (dinR+/dinRI) (Table 3). The levels of recA expression during growth were extremely different for the three strains. When the recA::xyIE fusion was introduced into the dinR1 mutant strain (MO573), the basal level of recA expression was only 10% of that of the wild-type strain (MO572). In the dinR merodiploid strain (MO574), the level of recA::xyIE expression was approximately twofold higher than that of the fusion in the wild-type strain. In all of these strains, a 7- to 10-fold increase of catO2ase activity was observed when cultures in rich medium entered the stationary phase of growth. A similar unexplained increase was observed by Gassel and Alonso when the recA::xyIE fusion was carried on a multicopy plasmid (7).

In the wild-type strain grown in rich medium, expression of the recA::xyIE fusion started to increase 1 h after the addition of MC. After 3 h of MC treatment, the recA::xyIE expression increased to 5.5-fold its expression in the untreated culture (Table 3 and Fig. 1). Induction of recA expression was also observed in the dinR merodiploid background (strain MO574), indicating that wild-type regulation of recA expression occurs in the merodiploid strain. In contrast, no induction of the recA::xyIE fusion occurred in strain MO573 after MC treatment. These results suggest the following hypotheses. (i) DinR is the repressor for the damage-inducible genes and is inactivated after DNA damage, thereby leading to the expression of the SOS genes. In this case, the DinR protein is a noninactivable mutant protein which acts as a super repressor. (ii) DinR is a positive regulator necessary for the induction of recA. Furthermore, the low level of expression of recA found in the dinR1 mutant might explain the Rec− phenotype of this strain (19).

**Effect of RecA on competence-induced dinR expression.**

When B. subtilis cells reach competence, specific induction of the din and recA genes occurs (11, 15). While induction of

| Time after induction (h) | MO534 (dinRI recA+) | MO569 (dinRI recA2) | MO556 (dinR+/dinRI recA+) | MO579 (dinR+/dinRI recA2) |
|--------------------------|---------------------|---------------------|---------------------------|--------------------------|
| -MC | +MC | -MC | +MC | -MC | +MC | -MC | +MC |
| 0.5 | 43 | 20 | 135 | 56 | 94 |
| 0 | 25 | 16 | 113 | 59 | 116 |
| 0.5 | 28 | 32 | 120 | 69 | 71 |
| 1 | 50 | 33 | 163 | 92 | 74 |
| 2 | 68 | 35 | 163 | 79 | 47 |
| 3 | 64 | 25 | 161 | 52 | 44 |

* - MC, growth in rich medium without added MC; + MC, growth in rich medium with added MC (150 µg/ml).
the dinA, dinB, and dinC genes is dependent on the wild-type RecA protein, induction of the recA gene occurs also in the presence of the RecA4 inactive protein. To investigate whether competence-related induction of dinR is dependent on the recA gene product, expression of the dinR-lacZ fusion in strains M0534 (dinRI recA4), M0569 (dinRI recA2), M056 (dinR+ dinR1 recA4), and M0579 (dinR+ dinR1 recA2) was measured (Table 4). In the dinR merodiploid strain M0556, a threefold increase in dinR expression was observed when the cells developed competence. In strain M0579 (dinR+ dinR1 recA2), a twofold increase of dinR expression was observed, but only 2 h after the end of exponential growth. In addition, the basal level of dinR expression (at 0.5 h before end of log-phase growth; T = 0) was fivefold lower in strain M0579 than in strain M0556. These results indicate that the RecA protein is (i) necessary for the normal timing of induction of dinR expression during competence and (ii) necessary for the maintenance of a basal level of DinR during growth.

A competence-related increase of β-galactosidase activity was also observed in strains M0534 (dinRI) and M0569 (dinRI recA2) (Table 4), indicating that in these strains the augmentation of dinR expression occurs in the presence of the DinRI protein and in the absence of RecA. These results may indicate that the competence-related dinR induction occurs independently of any activity of RecA on DinR. In addition, dinR expression during vegetative growth (T = 0.5) was lower in both strains M0534 and M0569 than in strain M0556. This suggests that the DinR protein also contributes to the basal level of dinR expression. Such a contribution is evident when strains M0556 and M0534 are compared.

**Effect of DinR on the competence-related induction of recA.** Previous work has shown that the recA gene is induced in competent cells (15). This induction occurs in the recA4 mutant strain, which encodes an SOS-inactive form of the RecA protein. To investigate whether the competence-related induction of recA is dependent on the activity of DinR, the expression of the recA::xyIE fusion was measured during competence. In the dinR+ strain (M0572), the recA::xyIE fusion was induced 10-fold 2 h after the end of exponential growth, whereas no induction of recA in the dinR1 mutant was observed (Fig. 2). These results suggest that the competence-related overexpression of recA requires the presence of a wild-type DinR protein. Taking into account that only 10 to 20% of cells reach competence (4), the observed values (in terms of milliunits of catO2ase per milligram of protein) for competence-related recA induction are underestimates in these experiments.

**Effect of comA on competence-related expression of dinR.** Transcriptional activation of the dinR gene has been observed when *B. subtilis* becomes competent, even in the absence of any known DNA-damaging agent (19). The competence-related induction of dinR expression occurs in the dinR1 mutant, thereby indicating that it is independent of the DinR wild-type protein. The induction of dinR could thus be controlled by comA, a major regulator of competence gene expression (9). To investigate the role of comA in competence-induced expression of dinR, the comA124 allele was introduced by transformation into strain M0534 (see Materials and Methods). β-Galactosidase activities in the dinR1 mutant (M0534) and in the dinR1-comA double mutant (M0551) were measured (Fig. 3). Comparable levels of expression of the dinR gene occurred in the two strains when the cells became competent, thereby indicating that dinR overexpression is independent of the comA gene product.

**DISCUSSION**

The development of the competence state in *B. subtilis* is accompanied by induction of the din and recA genes, the expression of which is also induced by DNA-damaging agents (11, 15). Both the competence- and the DNA damage-

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**TABLE 4. Effect of RecA on the expression of the dinR-lacZ fusion during competence**

| Strain | Relevant genotype | β-Galactosidase sp act (U/mg of protein) |
|--------|------------------|----------------------------------------|
|        |                  | T0 | T1 | T2 | T3 |
| M0556  | dinR+ dinRI recA2 | 216 | 256 | 420 | 552 | 654 |
| M0534  | dinRI recA2      | 82  | 84  | 167 | 204 | 246 |
| M0569  | dinRI recA2      | 51  | 46  | 92  | 169 | 247 |
| M0579  | dinR+ dinRI recA2| 44  | 46  | 55  | 92  | 101 |

* T0, the end of the exponential growth (subscript numerals are in hours).
related inductions of the din genes require a functional RecA protein. In contrast, while the induction of recA expression after DNA damage requires a functional RecA protein, the competence-induced expression of recA occurs independently of the presence of active RecA (15). The recA gene seems to be under two types of regulation, one active after DNA damage and the other active when the cells reach competence.

We have previously reported the identification of dinR, a regulator of din gene expression, which encodes a protein homologous to the LexA protein of E. coli (19). The dinR1 mutant, originally identified as deficient for homologous recombination, encodes a truncated form of the DinR protein which lacks 20% of the wild-type protein at the carboxyl terminus. Expression of dinR is induced following DNA damage and when the cells become competent. In contrast to the DNA damage induction of dinR, its competence-related induction occurs in the dinR1 mutant. In this work, we have investigated the epistatic relationship between the B. subtilis recA and dinR genes during both the SOS response and the development of competence.

Induction of a dinR-lacZ fusion following DNA damage was abolished in both recA2 and dinR1 mutants, indicating that similar effects result from the presence of the DinR protein (or absence of DinR) and from the absence of the active RecA protein. These observations support two hypotheses. (i) The amino acid motifs known to be necessary for the cleavage of the E. coli LexA protein are conserved in DinR, suggesting that the DinR protein could undergo a similar RecA-mediated cleavage (19). In this case, DinR normally acts as a repressor of the SOS genes and the DinR1 protein is a noncleavable form of DinR. (ii) DinR is a positive effector necessary for the expression of the SOS genes, and the DinR1 protein has lost this activity (discussed below).

Expression of the recA gene is highly influenced by the dinR allele present in the cell. In the presence of the dinR1 mutation, recA expression was only 10% of that obtained in the presence of the dinR wild-type allele. This low rate of recA expression in the dinR1 background might explain the Rec- phenotype of the DinR1 mutant (19). In the dinR merodiploid strain, both DinR and DinR1 are synthesized, and the level of recA::xyfE expression was approximately twofold higher than that of the fusion in the wild-type strain. Thus, our results show that dinR, dinC, and recA are poorly expressed in the presence of the dinR1 allele and that in the merodiploid strain the expression of recA and dinR is higher than that in the wild-type strain. These results lead us to favor the first hypothesis, in which DinR acts as a repressor for damage-inducible genes such as recA, dinC, and dinR.

The presence of the conserved SOS boxes in the regulatory regions of these genes and of dinA, dinB, and recM (3, 19) suggests a possible interaction of DinR with these DNA sequences. From the results concerning dinR and recA expression during vegetative growth, we can conclude that a reciprocal regulatory effect allows a basal level of expression of these genes. A permanent low level of DinR expression appears to be necessary to ensure a basal quantity of RecA, which accounts for the induction of the SOS response. The presence of a DinR protein leads to an SOS induction-deficient phenotype. In contrast, RecA wild-type activity is needed to maintain the DinR constitutive level and to derepress dinR transcription following DNA damage. These results allow a comparison between the SOS responses of B. subtilis and E. coli (22), in which there is an interdependence of RecA and LexA in the regulation of the SOS network.

dinR expression, like that of the din and recA genes, is induced when the cells become competent. In this case, the RecA protein has no effect on dinR induction in the dinR1 mutant, supporting the hypothesis that the DinR1 protein is insensitive to RecA activity. Moreover, the absence of the RecA protein in the merodiploid strain was associated with a diminution of competence-related dinR induction. These results suggest that the DinR protein synthesized in the merodiploid strain can be modified by some RecA activity, leading to overexpression of the dinR gene. This mechanism is consistent with dependence on RecA for the induction of the other din genes during competence. Moreover, in the dinR1 strain, when the cells reached competence, induction of recA expression was abolished whereas that of dinR1 was still present. These results suggest that DinR1 regulation of the recA promoter prevents recA induction. However, the activity of RecA on DinR does not seem to be the only way to activate dinR expression during competence. In the dinR1-recA2 double mutant, as in the dinR1 strain, the competence-related overexpression of dinR is still observed. Thus, the RecA-dependent activity on DinR, necessary for SOS induction, could be bypassed by another mechanism during competence. The competence-related overexpression of recA depends on the presence of a DinR wild-type protein (Fig. 2); nevertheless, this induction is also observed in the recA4 mutant, which encodes an inactive form of the RecA protein (14). These results could also indicate that the RecA-dependent activity on DinR is not necessary for the induction of the recA gene; it is possible that the dinR and recA genes are also under the regulation of an unidentified activator that is independent of the SOS regulation. This putative factor would be able to displace DinR (or DinR1) from its target sequence without RecA-dependent activity, thereby permitting the transcription of the dinR or the recA gene. Overexpression of dinR during competence was observed in the dinR1 comA124 double mutant, indicating that the competence-induced expression of dinR is independent of the regulator ComA. Thus, expression of the putative activator of dinR must also be independent of ComA. This putative factor is different from the CTF evoked as a regulator of the SOS competence phenomenon, since CTF is not synthesized in the comA124 mutant (4).

In conclusion, we propose the following hypothesis to
explain the double regulation of dinR and recA expression observed during competence. When competence is induced in the wild-type cell, a low level of expression of the dinR and recA genes occurs by a mechanism that involves the presence of an activating factor that displaces DinR from the dinR and recA promoters. This mechanism does not involve any activity of RecA on the DinR protein, thereby explaining the overproduction of DinR and RecA during competence in the dinR1 and recA47 mutants. A competition between DinR1 and the putative positive activating factor for binding to the recA promoter would account for the low level of transcription of recA in the dinR1 mutant and for its Rec− phenotype. In parallel, the SOS signal might be produced by a limited rate of DNA replication in competent cells (4). At this time, the survival of the cell would be guaranteed by a derepression of the SOS functions which might be due to RecA-mediated cleavage of DinR. Subsequently, full induction of the SOS system occurs, and the resultant increased quantity of RecA allows genetic recombination and derepression of the din genes.

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