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Comparative Gene Expression Profiles Following UV Exposure in Wild-Type and SOS-Deficient *Escherichia coli*

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ABSTRACT

The SOS response in UV-irradiated *Escherichia coli* includes the upregulation of several dozen genes that are negatively regulated by the LexA repressor. Using DNA microarrays containing amplified DNA fragments from 95.5% of all open reading frames identified on the *E. coli* chromosome, we have examined the changes in gene expression following UV exposure in both wild-type cells and *lexA1* mutants, which are unable to induce genes under LexA control. We report here the time courses of expression of the genes surrounding the 26 documented *lexA*-regulated regions on the *E. coli* chromosome. We observed 17 additional sites that responded in a *lexA*-dependent manner and a large number of genes that were upregulated in a *lexA*-independent manner although upregulation in this manner was generally not more than twofold. In addition, several transcripts were either downregulated or degraded following UV irradiation. These newly identified UV-responsive genes are discussed with respect to their possible roles in cellular recovery following exposure to UV irradiation.

IRRADIATION of growing *Escherichia coli* cultures with ultraviolet light (UV) produces DNA lesions that at least transiently block the essential processes of replication and transcription. A large amount of work has demonstrated that the cell responds to this stress by upregulating the expression of several genes that function to repair the DNA lesions, restore replication, and prevent premature cell division. A number of other genes are known to be upregulated, yet remain functionally uncharacterized. The changes in gene expression in response to DNA damage produced by UV and some other environmental agents have been collectively termed the SOS response, after the international distress signal (Radman 1974 and reviewed in Friedberg *et al.* 1995; Koch and Woodgate 1998).

Many of the DNA damage-induced genes are negatively regulated by the LexA repressor protein, which binds to a 20-bp consensus sequence in the operator region of the genes, suppressing their expression (Brent and Ptashne 1981; Little *et al.* 1981). Derepression of these genes occurs when the RecA protein binds to single-stranded regions of DNA created at replication forks when they are blocked by DNA damage. RecA bound to single-strand DNA becomes conformationally active, serving as a coprotease to cleave the LexA repressor. As the cellular concentration of LexA diminishes, the genes normally suppressed by LexA are more frequently transcribed (Sassanfar and Roberts 1990 and references therein; Friedberg *et al.* 1995). An interesting feature of the LexA/RecA regulatory circuit is that the timing, duration, and level of induction can vary for each LexA-regulated gene, depending upon the location and binding affinity of the LexA box(es) relative to the strength of the promoter. As a result of these properties, some genes may be partially induced in response to even endogenous levels of DNA damage, while other genes appear to be induced only when high or persistent DNA damage is present in the cell. In fact, the SOS response may represent a continuum in the monitoring of environmental stress, rather than simply operating as an emergency switch following acute injury.

The first systematic search for *d*amage-*in*ducible (*din*) genes was carried out by Kenyon and Walker (1980) by randomly inserting a *lac* reporter gene into the *E. coli* chromosome to identify promoters that were upregulated following DNA damage in a *recA*/*lexA*-dependent fashion. Using this same technique, subsequent studies identified additional *din* genes and in some cases identified genes previously characterized to be involved in the recovery from DNA damage (Bagg *et al.* 1981; Fogliano and Schendel 1981; Huisman and D'Ari 1981; Kenyon and Walker 1981; Shurvinton and Lloyd 1982; Lloyd *et al.* 1983; Siegel 1983; Bonner *et al.* 1990; Iwasaki *et al.* 1990; Ohmori *et al.* 1995b). Analysis of the known *din* genes revealed a 20-bp consensus LexA-binding motif, or "SOS box," shared by these genes in their promoter/operator regions (Walker 1984), which has been used in more recent studies to systemati-

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cally search and identify additional *lexA*-regulated genes (Lewis *et al.* 1994; Ohmori *et al.* 1995a; Fernandez De Henestrosa *et al.* 2000). These studies in total have identified 31 genes under *lexA/recA* control.

Other genes have been reported to be upregulated following DNA damage but are believed to be independent of the *lexA* regulon. In some cases, the induction is thought to be dependent on *recA*, but independent from the LexA repressor. In other cases, genes have been shown to be upregulated independently from both *recA* and *lexA* (Friedberg *et al.* 1995; Koch and Woodgate 1998). The mechanism of regulation in these cases is not understood. An additional, although as yet unexplored, possibility is that some genes may be repressed or their transcripts may be degraded in response to DNA damage.

It was of interest to us not only to learn whether additional genes can be regulated in a LexA-dependent manner but also to determine whether other cellular responses to UV irradiation exist that are *lexA* independent. The *lexA1* allele encodes an amino acid change at a position that is essential for the cleavage and inactivation of LexA (Slilaty and Little 1987). Thus, in *lexA1* mutants, the LexA1 concentration remains high and LexA-regulated genes are not induced, even in the presence of high levels of activated RecA. Therefore this system should allow an analysis of gene expression that occurs independent of the LexA repressor.

The changes in gene expression in the entire genome can be measured simultaneously using high-density cDNA microarrays (Schena *et al.* 1995). DNA microarrays contain PCR-amplified DNA fragments of known and predicted genetic sequences that are printed on the surface of a glass slide. Through the comparative hybridization of two cellular RNA preparations, the relative difference between transcript levels of any gene in these preparations can be determined. Using the complete sequence of the *E. coli* genome (Blattner *et al.* 1997), DNA microarrays were prepared containing PCR products corresponding to 95.5% (4101 out of 4295) of all annotated open reading frames in the *E. coli* genome (Khodursky *et al.* 2000). We utilized these microarrays to follow the changes in gene expression occurring during the first hour following UV irradiation in the wild-type strain, MG1655, and in an isogenic *lexA1* mutant.

MATERIALS AND METHODS

Bacteria: Strain MG1655 was used as the wild-type strain in this study since its genome has been completely sequenced (Blattner *et al.* 1997). The MG1655 *lexA1*(Ind−) *malB*::Tn9 was constructed by P1-mediated transduction of the *lexA1* allele from strain GC2281 (Taddei *et al.* 1995) into strain MG1655. Transfer of the *lexA1* allele was verified by resistance to chloramphenicol and hypersensitivity to UV irradiation.

Growth and irradiation: Cells were grown in Davis medium plus 0.4% glucose. Cultures were inoculated at a 1:200 dilution from a fresh overnight culture into 200 ml Davis media and incubated in a 1-liter Erlenmeyer flask at 37° in a New Brunswick Scientific (Edison, NJ) model G76 gyrotory water bath at 220 rpm to midlog (OD_{600} 0.4, $\sim 2 \times 10^8$ cells/ml). A 15-W germicidal lamp (254 nm, 0.66 J/m^2/sec at the sample position) provided the UV irradiation. A total of 70 ml of culture was placed into a 15-cm-diameter glass petri dish and irradiated for 60 sec with gentle agitation. Two 65-ml unirradiated samples were also agitated in a 15-cm petri dish but were not exposed to UV. A total of 70 ml of irradiated culture (in a 500-ml Erlenmeyer flask) and 30 ml of unirradiated culture (in a 250-ml Erlenmeyer flask) were then returned to the shaking water bath for the duration of the time course. At the appropriate times, 10-ml samples were placed into 20 ml of ice-cold NET (100 mm NaCl, 10 mm Tris, 10 mm EDTA), pelleted by centrifugation, washed with 1 ml cold NET, repelleted, and frozen at −80°. The limited availability of microarray chips constrained this experiment to a single time course containing seven samples (five irradiated, two unirradiated) for each strain.

Microarray procedures: Relative mRNA levels were determined by parallel two-color hybridization to cDNA microarrays representing 4101 open reading frames (ORFs) representing 95.5% of *E. coli* ORFs according to Blattner *et al.* (1997). cDNA arrays were manufactured as described in MGuide at http://cmgm.stanford.edu/pbrown/mguide/index.html. Total mRNA was extracted from 2–5 × 10^9 cells using QIAGEN (Chatsworth, CA) RNeasy spin columns. A total of 25–30 μg of total RNA was labeled with Cy-3-dUTP (or Cy-5-dUTP) in a standard reverse transcriptase (RT) reaction by Superscript II (+) (GIBCO BRL, Gaithersburg, MD) with 1 μg of random hexamer (Pharmacia, Piscataway, NJ) primers. Following purification through Microcon-30 (Millipore, Bedford, MA) (MGuide), Cy-3- and Cy-5-labeled cDNA were combined with SSC (2.5× final), SDS (0.25%), and 40 μg of *E. coli* rRNA (Boehringer Mannheim, Indianapolis) in a final volume of 16 ml and hybridized to a DNA microarray for 5 hr at 65°. Slides were washed as described in MGuide and scanned using an AxonScanner (Axon Instruments, Foster City, CA; GenPix 1.0) at 10 mm per pixel resolution. Acquired 16-bit TIFF images were analyzed using ScanAlyze software, which is publicly available at http://rana.stanford.edu/software/.

Comparative measurements of transcript abundance: Time course samples were analyzed directly by comparing the abundance of each gene's transcripts relative to the t_0 sample. RNA samples taken during the time course were labeled with Cy-5, and RNA from the t_0 sample was labeled with Cy-3.

Sequence analysis: Nucleotide sequences in the regions of induced genes were examined using the COLIBRI program provided by the Pasteur Institute at http://genolist.pasteur.fr/colibri/. Regions surrounding induced genes were searched for the consensus sequence CTG(N)$_{10}$CAG, allowing for one mismatch. Matching sequences that fell between −400 and +100 bp of a start codon were then examined for their heterology index. The heterology index was determined as reported in Lewis *et al.* (1994) on the basis of the formula developed by Berg and von Hipple (1988). Heterology index = $\sum \ln[(n_{(consensus)} + 0.5)/(n_{(actual)} + 0.5)]$, where n(consensus) refers to the number of times that the most common, or consensus, base occurs at a given position in the set of known binding sites, and $n_{(actual)}$ refers to the number of times that the base being analyzed occurs at the same position in the set of known binding sites. n values for each position of the 20-bp LexA binding site were determined using the known LexA-binding sites shown in Figure 1A and their respective complementary sequences.

Nomenclature: All genes are named according to the Rudd system at http://bmb.med.miami.edu/ecogene/ecoweb (Rudd

2000). In cases where we found no corresponding Rudd gene for the open reading frame examined, the original identification numbers of Blattner, b#### (Blattner *et al.* 1997), were used.

Raw data: The raw data from these experiments are available for download at the following web address http://www2.msstate.edu/~jcc129.

RESULTS AND DISCUSSION

We examined the response of *E. coli* strain MG1655 following a dose of 40 J/m^2 (254 nm). Previous studies in our laboratory have shown that exposing an exponentially growing culture of *E. coli* to 40 J/m^2 of UV produces approximately one cyclobutane pyrimidine dimer on each strand per 6 kb of DNA (Mellon and Hanawalt 1989; Crowley and Hanawalt 1998). This dose transiently inhibits both replication and transcription, and induces a strong SOS response (Courcelle *et al.* 1997; Crowley and Hanawalt 1998). More than half of the cells survive and genomic replication fully recovers within ~45 min following UV irradiation. Within that time, most of the DNA lesions have also been repaired (Mellon and Hanawalt 1989; Courcelle *et al.* 1999).

To examine the changes in gene expression in response to this dose of UV irradiation, we compared samples of total RNA taken 5, 10, 20, 40, and 60 min after irradiation to samples made just prior to irradiation. To control for UV-independent changes, total RNA preparations from nonirradiated samples at 20 and 60 min were also prepared. This analysis was carried out with the wild-type MG1655 strain as well as the isogenic *lexA1* derivative and represents the changes in transcript levels of each gene from up to seven independent comparative hybridizations for each cell line, which were observed within the same experiment.

The average change in transcript level in the irradiated samples compared to those in the unirradiated samples for each gene along the *E. coli* chromosome is plotted sequentially in Figure 1. In some cases, no data were plotted for a gene, because either the PCR reaction failed during microarray construction or the fluorescent signal in the unirradiated samples was too low for reliable detection. However, raw data for any or all genes are available for downloading at the web addresses indicated in materials and methods or upon request to the authors.

Genes induced in a LexA-dependent manner following UV irradiation: Twenty-six functional LexA-binding regions controlling at least 31 genes have been previously demonstrated to be functionally active following irradiation. At the time at which these bacterial microarrays were constructed, 3 of these genes, *ysdAB*, *dinQ*, and *dinS*, had not yet been identified as open reading frames (Fernandez De Henestrosa *et al.* 2000) and were not included in our analysis. The time courses observed for all other genes/operons known to be regulated by LexA and that contain LexA-binding sites are plotted in Figure 2A. For most of the LexA-regulated genes, the level and timing of the induction observed in our experiments are in good agreement with previous observations. In confirmation of previous studies, we find that *recN*, *recA*, and *sulA* are heavily induced within the first 5 min of irradiation whereas the *uvrD* induction is much less robust (Casaregola *et al.* 1982; Arthur and Eastlake 1983; Salles and Paoletti 1983; Picksley *et al.* 1984; Sandler 1994). *umuCumuD* are also known to be strongly induced; however, full induction of these genes is not observed until 20 min after UV irradiation (Woodgate and Ennis 1991). We were unable to assay *ftsK* induction in wild-type cells due to a problem amplifying the *ftsK* fragment when constructing the bacterial microarray. However, some lexA-dependent induction is observed in *lolA,* possibly representing some transcriptional readthrough from the *ftsK* gene. In some cases, we observed co-upregulation of the neighboring ORFs that are transcribed in the same orientation. This effect can clearly be seen in the induction of *dinB* transcription, which also renders an increase in *yafN*, *yafO*, and *yafP* mRNA. Similarly, *yebF* and *yebE* are also induced along with LexA-regulated *yebG.* In the case of *recN*, the downstream genes *smpA* and *smpB* also appear to be upregulated following UV irradiation. However, for *b2619* and *b2618* it is actually the antisense strand that would be transcribed following UV irradiation if this induction represents transcriptional readthrough. The actual mechanism of such coregulation could be produced by: (1) transcriptional readthrough resulting from inefficient transcriptional termination; (2) the actual operon spanning across the entire group of neighboring genes; or (3) a regional effect conferred through protein factors or DNA structural conformations within the region under consideration.

Some of the transcripts from documented LexA-regulated genes, *dinG*, *molR*, *uvrD*, and *uvrA*, did not significantly rise following UV irradiation. However, in each of these cases, the samples of these transcripts in the unirradiated (control) culture were significantly decreased during the time course. The reason for this observation is unclear. However, both initial and unirradiated samples were "mock" UV treated by gentle agitation for 60 sec in a 15-cm glass petri dish and it is possible that some genes were affected by this treatment. Importantly, however, when comparing the net change in irradiated and unirradiated samples, the *lexA*-dependent induction of these genes is clearly evident: 1.77-, 1.78-, 2.51-, and 3.85-fold increases, respectively.

Of the reported LexA-regulated genes, we did not detect significant induction of either *hokE* or *ssb* in our experiment. *recA*/*lexA*-dependent transcription from *hokE* has previously been shown to occur in the *E. coli* strain RW118 following mitomycin C treatment (Fernandez De Henestrosa *et al.* 2000). However, the SOS induction of *ssb* is less clear. Although a plasmid-encoded *ssb* has been shown to be slightly upregulated

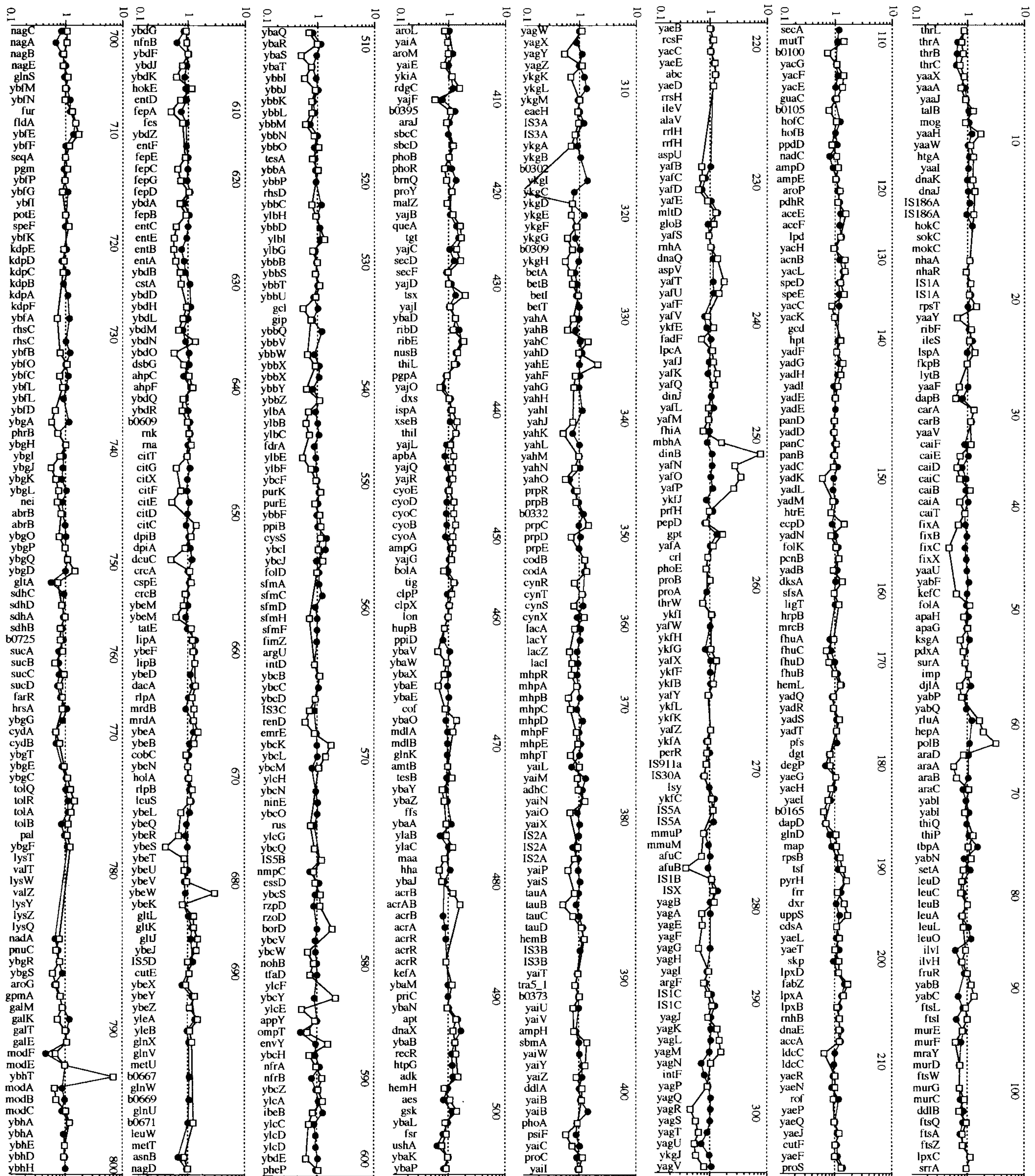

FIGURE 1.—Changes in gene expression within the *E. coli* genome following UV irradiation. The average change in transcript levels in the irradiated samples compared to unirradiated samples for each gene along the *E. coli* chromosome is plotted sequentially. Open squares, wild type; solid circles, *lexA1*. The location of genes on the chromosome, in kilobase pairs, is indicated along the top of each graph. The average change in transcript levels was calculated as the (average change in irradiated samples)/(average change in unirradiated samples). The data for all plotted genes represent an average of between 3 and 5 irradiated time points and at least one unirradiated time point. Time points for which the PCR or hybridization failed, or the fluorescent signal generated by the unirradiated sample was <30% above the background level of fluorescence, were not included in the averages.

FIGURE 1.—*Continued.*

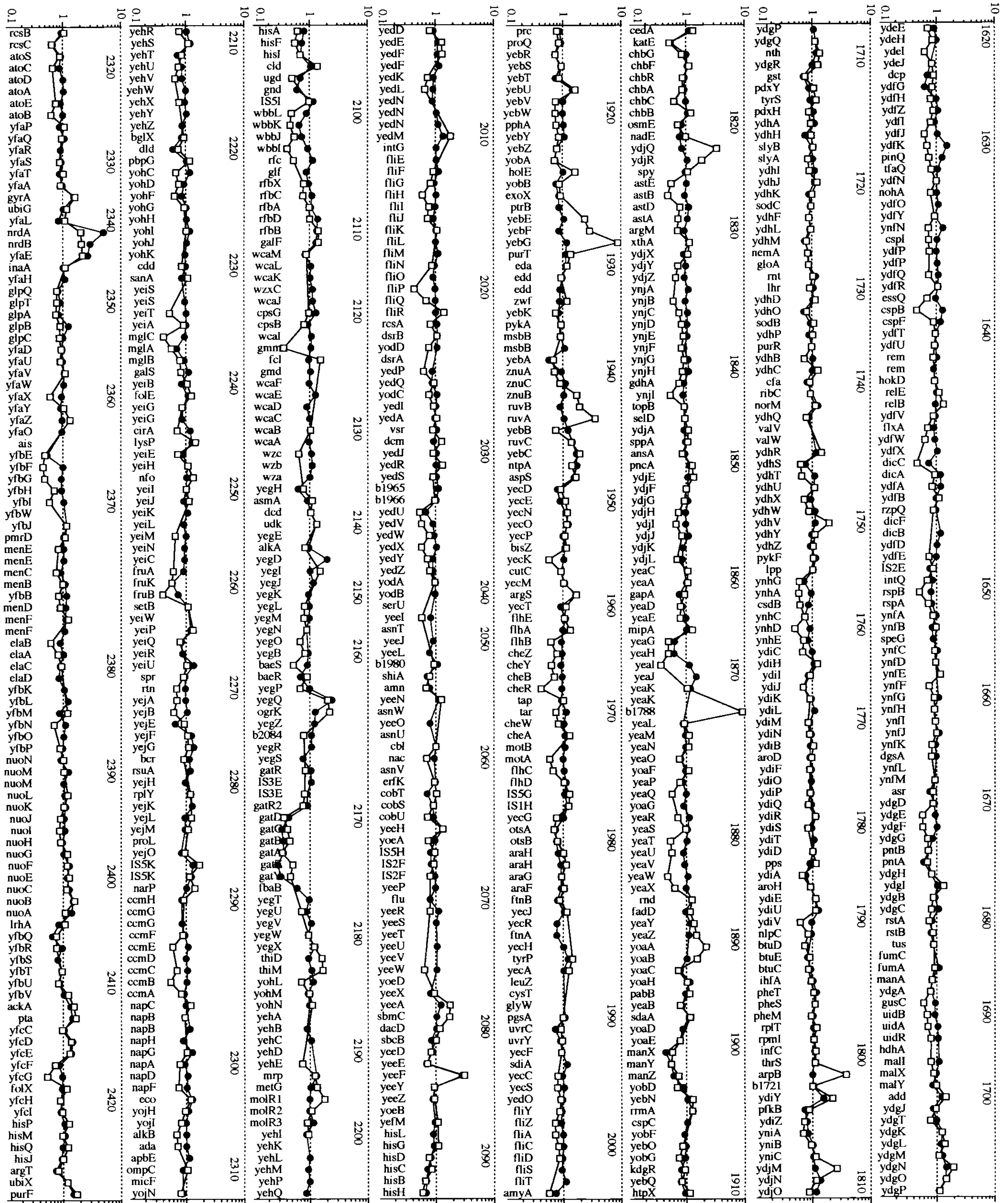

Figure 1.—*Continued.*

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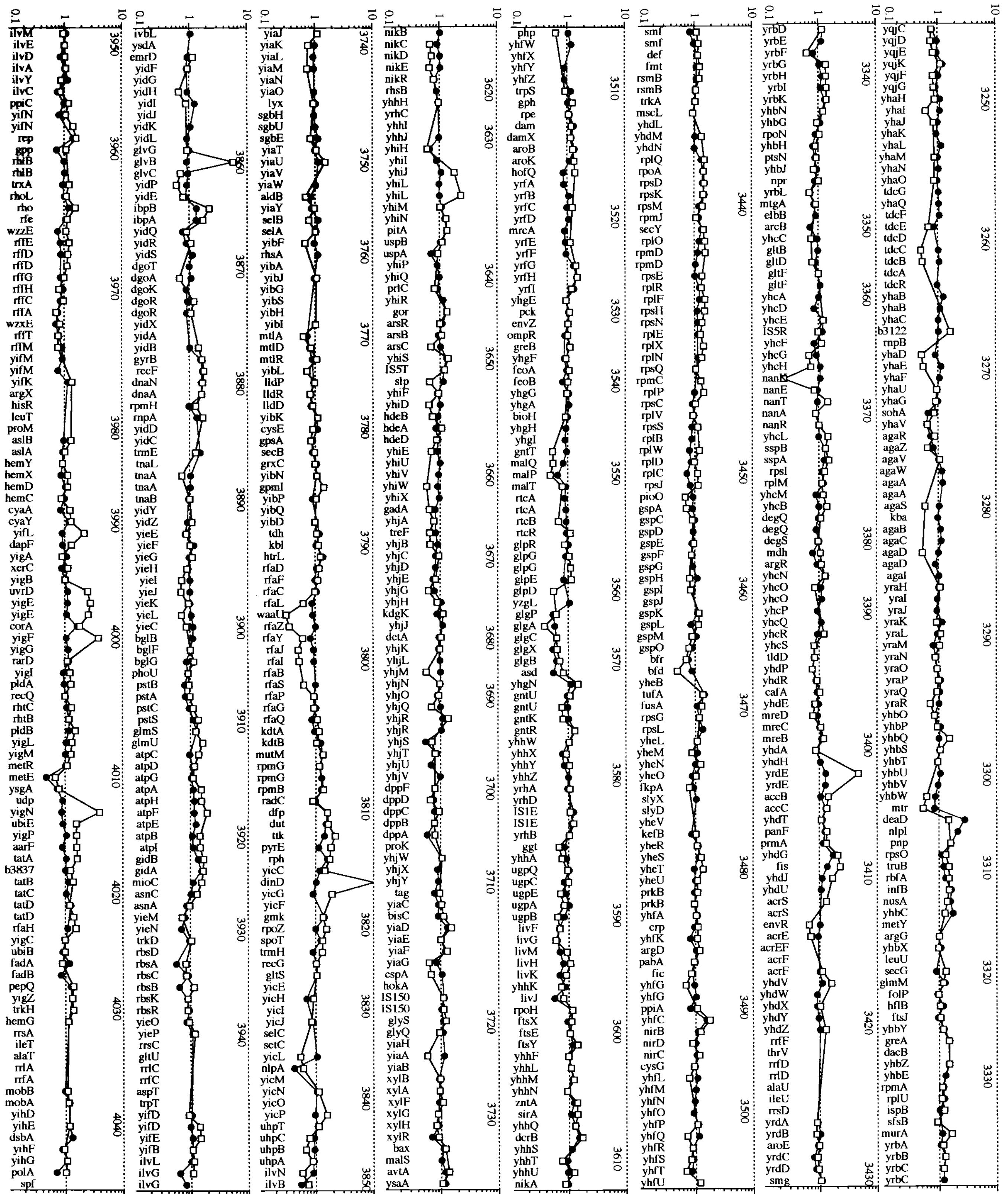
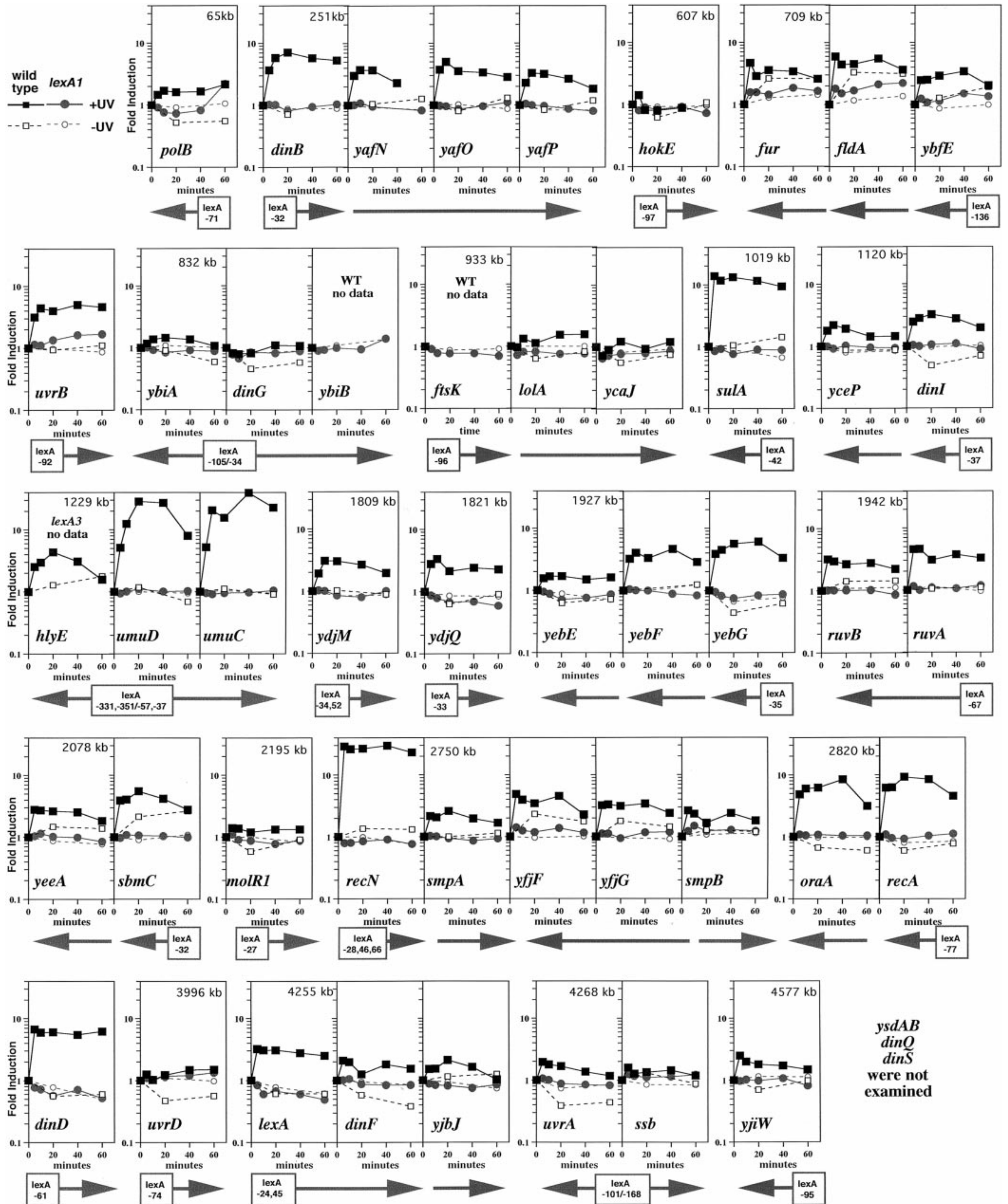


Figure 1.—*Continued.*

FIGURE 1.—*Continued.*


wild type
lexA1
+UV
-UV
Fold Induction
minutes
65kb
polB
lexA -71
251kb
dinB
yafN
yafO
yafP
lexA -32
607 kb
hokE
lexA -97
709 kb
fur
fldA
ybfE
lexA -136
uvrB
lexA -92
832 kb
ybiA
dinG
WT no data
ybiB
lexA -105/-34
933 kb
WT no data
ftsK
time
lolA
ycaJ
lexA -96
1019 kb
sulA
lexA -42
1120 kb
yceP
dinI
lexA -37
1229 kb
lexA3 no data
hlyE
umuD
umuC
lexA -331,-351/-57,-37
1809 kb
ydjM
lexA -34,52
1821 kb
ydjQ
lexA -33
1927 kb
yebE
yebF
yebG
lexA -35
1942 kb
ruvB
ruvA
lexA -67
2078 kb
yeeA
sbmC
lexA -32
2195 kb
molR1
lexA -27
recN
lexA -28,46,66
2750 kb
smpA
yfjF
yfjG
smpB
2820 kb
oraA
recA
lexA -77
dinD
lexA -61
3996 kb
uvrD
lexA -74
4255 kb
lexA
dinF
yjbJ
lexA -24,45
4268 kb
uvrA
ssb
lexA -101/-168
4577 kb
yjiW
lexA -95
ysdAB
dinQ
dinS
were not examined

following SOS induction, no induction of the chromosomally encoded *ssb* has been reported and SOS induction does not lead to higher levels of SSB protein (Brandsma *et al.* 1983; Perrino *et al.* 1987).

If the LexA-binding site is located within the operator regions of two diverging transcripts, it is possible that a single site may regulate the transcription of both operons. This is presumed to be the case at *uvrA* and *ssb* and also appears to occur between *ybiA* and *dinG* as well as *umuCD* and *hylE*.

In addition to the previously reported *lexA*-regulated genes, we observed several other genes that appeared to be upregulated in a LexA/RecA-dependent manner (Figure 2B). However, to determine whether these genes are directly under LexA control will require further investigation. Some of these genes do appear to have potential candidate LexA boxes (Table 1B). One method of predicting whether a LexA-like sequence will bind LexA is to examine its heterology index (HI), which is a value derived from a mathematical formula ranking the relatedness of a potential sequence to that of known LexA-binding sequences (Lewis *et al.* 1994). Low HI values predict that a potential sequence is more likely to bind LexA protein. Previous studies found that *lexA* sequences with an HI value of <15 generally bound LexA (Lewis *et al.* 1994) and recently this method was used to identify seven new *lexA*-regulated genes (Fernandez De Henestrosa *et al.* 2000). However, there are exceptions to the predictions from HI values. On the basis of earlier calculations that were based upon a smaller set of *lexA* sequences, Fernandez De Henestrosa *et al.* (2000) found that although *dinJ* had an HI value of 7.06, it did not bind LexA. Yet *ybfE*, which they calculated to have an HI value of 14.07, did bind LexA (Lewis *et al.* 1994). This observation suggests that all factors comprising a functional LexA box have not yet been identified. Therefore, we have recalculated the HI values using all 28 functional LexA sequences found on the chromosome and we have included any potential LexA-binding sequence with an HI value of <20 in Table 1B.

While some of these newly identified genes appear to have potential LexA-binding sequences, many of the induced genes do not, suggesting that in some cases the regulation may occur indirectly. Indirect *lexA*-dependent induction of these genes might occur through regulatory proteins that are themselves under lexA regulation, or through other regulatory proteins that are inactivated by a similar RecA-catalyzed proteolysis. The activated form of RecA is also known to induce proteolytic cleavage of other proteins containing "LexA-like" cleavage motifs such as those found in UmuD, the plasmid-encoded MucA, and the repressor proteins of several bacteriophage (Little 1984; Perry *et al.* 1985). This possibility is especially attractive considering that several of the newly identified genes, *borD*, the *lit-intE* region, and *ogrK*, share homology with cryptic prophage genes. The *borD* gene product is a homolog of the phage λ Bor protein, a lipoprotein expressed during lysogeny that is present in the outer membrane. The *borD* gene product shares homology to other bacterial virulence proteins and its expression increases *E. coli* survival in animal serum (Barondess and Beckwith 1990, 1995).

lit, *intE*, and several genes of unknown function in this same region are expressed at relatively late times following UV exposure (Figure 3). *lit* encodes a protease specific for elongation factor EF-tu. Expression of *lit* is induced at late times following phage T4 infection and prevents late phase phage amplification through its EF-tu proteolysis (Georgiou *et al.* 1998). Following T4 infection, the endogenous *lit* gene has been suggested to trigger an apoptotic-like death of the infected cell, thereby thwarting the reproduction of the virus and precluding the widespead infection of the population. Further characterization is required to know whether this activity or any of the genes in this region affect recovery following DNA damage.

ogrK is a prophage gene from a phage P2. Ogr has been shown to regulate late P2 gene transcription through an interaction with the host RNA polymerase (Wood *et al.* 1997). Additionally, an *ogr*-like gene, *regC* in *Serratia marcescens*, has been shown to be induced following mitomycin C treatment in an SOS-dependent manner (Jin *et al.* 1996).

grxA is a glutoredoxin that acts as a hydrogen donor for the *E. coli* ribonucleotide reductases. Several thioredoxin and glutoredoxin genes in *E. coli* are coregulated with ribonucleotide reductase gene expression (Prieto-Alamo *et al.* 2000). From this perspective, it is interesting to note that the ribonucleotide reductase genes *nrdA* and *nrdB* are among the strongest *lexA*-independent induced genes following UV exposure (Figure 4). *grxA* is also induced in an *oxyR*-dependent manner under some conditions (Prieto-Alamo *et al.* 2000).

Figure 2.—Transcriptional induction following UV irradiation in the genes surrounding known LexA boxes. The change in transcript levels for the indicated gene is plotted over time. The arrows indicate the direction of transcription within the operon relative to the LexA box. Arrows pointing left are transcribed on the minus strand and arrows pointing right are transcribed on the plus strand. The locations and distances, in base pairs, of the LexA box from the initial ATG codon are indicated in the boxes. The graphs of genes that are directly adjacent on the chromosome are joined together. The location of the gene(s) on the chromosome, in kilobase pairs, is indicated along the top of each plot. Solid squares, irradiated wild type; open squares, unirradiated wild type; solid circles, irradiated *lexA1*; open circles, unirradiated *lexA1*. Time points in which the PCR or hybridization failed, or the fluorescent signal generated by the unirradiated sample was $<30\%$ above the background level of fluorescence, are not plotted.

TABLE 1

Known and potential LexA boxes surrounding induced genes

A. Known genes	LexA box sequence	HI value[a]
ysdAB	tactgtttatttatacagta	1.81
umuDC	tactgtatataaaaacagta	2.12
sbmC	tactgtatataaaaacagta	2.12
pcsA	aactgtatataaatacagtt	2.19
recN no. 1	tactgtatataaaaaccagtt	3.53
dinQ	tactgtatgattatccagtt	3.92
urvB	aactgttttttttatccagta	4.26
dinI	acctgtataaataaccagta	4.84
hokE	cactgtataaataaacagct	4.92
recA	tactgtatgctcatacagta	4.98
sulA	tactgtacatccatacagta	5.39
uvrA	tactgtatattcattcaggt	6.23
ssb	acctgaatgaatatacagta	6.23
yebG	tactgtataaaatcacagtt	6.26
lexA/denF no. 2	aactgtatatacacccaggg	7.25
ydjQ	cactggatagataaccagca	7.42
lexA/dinF no. 1	tgctgtatatactcacagca	7.45
ruvAB	cgctggatatctatccagca	7.59
yjiW	tactgatgatatatacaggt	7.92
molR	aactggataaaattacaggg	8.14
dinS	agctgtatttgtctccagta	8.24
uvrD	atctgtatatatacccagct	8.46
recN no. 2	tactgtacacaataacagta	8.50
dinG	tattggctgtttatacagta	8.65
yigN	aactggacgtttgtacagca	8.82
ydjM1	tactgtacgtatcgacagtt	9.05
ftsK	tcctgttaatccatacagca	9.18
dinB	cactgtatactttaccagtg	9.40
recN no. 3	taatggtttttcatacagga	10.08
ydjM2	cactgtataaaaatcctata	10.85
ybfE	aactgattaaaaacccagcg	10.92
polB	gactgtataaaaccacagcc	12.55
Concensus	taCTGtatatatataCAGta	

(*continued*)

TABLE 1

(Continued)

B.	Potential LexA box	HI value[b]	Bases from start
intE region			
intE	ggctgctgaaaaatacagaa	16.94	−195
ymfI	ttctgtaccagaaaacagtt	15.48	84
ymfM	agctgcaggagcatgcagca	19.32	−122
lit	tgatgacagagtgtccagtg	20.32	−193
ymfE	cactggacactctgtcatca	20.32	−280
intE	ggcggtataagcatccagtg	14.76	84
intE	tgctgaaaaatacagaagta	20.81	−192
ymfM	ggcagttattcaaaacagat	19.98	−222
ymfM	aaccgcatgagaagacagca	18.91	−173
ymfN	aactgattgcgcttcctgta	16.89	−312
ymfN	cgctggttcaaagatcacta	20.90	−152
ymgF region			
ymgF	cggtgtaattatagacagct	15.08	−105
ymgH	aactgaaaaaaactccccggg	19.13	6
ydeO region			
ydeO	aaatgcatgcgaccacagtg	20.68	−272
ydeT region			
ydeS	tactgaaccagcagacagca	16.79	−43
yneL	cactgcatacgaaaacacca	18.21	−57
yoaA region			
yoaB	ccctgttgatttgaacaggg	13.32	−123
yoaA	ccctgttcaaatcaacaggg	13.32	−24
ogrK region			
ogrK	cattgtcctttatgccagca	19.29	8
ogrK	gactggacaatcactaaggt	19.35	−193
yqgC region			
yqgC	acatggattttccagcagtg	18.78	−193
yqgC	ctcagtaactgtaaccagct	20.65	−41
yhiL region			
yhiL	atctgtttttcagacaagta	18.22	−63
yhiL	tgctgttgtttttttacaatt	12.30	−187
glvB region			
glvG	tcctgaagtggcattcagcg	17.45	211
glvG	taatgaccaaattctcagtg	19.17	0
glvB	tgctggtgggaattaccgaa	20.04	−174
glvC	ggctggccaaaagtacaaat	20.85	578
glvC	tgctgtcggtttacccattg	15.97	214
ipbA region			
ibpA	tgctgaaaataacatcatca	17.25	−249
yigN region			
yigN	aactggacgtttgtacagca	8.82	−61

[a] HI values were calculated from all sequences reported by Lewis *et al.* (1994) and Fernandez de Henestrosa *et al.* (2000). The HI values reported here were calculated to include the results of Fernandez de Henestrosa *et al.* (2000) and therefore differ from the values used in their previous study.

[b] No sequences with HI values <20 were found for *grxA*, *borD*, *ybiN*, *arpB*, *yccF*, or *yifL*.

glvB induction is unusual in that *glvB* lies in the middle of a predicted operon and encodes a portion of a protein transport system. Nevertheless, *glvB* induction was also observed following exposure to gamma irradiation (data not shown) and may be driven from an alternative promoter or alternative open reading frame in the region.

In the case of *yigN*, a previous study has demonstrated that it contains a functional LexA binding site; however, no further increase in *yigN* expression was observed following treatment with mitomycin C in wild-type, *lexA51* (deficient), or *lexA1* (uninducible) *E. coli* cultures. We have no clear explanation of this difference. However, alternative promoters proximal to *yigN* could have allowed for full expression to occur in these previous studies prior to mitomycin treatment since *yigN* appeared to be heavily expressed under all conditions in that study (Fernandez De Henestrosa *et al.* 2000).

ibpA (*hslT*) and *ibpB* (*hslS*), encoding heat-inducible chaperonins, were also induced in a LexA-dependent manner.

There has been little functional characterization of the remaining induced genes. *yoaA* shares homology

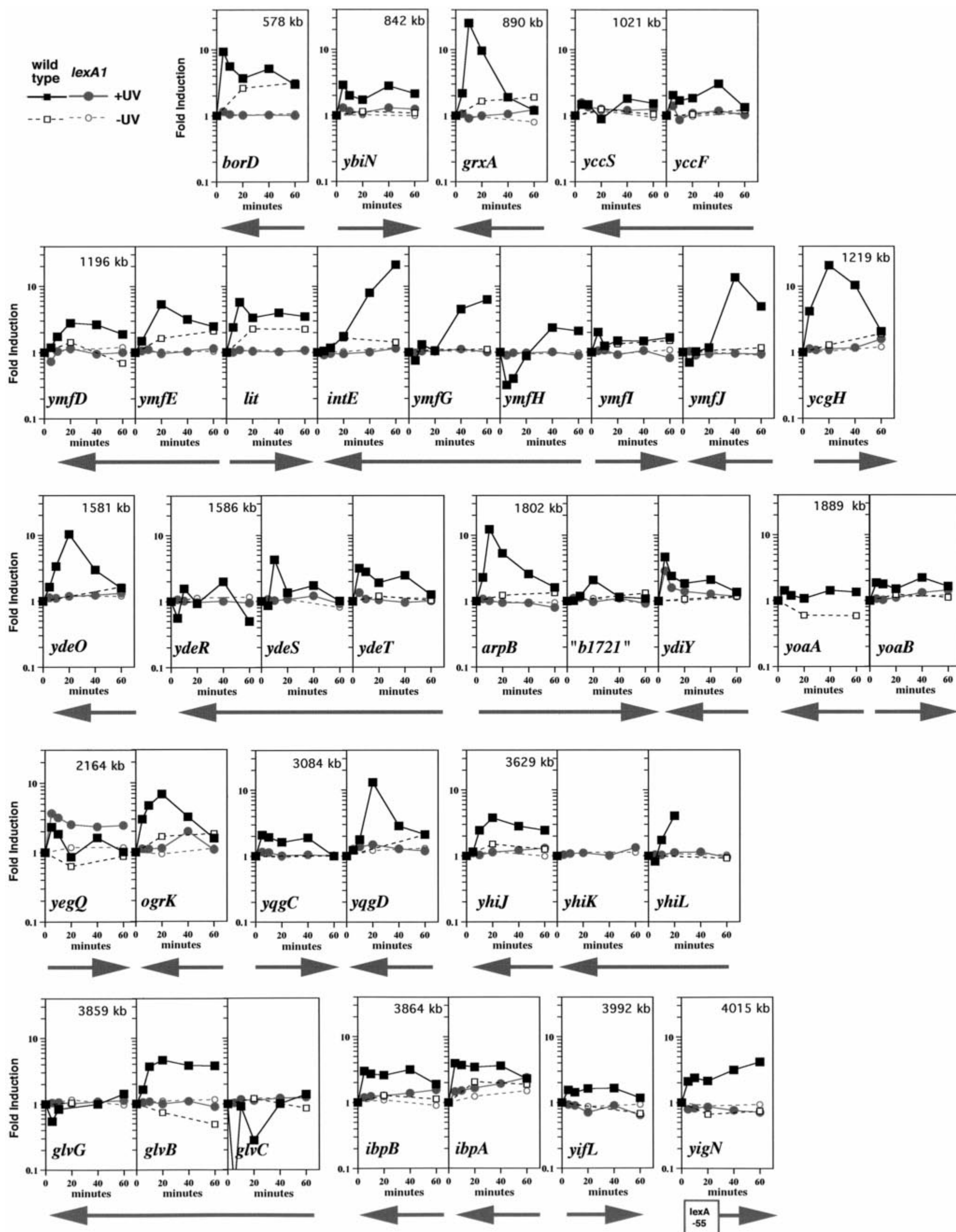


FIGURE 3.—Genes that displayed the largest LexA-dependent transcriptional induction following UV irradiation are plotted. The change in transcript levels for the indicated gene is plotted as in Figure 2. Solid squares, irradiated wild type; open squares, unirradiated wild type; solid circles, irradiated *lexA1*; open circles, unirradiated *lexA1*.

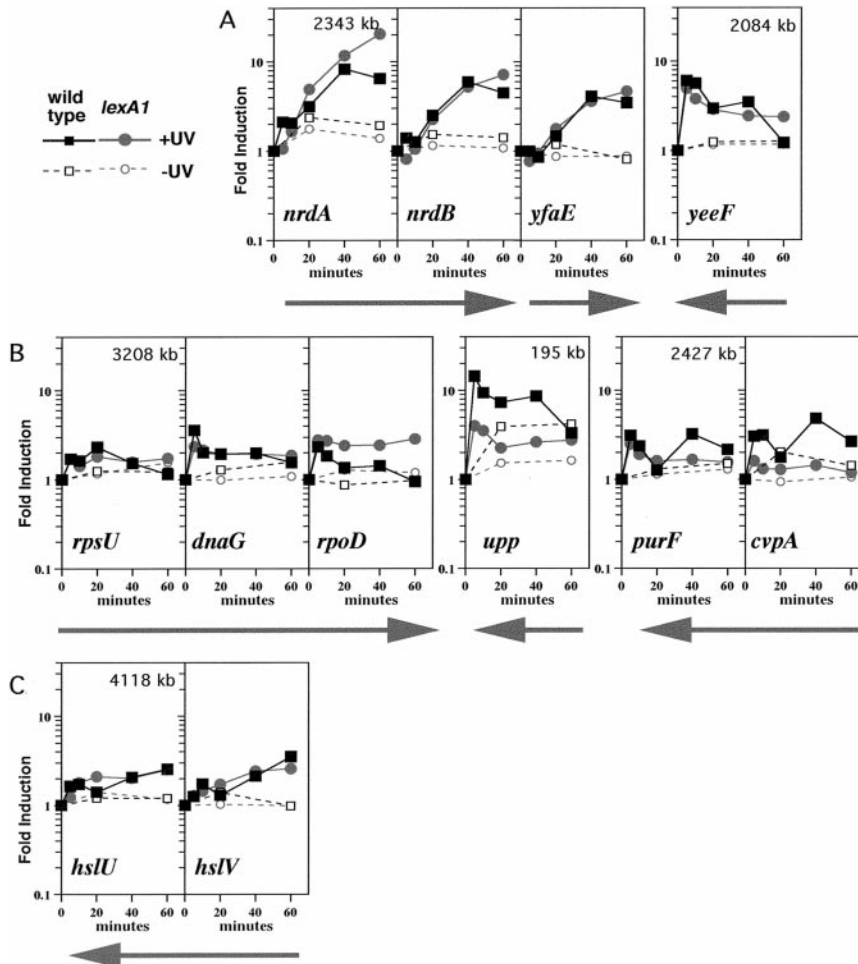


FIGURE 4.—Representation of genes displaying a LexA-independent transcriptional induction following UV irradiation. The change in transcript levels for the indicated gene is plotted as in Figure 2. (A) The largest LexA-independent inductions. (B) Typical profiles of genes with an early UV-dependent transcriptional induction. (C) Typical profiles of genes with a slow UV-dependent transcriptional induction. Solid squares, irradiated wild type; open squares, unirradiated wild type; solid circles, irradiated *lexA1*; open circles, unirradiated *lexA1*.

with other ATP-dependent helicases. The gene products of *ydeT*, *ydeS*, and *ydeR* share homology to other fimbrial proteins. *b1169 ycgH* has homology with other ATP-binding subunits of transport systems. Both *ydeO* and *ydiW* have motifs that suggest they may function as transcriptional regulators. No significant homology between *ybiN*, *yqgC*, *yhiJL*, or *yifL* and any other characterized proteins has been reported.

Genes induced independently of LexA following UV irradiation: The time courses of the largest *lexA*-independent inductions are plotted in Figure 4A. Most striking is precisely how few *lexA*-independent changes occur following UV exposure. In general, *lexA*-independent inductions, with the exception of *nrdA*, *nrdB*, and *yeeF*, are in the range of twofold effects. Furthermore, many of these *lexA*-independent profiles appear to rise very rapidly (within the first 5 min) and then either subside or plateau. A large number of genes and regions were observed to be regulated in this manner and some generalizations are apparent from both Figure 1 and Table 2. Many proteins associated with the replication machinery are slightly induced following UV irradiation. Additionally, several genes associated with purine and pyrimidine metabolism seem to be upregulated in a similar manner. Although not dramatic, these results are particularly impressive considering that these nucleotide metabolism genes are often found in very small operons spaced throughout the genome. Other categories of genes that appeared to be upregulated included heat-shock or chaperone proteins as well as several of the genes involved in RNA metabolism. It is notable that nearly half of the genes that were upregulated have had little or no functional characterization.

Loss of gene expression following UV irradiation: Whereas several studies have focused upon the need to upregulate certain gene products following UV irradiation, it has remained relatively unexplored, yet very possible, that repression or even active degradation of some gene transcripts will also be an important factor in cellular recovery. The bacterial microarray offers an opportunity to address this very question. Indeed, repression was observed in a large number of genes following UV irradiation. However, our results do not allow us to determine whether the decrease in a given transcript represents diminished transcription or accelerated degradation in response to UV irradiation. Nevertheless, a large number of genes in the wild-type, but not in the *lexA1*, samples were reduced in their transcript levels at the 5-, 10-, and 20-min time points following irradiation. This observation may suggest that some inhibition of

TABLE 2

Genes with increased transcript levels following UV-irradiation

Gene	WT (ave)[b]	*lexA1* (ave)	Wild type: minutes post irradiation[c]							*lexA1*: minutes post irradiation							Possible function
			5	10	20	40	60	20 (No UV)	60 (No UV)	5	10	20	40	60	20 (No UV)	60 (No UV)	
									Replication and repair								
umuC[a]	20.6	0.974	5.22	20.59	15.58	39.36	22.74	1.11	0.9	0.95	0.91	0.95	0.97	1.04	1.04	0.94	PolV, SOS mutagenesis
recN[a]	20.18	0.937	28.94	26.2	26.7	29.76	23.07	1.36	1.31	0.79	0.8	0.85	0.9	0.76	0.99	0.76	Protein used in DNA repair
umuD[a]	17.36	1.046	5.13	12.42	28.56	27.07	8	1.18	0.69	0.94	1.01	1.11	1.01	1.03	1	0.95	SOS mutagenesis; forms complex with PolV
recA[a]	10.08	1.251	6.16	6.28	9.26	8.49	4.57	0.6	0.78	1.09	0.95	0.93	1.04	1.12	0.8	0.84	Homologous strand pairing, DNA strand exchange
dinB[a]	7.749	1.098	3.66	5.8	7.08	5.72	5.25	0.71		1.03	1.02	0.81	0.94	1.03	0.88	0.88	PolIV
uvrB[a]	4.142	1.496	3.16	4.42	4	5.03	4.62	0.94	1.11	1.15	1.11	1.35	1.63	1.68	0.98	0.87	DNA repair; excision nuclease subunit B
uvrA[a]	3.846	1.167	1.96	1.79	1.68	1.37	1.18	0.39	0.44	1.07	1.01	0.88	0.85	0.83	0.74	0.85	DNA repair; excision nuclease subunit A
ruvA[a]	3.552	1.043	4.54	4.6	3.07	3.76	3.3	1.06	1.11	1.16	1	1.1	1.07	1.2	1.15	0.97	Holliday junction helicase subunit B; branch migration
polB[a]	3.2	1.098	1.46	1.7	1.61	1.65	2.14	0.52	0.55	0.91	0.76	0.73	0.82	2.19	0.91	1.06	DNA polymerase II
ydjQ[a]	3.361	0.852	2.78	3.35	2.13	2.41	2.27	0.63	0.91	0.87	0.79	0.68	0.69	0.59	0.86	0.84	Putative excinuclease subunit
uvrD[a]	2.505	1.127	1.25	1.02	1.22	1.47	1.49	0.47	0.56	1.1	1.03	1.17	1.2	1.33	1.09	0.98	DNA excision repair; helicase II
recF	1.792	1.675	1.22	1.1	0.99	0.98	1.31	0.59	0.66	2.01	1.65	1.11	0.98	0.91	0.86	0.73	ssDNA and dsDNA binding, ATP binding
dnaN	1.684	1.59	1.83	1.46	1.09	1.05	1.35	0.81	0.8	2.25	1.75	1.6	1.15	1.08	1.02	0.95	DNA polymerase III, β-subunit
dnaA	1.627	1.633	2.45	1.32	1.59	1.2	1.37	0.84	1.11	2.57	2.07	1.72	1.35	1.27	1.03	1.17	DNA initiation of chromosome replication
dnaC	1.482	1.621	2.06	1.52	1.32	1.64	1.28	1.2	0.91	1.68	1.41	1.54	1.88	1.92	1.15	0.93	DNA replication initiation and chain elongation
rep	1.622	1.433	1.13	0.83	0.8	1.1	1.33	0.57	0.71	1.67	1.53	1.56	1.68	1.91	1.14	1.19	Rep helicase, chromosome replication
ruvB[a]	1.931	0.889	3.19	2.96	2.66	2.78	2.22	1.42	1.44	1.01	1.01	1.01	1.02	0.86	1.09	1.12	Holliday junction helicase subunit A; branch migration
ruvC	1.375	1.452	1.01	0.76	0.89	0.81	0.62	0.5	0.69	1.58	1.51	1.55	1.49	1.64	1.16	0.98	Holliday junction nuclease; resolution of structures
									Transcription and transcriptional regulation								
lexA[a]	4.8	0.914	3.21	3.07	3.08	2.77	2.51	0.61	0.61	0.84	0.6	0.67	0.6	0.49	0.78	0.62	Regulator for SOS(lexA) regulon
dinI[a]	4.461	1.02	2.46	2.81	3.24	2.78	1.98	0.49	0.7	1.04	1	1.06	1.1	0.9	0.97	1.03	Damage-inducible protein I
deaD	1.479	2.819	1.31	0.65	0.85	0.64	0.58	0.5	0.59	2.11	1.35	1.62	1.7	2.1	0.65	0.61	Inducible ATP-independent RNA helicase
rpoD	1.71	2.131	2.33	1.84	1.36	1.43	0.95	0.87	0.98	2.78	2.73	2.41	2.43	2.86	1.28	1.2	RNA polymerase, sigma(70) factor
hepA	1.92	ND	1.84	1.82	1.77	2.16	1.53	0.89	1.01								Probable ATP-dependent RNA helicase
fis[a]	2.294	1.366	3.02	2.19	1.94	2.01	1.22	0.83	0.98	1.63	1.43	1.56	1.44	1.52	1.05	1.17	DNA inversion factor, transcription factor
suhB[a]	2.081	1.555	1.94	1.33	1.35	1.6	0.96	0.69	0.69	2.38	2.07	2.25	2.56	2.71	1.44	1.64	Enhances synthesis of σ32 in mutant
ttk[a]	2.179	1.408	2.67	2.07	1.77	2.56	1.77	1.09	0.9		1.4	1.52	1.58	1.44	1.17	0.94	Putative transcriptional regulator
ydeO[a]	2.412	1.009	1.64	3.37	10.33	2.98	1.58		1.65	1.13	1.11	1.19	1.23	1.32	1.18	1.19	Putative ARAC-type regulatory protein
rph	1.75	1.595	1.53	1.08	0.82	1.4	1.25	0.69	0.7	1.69	1.66	1.76	2.04	1.82	1.22	1.03	RNase PH
srmB	1.474	1.839	1.96	1.42	1.16	1.54	1.29	0.88	1.12	2.78	2.4	2.3	2.49	2.72	1.31	1.45	ATP-dependent RNA helicase
soxS	1.728	1.345	2.07		1.48	2.1	0.95	1.06	0.85	1.06	1.1	1.23	1.48	1.05	0.93	0.83	Regulation of superoxide response regulon
rnpA	1.696	1.329	2.32	1.47	1.24	1.34	1.05	0.81	0.94	1.45	1.28	1.24	1.29	1.45	0.94	1.08	RNase P component; processes tRNA, 4.5S RNA

(*continued*)

TABLE 2

(Continued)

Gene	WT (ave)[b]	*lexA1* (ave)	Wild type: minutes post irradiation[c]							*lexA1*: minutes post irradiation							Possible function
			5	10	20	40	60	20 (No UV)	60 (No UV)	5	10	20	40	60	20 (No UV)	60 (No UV)	
									Nucleoside metabolism								
nrdA	2.051	5.046	2.11	2.06	3.15	8.28	6.5	2.37	1.94	1.06	1.66	4.91	11.74	20.49	1.77	1.39	Ribonucleoside reductase 1, α-subunit, B1
grxA[a]	4.515	1.205	2.18	25.25	9.65	1.9	1.2	1.66	1.9	1.07	0.91	1	1.06	1.23	0.95	0.8	Glutaredoxin coenzyme for ribonucleotide reductase
nrdB	2.117	2.956	1.4	1.26	2.51	5.92	4.47	1.53	1.41	0.81	1.05	2.25	5.23	7.14	1.15	1.08	Ribonucleoside reductase 1, β-subunit, B2
upp	2.126	1.922	14.45	9.43	7.34	8.62	3.32	3.93	4.19	4	3.53	2.26	2.63	2.76	1.53	1.63	Uracil phosphoribosyltransferase
pyrF	1.734	1.701	2.13	1.97	1.2	2.66	2.1	1.1	1.22	1.96	1.57	1.49	1.76	1.98	0.93	1.13	Orotidine-5′-phosphate decarboxylase
tsx	1.964	1.331	4.08	2.34	1.61	2.12	1.24	0.93	1.39	1.72	1.51	1.42	1.26	1.21	1.05	1.09	Nucleoside channel; phage T6 receptor and colicinK
guaA	1.833	1.447	2.04	1.89	1.08	2.09	1.47	0.91	0.96	1.72	1.89	1.5	1.6	1.61	1.05	1.25	GMP synthetase (glutamine-hydrolyzing)
speB	1.494	1.757	1.13	1.06	0.89	0.88	0.97	0.62	0.7	1.68	1.49	1.81	1.75	1.66	0.92	0.99	Agmatinase
purF	1.728	1.515	3.13	2.37	1.27	3.24	2.17	1.31	1.51	2.5	1.9	1.61	1.67	1.56	1.14	1.3	PRPP aminotransferase
dfp	1.601	1.602	2.9	1.95	1.96	2.19	2.65	1.41	1.5	2.06	1.62	1.8	1.78	1.75	1.29	0.96	Flavoprotein affecting DNA panthothenate metabolism
pyrH	1.602	1.56	2.36	1.33	1.13	1.8	1.23	0.85	1.11	1.71		1.53	1.72	1.56	1.01	1.08	Uridylate kinase
ntpA	1.416	1.732	1.54	1.53	1.25	1.76	1.39	0.92	1.19	1.55	1.79	1.8	1.88	1.9	1	1.06	dATP pyrophosphohydrolase
dut	1.641	1.504	1.68	1.28	0.95	1.42	1.48	0.84	0.82	1.36	1.43	1.5	1.55	1.64	1.06	0.93	Deoxyuridinetriphosphatase
gauB	1.509	1.532	2.64	1.76	1.53	2.33	1.81	1.23	1.44	2.02	1.98	1.75	1.97	1.51	1.21	1.2	IMP dehydrogenase
purB	1.479	1.526	3.31	2.82	2.39	3.38	2.26	1.49	2.34	2.14	2.2	1.86	2.18	1.92	1.36	1.34	Adenylosuccinate lyase
gpt	1.666	1.319	2.85	2.01	2	1.9	1.03	1.09	1.26	1.67	1.51	1.36	1.48	1.4	1.01	1.24	Guanine-hypoxanthine phosphoribosyltransferase
pyrD	1.557	1.426	8.04	5.09	2.98	5.8	2.73	2.76	3.57	2.02	1.7	1.17	1.29	1.2	1.01	1.06	Dihydro-orotate dehydrogenase
purH	1.494	1.478	0.72	0.93	0.49	1.51	1.73	0.58	0.86	1.65		1.55	1.8	1.68	1.12	1.14	AICAR formyltransferase; IMP cyclohydrolase
pyrE	1.839	1.124	1.38	1.06	0.85	1.3	1.25	0.6	0.67	1.13	1.06	0.92	1.02	0.93	0.94	0.86	Orotate phosphoribosyltransferase
murB	1.457	1.438	3.18	2.01	2.35	2.68	2.6	1.63	1.89	1.2	1.17	1.25	1.33	1.09	0.87	0.81	UDP-N-acetylenolpyruvoylglucosamine reductase
cmk	1.5	1.37	1.69	1.8	0.99	2.18	1.48	1.24	0.93	1.48	1.46	1.37	1.46	1.39	1.08	1.01	Cytidylatate kinase
kdsB	1.623	1.225	0.82	1.11	0.66	1.85	1.32	0.7	0.72	1	1.15	0.81	1.05	0.95	0.85	0.77	CTP:CMP-3deoxy-D-manno-octulosonate transferase
									Translation/amino acid metabolism								
argS	1.708	1.645	2.51	2.8	1.59	3.36	2.55	1.67	1.33	1.86	1.7	1.46	1.83	1.91	1.13	1	Arginine tRNA synthetase
prfC	1.656	1.576	2.01	1.49	1.28	1.75	1.79	0.98	1.03	1.98	1.58	1.68	1.9	2	1.1	1.22	Peptide chain release factor RF-3
aspS	1.662	1.54	2.13	1.65	1.27	1.53	1.69	1.01	0.98	2.19	1.82	2.04	2.15	2.04	1.38	1.28	Aspartate tRNA synthetase
speA	1.485	1.715	1.62	1.49	1.1	1.26	1.25	0.87	0.94	2.12	2.24	1.76	1.87	1.7	1.03	1.23	Biosynthetic arginine decarboxylase
prfB	1.598	1.594	1.97	1.39	1.37	1.73	2.13	0.97	1.18	1.6	1.62	1.59	1.67	1.45	0.96	1.03	Peptide chain release factor RF-2
fabZ	1.698	1.492	3.63	2.7	2.71	2.94	1.82	1.64	1.61	1.51	1.46	1.5	1.66	1.59	1.04	1.03	(3R)-hydroxymyristol acyl carrier dehydratase
tgt	1.682	1.507	2.2	1.65	1.64	1.96	1.59	1.07	1.08	1.9	1.84	1.86	1.9	1.69	1.2	1.24	tRNA-guanine transglycosylase
yjeA	1.759	1.298	1.85	1.63	1.14	1.4	0.93	0.95	0.63	1.58	1.39	1.45	1.45	1.43	1.05	1.2	Putative lysyl-tRNA synthetase
gltX	1.67	1.375	3.65	2.84	1.88	3.49	2.67	1.95	1.53	1.68	1.67	1.6	1.97	2.05	1.2	1.41	Glutamate tRNA synthetase, catalytic subunit
*que*A	1.613	1.381	1.22	0.84	0.83	1.05	0.9	0.61	0.59	1.8	1.5	1.55	1.58	1.82	1.14	1.25	Synthesis of queuine in tRNA
yafT[a]	1.747	1.123	1.16	1.15	1.9	1.73	0.96	0.89	0.69	1.16	1.05	1.14	1.34	1.15	1.01	1.07	Putative aminopeptidase

(*continued*)

TABLE 2

(Continued)

Gene	WT (ave)[b]	*lexA1* (ave)	Wild type: minutes post irradiation[c] 5	10	20	40	60	20 (No UV)	60 (No UV)	*lexA1*: minutes post irradiation 5	10	20	40	60	20 (No UV)	60 (No UV)	Possible function
										Heat shock, transport proteins							
ycgH[a]	5.823	1.086	4.16		20.67	10.37	2.07	1.29	1.91	1.13	1.08	1.12	1.17	1.58	1.04	1.2	Putative ATP-binding transport system component
glvB[a]	5.737	0.902	1.65	3.69	4.64	3.84	3.82	0.74	0.49	1.05	1.08	1	1.1	0.91	1.11	1.17	PTS system, arbutin-like IIB component
yeeF	3.086	2.815	6.08	5.67	2.97	3.5	1.22	1.25	1.27	4.98	3.8	2.87	2.44	2.38	1.17	1.17	Putative amino acid/amine transport protein
ybeW[a]	2.986	0.913	0.9	1.76	8.27	1.97			1.08	0.84	0.9	1.03	1.12	1.04	0.96	1.2	Putative dnaK protein
*ibpB**	2.216	1.335	2.99	2.71	2.59	3.16	1.9	1.28	1.13	1.19	1.24	1.26	1.39	1.56	1.09	0.9	Heat-shock protein
corA	1.805	1.63	1.88	1.37	1.15	2.21	2.01	0.98	0.93	2.39	2.04	2.08	2.52	2.38	1.43	1.37	Mg^{2+} transport, system I
yhfC	1.771	1.573	2.11	2.01	1.27	2.22	1.51	1.03	1.03	2.27	1.6	1.81	1.79	1.77	1.14	1.21	Putative transport
sdaC	1.762	1.384	1.82	1.85	0.72	1.89	1.43	1.17	0.58	1.7	1.6	1.59	1.54	1.7	1.26	1.09	Probable serine transporter
potB[a]	1.833	1.226	4.54	3.13	1.98	2.77	2.06	1.54	1.62	1.76	1.29	1.04	1.09	1.01	0.97	1.05	Spermidine/putrescine transport system permease
hslU	1.559	1.498	1.64	1.73	1.41	2.07	2.54	1.21	1.2	1.24	1.79	2.1	2.02	2.51	1.41	1.17	Heat-shock protein hslVU, ATPase subunit
potC	1.501	1.489	4.53	3.66	1.75	2.61	2.05	1.82	2.07	2.44	2.02	1.34	1.26	1.28	1.01	1.23	Spermidine/putrescine transport system permease
uraA	1.554	1.38	2.85	2.14	0.88	1.65	0.99	0.82	1.37	2.37	2.05	1.15	1.31	1.16	1.06	1.27	Uracil transport
potD	1.383	1.498	2.03	2.3	1.99	1.58	1.23	1.24	1.4	1.98	2.69	2.23	2.09	1.72	1.5	1.36	Spermidine/putrescine periplasmic transport protein
potA	1.61	1.241	4.43	3.36	1.75	2.51	1.47	1.98	1.38	1.74	1.82	1.38	1.19	1.16	1.06	1.29	ATP-binding component of spermidine transport
artP	1.503	1.305	3.15	2.89	1.43	2.92	1.52	1.75	1.42	1.75	1.58	1.32	1.64	1.54	1.28	1.12	Third arginine transport system, ATP-binding
										Cell Division							
*sulA**	9.561	1.108	13.74	11.7	13.17	11.55	9.36	1.05	1.44	0.85	0.92	0.76	0.9	0.89	0.89	0.67	Suppressor of lon; inhibits cell division
										Prophage genes							
ymfN[a]	7.57	1.034	0.09			3.35	7.12	0.54	0.39	1.1	1.03	1.14	1.1	0.93	1.15	0.9	Orf, hypothetical protein
intE[a]	4.292	0.905	1.05	1.17	1.76	7.96	21.11	1.65	1.43	0.91	0.96	0.95	1.01	1.15	1.03	1.17	Prophage e14 integrase
ymfJ[a]	3.658	1.004	0.7	1.02	1.17	13.56	4.95		1.17	1.03	0.9	0.94	0.96	0.94	1	0.9	Orf, hypothetical protein
ymfG[a]	2.514	0.996	0.75	1.31	1.05	4.5	6.28	1.1	1.11	0.98	1.04	1.03	1.12	1.01	1.05	1.03	Orf, hypothetical protein
ogrK[a]	2.191	1.242	2.98	4.7	6.89	3.23	1.59	1.69	1.85	1.12	1.11	1.14	2	1.09	0.94	1.14	Prophage P2 ogr protein
ybcU[a]	1.854	1.004	9.35	5.6	3.68	5.15	2.97	2.61	3.16	1.16	1.05	1.02	1.03	1.01	1.02	1.08	Bacteriophage λ Bor protein homolog
										Others							
prfB	2.159	ND	1.96	1.41	1.24	1.73	2.08		0.78								
fabF	1.788	2.249	3.05	2.23	2.53	2.73	1.8	1.38	1.38	1.8	1.89	1.97	2.24	2.95	1.02	0.91	3-oxoacyl-[acyl-carrier-protein] synthase II
pphB	2.743	1.189	2.18	3.18	8.5	1.29	1.24	1.3	1.09	1.11	1.02	1.01	1.07	1.08	0.94	0.84	Protein phosphatase 2
yhdG	2.087	1.744	1.83	1.59	1.24	1.66	0.88	0.68	0.7	2.38	2.01	2.13	1.93	1.84	1.18	1.18	Putative dehydrogenase
hlyE	1.894	ND	2.52	2.97	4.35	3.08	1.57	1.28	1.78								Hemolysin E
ribH	1.857	1.703	1.14	0.99	0.8	1.1	1.17	0.41	0.71	1.64	1.57	1.48	1.6	1.63	0.93	0.93	Riboflavin synthase, β chain
gidA	1.678	1.558	1.14	0.87	0.78	0.93	1.02	0.52	0.61	1.39	1.42	1.65	1.46	1.44	0.83	1.06	Glucose-inhibited division; chromosome replication?
prmA	1.595	ND	1.29	1.18	1.04	1.31	1.12	0.63	0.86								
cvpA	1.791	1.368	3.05	3.15	1.77	4.85	2.67	2.03	1.43	1.61	1.31	1.29	1.44	1.19	0.94	1.06	Membrane protein required for colicin V production
pabC	1.555	ND	1.25	1.19	1.25	1.57	1.27	0.84	0.84								4-amino-4-deoxychorismate lyase

(*continued*)

TABLE 2

(Continued)

Gene	WT (ave)[b]	*lexA1* (ave)	Wild type: minutes post irradiation[c] 5	10	20	40	60	20 (No UV)	60 (No UV)	*lexA1*: minutes post irradiation 5	10	20	40	60	20 (No UV)	60 (No UV)	Possible function
										Others (*continued*)							
pta	1.66	1.444	1.72	1.73	1.14	2.57	2.26	1.26	1.01	1.47	1.73	1.76	1.99	2.11	1.2	1.31	Phosphotransacetylase
nuoB	1.567	1.492	2.11	2.46	1.23	3.12	2.48	1.51	1.4	2.17	1.48	1.53	1.48	1.32	1.09	1.05	NADH dehydrogenase I chain B
gidB	1.698	1.354	2.08	1.4	1.79	1.7	1.52	0.85	1.15	1.35	1.3	1.39	1.31	1.35	1	0.98	Glucose-inhibited division; chromosome replication?
fldA	1.491	1.474	5.95	4.41	4.5	5.5	3.64	3.29	3.15	1.8	1.51	1.68	2.11	2.22	1.17	1.36	Flavodoxin 1
plsX	1.594	1.363	2.01	1.37	1.1	1.8	1.33	0.99	0.92	1.48	1.32	1.31	1.5	1.41	1.04	1.02	Glycerolphosphate auxotrophy in plsB background
smpA[a]	1.95	0.984	2.15	2.05	2.6	1.96	1.67	1	1.14	1.03	1.01	0.94	0.86	0.93	0.94	1	Small membrane protein A
smpB[a]	1.755	1.169	2.64	2.33	1.67	2.39	1.81	1.28	1.19	1.23	1.49	1.23	1.28	1.23	1.08	1.13	Small protein B
emrB	1.606	1.286	1.28	1.06	1.04	1.15	1.05	0.65	0.74	1.32	1.06	1.16	1.43	0.98	1.05	0.8	Multidrug resistance; putative translocase
ackA	1.541	1.342	3.41	2.91	2.97	3.27	2.62	1.92	2.02	1.23	1.34	1.25	1.37	1.52	0.99	1.01	Actate kinase
fabG	1.265	1.576	2.13	1.84	1.86	2.04	1.27	1.3	1.59	1.24	1.44	1.47	1.72	2.01	0.94	1.06	3-oxoacyl-[acyl-carrier-protein] reductase
sbp	1.344	1.481	0.84	0.81	1.14	3.66	3.36	1.47	1.45	0.6	1.1	1.6	2.21	2.97	1.18	1.11	Periplasmic sulfate-binding protein
										Unknowns							
dinD[a]	10.47	1.028	6.71	5.95	6.02	5.48	6.19	0.56	0.6	0.77	0.72	0.57	0.71	0.52	0.78	0.5	DNA-damage-inducible protein
oraA[a]	9.002	1.087	4.83	5.99	6.2	8.43	3.13	0.67	0.6	1.09	1.05	1.08	1.04	1.04	0.97	0.98	Regulator, OraA protein
yebG[a]	8.853	1.175	3.87	4.51	5.64	6.1	3.34	0.44	0.62	0.95	0.82	0.75	0.84	0.87	0.67	0.77	Orf, hypothetical protein
yfaE	2.18	2.69	1	0.86	1.47	4.09	3.48	1.19	0.81	0.77	0.94	1.78	3.61	4.67	0.87	0.88	Orf, hypothetical protein
yigF[a]	3.813	1.057	1.84	15.18	3.02	2.03	1.19	1.22		1.09	0.98	0.97	1.08	1.06	1.02	0.94	Orf, hypothetical protein
yigN[a]	3.969	0.866	2.11	2.38	2.13	3.13	4.14	0.66	0.74	0.79	0.82	0.86	0.76	0.71	0.89	0.93	Putative α-helix chain
arpB[a]	3.749	0.996	2.29	12.29	5.32	2.58	1.61	1.23	1.34	1.08	1.01	0.95	0.94	0.8	0.97	0.95	Orf, hypothetical protein
dinF[a]	3.621	1.029	2.1	1.98	1.26	1.81	1.54	0.58	0.38	1.02	1.06	0.87	0.84	0.84	0.95	0.85	DNA-damage-inducible protein F
yafO[a]	3.486	1.031	3.73	4.97	3.51	3.35	2.83	0.8	1.31	0.98	0.96	0.86	0.96	1.11	1.02	0.87	Orf, hypothetical protein
yegQ	2.034	2.403	2.32	1.85	0.86	1.63	1.02	0.63	0.88	3.66	3.18	2.53	2.35	2.46	1.18	1.18	Orf, hypothetical protein
yoaA	2.182	ND	1.43	1.2	1.08	1.43	1.35	0.6	0.59								Putative enzyme
yafN[a]	2.738	1.097	3	3.71	3.66	2.28		1.05	1.26	1.01	1.08	0.95		0.82	0.92	0.84	Orf, hypothetical protein
yebF[a]	2.909	0.859	3.24	4.06	3.33	4.67	2.88		1.25	1.05	1.01	1	0.89	0.84	1	1.23	Orf, hypothetical protein
yafP[a]	2.622	1.13	2.29	3.29	3.18	2.65	1.83	0.84	1.18	1.05	1	0.97	0.87	0.8	0.83	0.83	Orf, hypothetical protein
ydiY[a]	2.197	1.539	4.65	2.39	1.85	2.1	1.37	1.07	1.18	2.86	1.59	1.4	1.28	1.18	1.02	1.14	Orf, hypothetical protein
ydjM[a]	2.629	1.104	1.92	3.08	3.03	2.65	1.94	1.03	0.89	1.03	1.01	0.85	0.8	1	0.84	0.86	Orf, hypothetical protein
yebC	1.944	1.69	2.67	2.14	2.17	1.9	1.28	0.97	1.12	1.51	1.92	1.64	2.33	2.36	1.2	1.11	Orf, hypothetical protein
yfgB	2.064	1.555	2.05	1.29	1.08	1.33	1.06	0.6	0.72	1.56	1.41	1.38	1.44	1.48	0.96	0.91	Orf, hypothetical protein
ygjO	1.796	1.703	1.14	1.1	0.71	1.06	0.93	0.58	0.52	2.11	1.48	1.62	1.59	1.63	1.01	0.97	Putative enzyme
yebE[a]	2.379	1.047	1.56	1.68	1.69	1.49	1.61	0.63	0.72	0.95	0.88	0.71	0.76	0.86	0.88	0.71	Orf, hypothetical protein
ydgN	1.931	1.454	1.95	1.05	0.96	0.99	1.23	0.54	0.74	1.85	1.35	1.31	1.26	1.21	0.95	0.97	Putative membrane protein
yliG	1.774	1.544	2.14	1.76	1.3	2.28	1.3	1.01	0.97	2	1.58	1.7	1.95	1.84	1.16	1.19	Orf, hypothetical protein
ybiN[a]	2.072	1.222	2.9	2	1.72	2.8	2.13	1.15	1.08	1.31	1.16	1.1	1.3	1.24	1.02	0.98	Orf, hypothetical protein
dcrB	1.732	1.498	1.98	1.94	1.51	2.4	2.65	1.21	1.21	1.74	1.48	1.71	2.33	2.18	1.14	1.38	Orf, hypothetical protein

(*continued*)

TABLE 2

(Continued)

Gene	WT (ave)[b]	*lexA1* (ave)	Wild type: minutes post irradiation[c]							*lexA1*: minutes post irradiation							Possible function
			5	10	20	40	60	20 No UV)	60 (No UV)	5	10	20	40	60	20 (No UV)	60 (No UV)	
										Unknowns (*continued*)							
yceP[a]	2.028	1.183	1.79	2.21	1.93	1.44	1.45	0.87	0.87	1	0.92	1.03	0.96	0.94	0.8	0.84	Orf, hypothetical protein
yciH	1.971	1.221	2.33	1.96	1.41	2.08	1.63	0.9	1.01	1.63	1.34	1.22	1.25	1.46	1.01	1.25	Orf, hypothetical protein
yedM	1.826	1.355	1.86	3.91	4.61	2.73	1.73	1.81	1.44	1.02	0.98	2.11	1.2	0.89	0.95	0.88	Orf, hypothetical protein
ycgW	1.582	ND	1.83	3.12	2.99	3.77	4.47	1.97	2.12								Orf, hypothetical protein
yebU	1.641	1.476	4.46	2.75	2.53	3.35	2.21	1.97	1.76	1.74	1.33	1.48	1.5	1.44	1.11	0.92	Putative nucleolar proteins
ycfB	1.757	1.358	1.89	1.42	0.96	1.68	1.43	0.99	0.69	1.43	1.2	1.26	1.43	1.37	0.88	1.09	Orf, hypothetical protein
yjiW[a]	2.255	0.855	2.49	2	1.8	1.71	1.47	0.7	0.98	0.97	1.02	0.98	1.07	0.81	1.15	1.12	Orf, hypothetical protein
yifL[a]	2.185	0.907	1.55	1.43	1.63	1.65	1.17		0.68	0.93	0.9	0.71	0.9	0.64	0.87	0.93	Orf, hypothetical protein
ybfE	1.677	1.364	2.46	2.5	2.91	3.42	2.04	1.27	1.91	1.24	1.08	1.14	1.5	1.35	0.86	0.99	Orf, hypothetical protein
yhbC	1.276	1.754	1.78	0.99	1.09	0.77	0.76	0.82	0.87	1.41	1.06	1	0.95	0.93	0.62	0.6	Orf, hypothetical protein
ydeT[a]	2.083	0.96	3.17	2.8	1.91	2.47	1.26	1.2	1.03	1.35	1.08	1.05	0.96	1.01	1.17	1.1	Putative outer membrane protein
ygcM	1.78	1.25	2.27	1.93	2.22	2.91	1.66	1.33	1.14	1.12	1.2	1.08	1.09	1.26	0.84	1	Putative 6-pyruvoyl tetrahydrobiopterin synthase
yfjA	1.548	1.467	2.71	1.48	1.32	1.95	1.48	1	1.31	1.43	1.48	1.57	1.6	1.88	1.09	1.08	Orf, hypothetical protein
ychF	1.613	1.394	2.34	1.96	1.53	2.07	1.7	1.1	1.28	1.84	1.77	1.85	1.87	1.87	1.27	1.37	Putative GTP-binding protein
ygiR	1.513	1.494	1.5	0.89	0.77	0.92	1.14	0.55	0.83	1.91	1.44	1.42	1.53	1.32	1.05	0.99	Orf, hypothetical protein
IS5K	1.696	1.292	1.82	1.44	1.52	1.75	1.23	0.93	0.9	1.05	1.18	1.07	1.15	1.04	0.83	0.87	Orf, hypothetical protein
yfhE	1.157	1.828	0.47	0.57	0.47	0.61	0.57	0.42	0.51	1.34	1.8	1.84	1.63	1.66	0.9	0.91	Orf, hypothetical protein
yeeA	1.753	1.229	2.78	2.71	2.63	2.53	1.84	1.47	1.38	1.04	1.14	1.02	0.99	0.85	0.87	0.77	Orf, hypothetical protein
yaeS	1.679	1.221	1.68	1.15	1.11	1.4	1	0.88	0.63	1.22	1.26	1.22	1.28	1.28	1.06	0.99	Orf, hypothetical protein
yaaH	1.706	1.193	1.53	0.95	0.84	0.9	0.77	0.46	0.71	1.1	1.15	1.08	0.9	0.99	0.93	0.82	Orf, hypothetical protein
ycfQ	1.675	1.285	3.39	3.33	3.43	3.03	1.98	1.89	1.73	1.48	1.32	1.45	1.53	1.48	1.34	0.92	Orf, hypothetical protein
b1228	1.624	1.33	0.61	0.86	0.98	1.37	1.01	0.56	0.63	1.23	1.15	1.17	1.27	1.3	0.89	0.95	Orf, hypothetical protein
ydjR[a]	1.884	1.055	1.76	2.17	1.58	1.68	1.71	0.85	1.04	1.03	0.84	0.94	0.91	0.92	0.95	0.81	Orf, hypothetical protein
yrfH	1.481	1.453	1.24	1.29	0.93	1.66	1.88	1	0.89	1.18	1.23	1.4	1.6	1.6	1.01	0.92	Orf, hypothetical protein
ybjF	1.627	1.338	1.7	1.31	1.31	1.73	1.27	0.81	0.99	1.68	1.46	1.6	1.5	1.62	1.18	1.17	Putative enzyme
yacL	1.496	1.385	2.79	2.66	2.31	2.69	2.34	1.72	1.7	1.41	1.41	1.48	1.61	2.02	1.13	1.16	Orf, hypothetical protein
yfgK	1.395	1.499	1.1	0.99	0.98	1.12	1.18	0.74	0.8	1.13	1.33	1.47	1.5	1.69	0.91	0.99	Putative GTP-binding factor
ydgO	1.449	1.415	1.67	1.28	1.14	1.04	0.74	0.74	0.88	1.53	1.17	1.17	1.08	1.03	0.88	0.81	Orf, hypothetical protein
yleA	1.512	1.345	1.45	1.19	1.01	1.21	0.96	0.66	0.88	1.18	1.45	1.02	1.13	1.24	0.89	0.9	Orf, hypothetical protein
yiaD	1.532	1.318	1.95	1.3	1.98	1.61	1.47	0.88	1.29	1.21	1.45	1.74	1.65	1.33	1.1	1.14	Putative outer membrane protein
ygeQ	1.424	1.423	2.43	1.57	1.73	1.54	1.24	1.3	1.09	1.18	1.06	1.15	1.09	1.21	0.74	0.86	Orf, hypothetical protein
yccF[a]	1.765	1.067	2.05	1.7	1.84	3.04	1.34	1.04	1.22	1.4	0.86	1.09	1.18	1.02	0.98	1.1	Orf, hypothetical protein
yabO	1.624	1.207	1.19	1.1	1.15	1.1	0.82	0.61	0.71	1.09	1.1	1.01	1.01	1.1	0.87	0.89	Orf, hypothetical protein
yhdJ	1.686	1.117	2.55	1.71	1.23	1.38	0.76	0.73	1.08	1.55	1.07	1.16	1.03	1.11	0.95	1.17	Putative methyltransferase

[a] Induced in a *lexA*-dependent manner.
[b] Ave represents the average transcript level in irradiated samples compared to that in unirradiated samples as described in MATERIALS AND METHODS.
[c] Values represent the fold change in transcript level at the time indicated compared to the beginning of the experiment as described in MATERIALS AND METHODS.

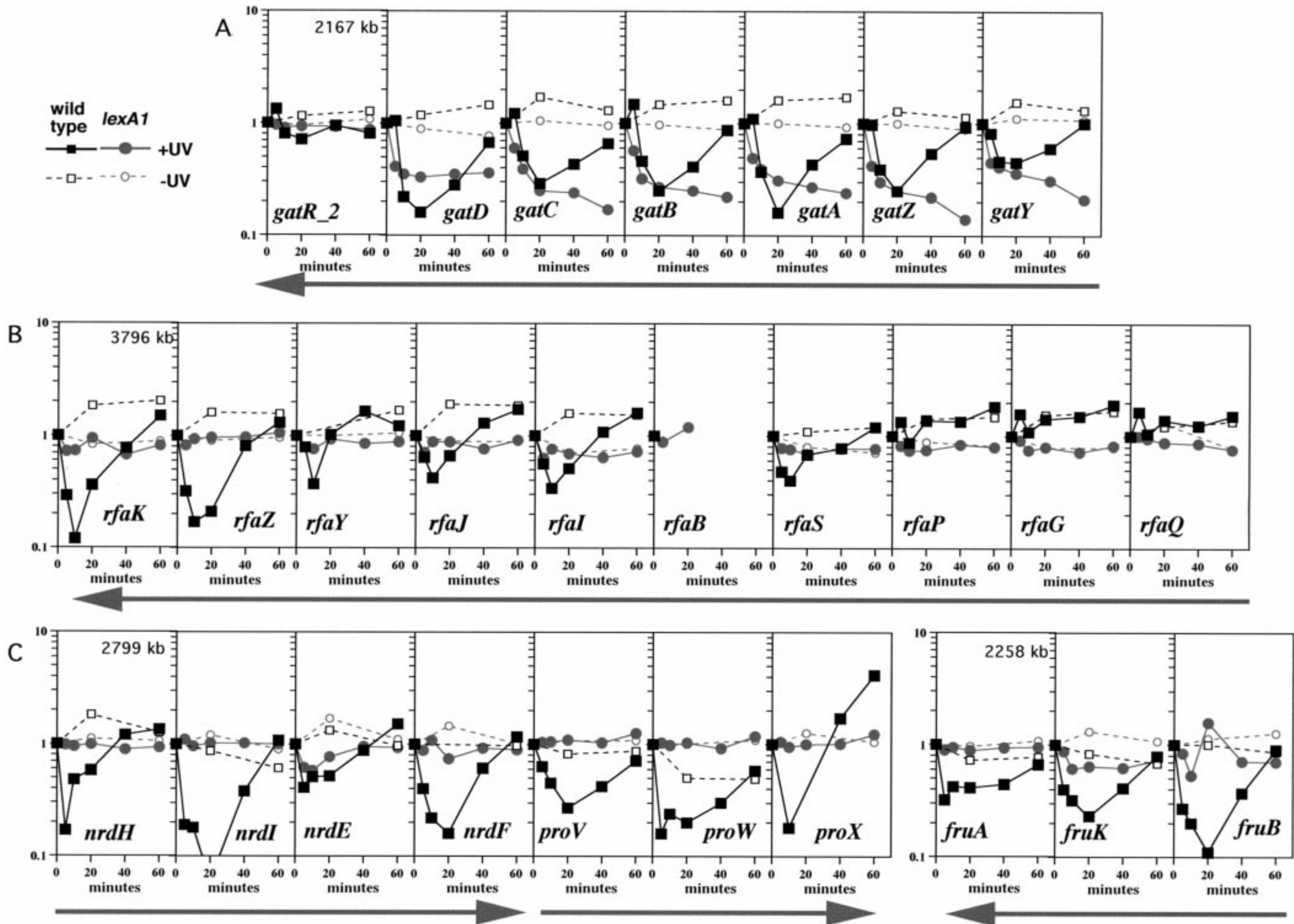


Figure 5.—Representation of genes that displayed a reduced level of transcripts following UV irradiation. The change in transcript levels for the indicated gene is plotted as in Figure 2. (A) A typical operon in which the transcript from both wild-type cells and *lexA1* mutants was observed to be reduced. (B) An operon in which the transcript from wild-type cells was observed to be severely reduced at the 5′ end. (C) Three operons in which transcripts were reduced in wild-type cells throughout the operon.

transcription or degradation of transcripts occurs in a *lexA*-dependent manner, and it may point to a lexA-dependent mechanism for inhibition of transcription or enhanced degradation of these transcripts. Due to the small sample size of this experiment, we are inclined to interpret these findings cautiously. However, further investigation of these observations is clearly warranted.

The phenomenon of downregulation is also interesting to consider with respect to DNA repair. Actively transcribed genes in *E.coli* are repaired preferentially compared to nontranscribed genes (Mellon and Hanawalt 1989). When the *lac* operon is actively transcribed at the time of UV irradiation, repair of the transcribed strand occurs within 5 min after irradiation. While it is assumed that this response allows for the rapid transcriptional recovery of expressed genes, little is known about the actual inhibition or recovery of transcription for operons other than *lac* in *E. coli*.

Diminished transcript levels were clearly evident in some operons. Several different temporal profiles were observed (Figure 5). In some cases, exemplified by the *gat* operon, the decrease in transcript levels following UV irradiation was observed in both wild-type and *lexA1* cells (Figure 5A). In the wild-type cells, the time at which transcripts recovered to preirradiation levels varied but generally occurred prior to 40 min postirradiation. Interestingly, whereas transcripts recovered to pretreatment levels in the wild-type cells, transcripts of several genes failed to recover in *lexA1* mutants within the period observed in these experiments, suggesting that *lexA*-regulated genes have an important role in this transcriptional recovery (Figure 5A).

The *gat* operon, controlling galactitol uptake and metabolism, is one of several operons involved in the metabolism of different carbon sources, which were found to have reduced transcript levels following UV irradiation. Other carbon metabolism operons whose transcript levels decreased include *fru*, *man*, *wwb*, *glg*, and *mal* . Our cultures were grown with glucose as the sole carbon source. It may be interesting to know how these metabolic pathways are regulated when cells are grown in the presence of their respective carbon sources.

TABLE 3

Operons with reduced transcript levels following UV-irradiation

Operon	Location in kb and strand transcribed	WT (ave)[a]	*lexA1* (ave)	Wild type: minutes postirradiation[b]							*lexA1*: minutes postirradiation							Possible function
				5	10	20	40	60	20 (No UV)	60 (No UV)	5	10	20	40	60	20 (No UV)	60 (No UV)	
araD	65−	0.85	1.07	0.82	1.08	0.98	1.33	1.4	1.12	1.51	1.04	1.06	1.01	1.07	1.15	1	1	L-ribulose-5-phosphate 4-epimerase
araA		0.58	ND		0.48	0.38	0.84	0.79	0.93	1.22	0.93	1.08	0.98	0.99	0.96			L-arabinose isomerase
araB		0.65	1.05	0.63	0.71	0.24	1.04	1.16	1.14	1.19	0.97	1.05	1	1.05	1.13	1	0.99	L-ribulokinase
modF	791−	0.67	0.45	0.44	0.54	0.54	1.37	1.9	1.19	1.68	0.4	0.68	0.73	0.68	0.44		1.3	ATP-binding component of molybdate transport system
ybiC	835+	0.62	0.51	1.28	1.01	1.04	0.93	0.93	1.48	1.86	0.85	0.9	0.89	0.84	0.57	1.57	1.61	Putative dehydrogenase
ybiK	865+	0.83	0.93	0.74	0.84	1	1.52	2.22	1.44	1.59	0.49	1.06	1.38	1.42	1.13	1.2	1.15	Putative asparaginase
yliA		0.63	0.73	0.35	0.42	0.54	0.8	1.22	0.79	1.32	0.57	0.76	0.76	0.71	0.63	0.92	0.95	Putative ATP-binding component of a transport system
yliA		ND	0.79								1.01	0.7	0.79	0.68	0.59	1.04	0.86	Putative ATP-binding component of a transport system
yliB		0.62	0.56	0.36	0.36	0.43	0.65	0.88	0.85	0.89	0.3	0.46	0.7	0.59	0.46	0.95	0.83	Putative transport protein
yliC		0.6	0.83	0.47	0.34	0.42	0.55	0.79	0.8	0.91	0.71	0.85	0.85	0.82	0.71	0.93	0.97	Putative transport system permease protein
yliD		0.74	0.61	0.58	0.32	0.44	0.63	0.78	0.85	0.63	0.46	0.49	0.65	0.61	0.52	0.89	0.89	Putative transport system permease protein
minC	1223−	0.33	0.65	1.31	0.73	0.52	0.64	0.96	2.45	2.61	0.79	0.94	0.83	0.82	0.58	1.2	1.23	Cell division inhibitor, inhibits ftsZ ring formation
minD		0.45	0.63	1.52	0.81	0.6	0.74	0.78	2.1	1.84	0.8	0.93	0.88	0.72	0.54	1.37	1.1	Cell division inhibitor, a membrane ATPase, activates minC
minE		0.33	0.94	1.03	0.59	0.46	0.58	0.64	1.87	2.17	0.96	0.98	1.02	1.1	0.84	1.05	1.03	Cell division topological specificity factor
treA	1244−	0.48	0.7	2.29	1.15	1.27	1	1.09	3	2.7	0.99	0.89	0.88	0.8	0.84	1.27	1.25	Trehalase, periplasmic
ycgC	1246−	0.49	0.54	0.65	0.65	0.71	0.88	1.23	1.39	1.96	0.54	0.59	0.58	0.56	0.4	0.99	0.99	Putative PTS system enzyme I
ycgS		0.69	0.67	0.77	0.52	0.35	0.58	0.88	0.88	0.92	0.77	0.74	0.72	0.7	0.67	1.12	1.04	Putative dihydroxyacetone kinase
ycgT		0.8	ND	0.99	0.59	0.62	0.77	1.01	0.86	1.12			0.95	0.94	1.1			Putative dihydroxyacetone kinase
ycgT		ND	0.78								0.81	0.8	1.14	0.79	0.7	1.12	1.06	Putative dihydroxyacetone kinase
ydcL	1500+	0.59	0.57	1.97	1.23	1.43	0.9	0.77	2.19	2.05	0.9	0.85	0.72	0.43	0.31	1.3	0.96	Orf, hypothetical protein
yddU	1561−	ND	ND								0.68	0.79	0.74	0.84	1.09			Putative enzyme
yddV		0.27	0.79	0.71	0.57	0.73	0.56	0.73	2.29	2.61	0.85	0.97	0.92	0.73	0.88	1.07	1.14	Orf, hypothetical protein
pqqL	1570−	ND	0.71								0.72	0.79	0.77	0.91	0.88	1.24	1.05	Putative peptidase
yddB		0.38	0.66	0.93	0.6	0.64	1.18	1.25	2.25	2.54	0.68	0.83	0.84	1.19	1.01	1.7	1.05	Orf, hypothetical protein
yddA		0.6	0.87	0.73	0.5	0.84	1.2	1.19	1.3	1.67	0.96	1.1	1.08	1.21	1.14	1.3	1.22	Putative ATP-binding component of a transport system
yeaG	1864+	0.5	0.63	1.41	1.32	1.46	1.1	0.96	2.86	2.18	0.88	0.93	1.07	0.72	0.58	1.49	1.16	Orf, hytpothetical protein
yeaH	1866+	0.5	0.62	0.61	0.62	0.95	0.58	0.52	1.2	1.44	0.69	0.95	0.84	0.65	0.58	1.21	1.18	Orf, hypothetical protein
manX	1900+	0.58	0.44	1.16	0.51	0.39	0.54	0.75	1.01	1.31	0.71	0.67	0.46	0.38	0.3	1.28	1.02	PTS enzyme IIAB, mannose-specific
manY		0.55	0.55	1.18	0.72	0.33	0.61	0.74	1.3	1.31	0.82	0.64	0.53	0.48	0.42	1.07	1.04	PTS enzyme IIC, mannose-specific
manZ		0.73	0.6	1.23	0.65	0.48	0.55	0.7	0.92	1.05	0.82	0.74	0.57	0.49	0.42	1.06	0.96	PTS enzyme IID, mannose-specific

(*continued*)

TABLE 3

(Continued)

Operon	Location in kb and strand transcribed	WT (ave)[b]	*lexA1* (ave)	Wild type: minutes post irradiation[b] 5	10	20	40	60	20 (No UV)	60 (No UV)	*lexA1*: minutes post irradiation 5	10	20	40	60	20 (No UV)	60 (No UV)	Possible function
IS5I	2099−	0.98	1.16	1.07	0.7	0.56	0.76	0.76	0.56	1.01	1.15	1.1	1.17	1.31	1.28	1.01	1.06	IS5 transposase
wbbL		0.49	0.89	0.61	0.25	0.41	1.12	1.25	1.27	1.7	0.73	0.77	0.76	0.86	0.96	0.92	0.91	Putative creatinase
wbbK		0.47	0.66	0.49	0.24	0.39	1.3	1.6	1.53	1.91	0.27	0.26	0.29	0.45	0.83	0.69	0.58	Putative glucose transferase
wbbJ		0.55	0.74	0.77	0.53	0.79	1.41	1.65	1.64	2.12	0.48	0.54	0.56	0.71	0.71	0.79	0.83	Putative o-acetyl transferase
wbbI		0.41	0.93	0.52	0.5	0.9	1.37	1.31	2	2.46	0.61	0.69	0.79	0.87	0.93	0.87	0.8	Putative Galf transferase
rfc		0.53	1.14	0.37	0.3	0.59	1.05	0.95	1.14	1.3	0.6	0.59	0.71	0.75	1.04	0.68	0.61	o-Antigen polymerase
glf		ND	0.88								0.62	0.64	0.62	0.7	0.88	0.79	0.78	UDP-galactopyranose mutase
gatR_2	2069−	0.76	0.9	1.33	0.8	0.71	0.94	0.8	1.15	1.26	0.96	0.9	0.93	0.92	0.86	0.95	1.07	Split galactitol utilization operon repressor, fragment 2
gatD		0.36	0.43	1.05	0.22	0.16	0.28	0.67	1.18	1.46	0.41	0.35	0.33	0.35	0.36	0.89	0.77	Galactitol-1-phosphate dehydrogenase
gatC		0.41	0.33	1.22	0.51	0.29	0.43	0.66	1.71	1.3	0.6	0.39	0.25	0.24	0.17	1.05	0.95	PTS system galactitol-specific enzyme IIC
gatB		0.45	0.35	1.48	0.46	0.25	0.41	0.87	1.47	1.6	0.57	0.32	0.27	0.25	0.22	0.97	0.88	Galactitol-specific enzyme IIB phosphotransferase system
gatA		0.34	0.35	1.1	0.37	0.16	0.43	0.73	1.61	1.71	0.49	0.39	0.31	0.27	0.24	1	0.93	Galactitol-specific enzyme IIA phosphotransferase system
gatZ		0.51	0.28	0.98	0.39	0.25	0.54	0.93	1.29	1.14	0.42	0.3	0.25	0.22	0.14	1	0.89	Putative tagatose 6-phosphate kinase 1
gatY		0.46	0.32	0.82	0.46	0.45	0.6	1	1.55	1.32	0.45	0.41	0.36	0.31	0.21	1.11	1.08	Tagatose-bisphosphate aldolase 1
fruA	2260−	0.6	0.9	0.32	0.42	0.41	0.44	0.66	0.72	0.77	0.89	0.94	0.88	0.93	0.94	0.96	1.07	PTS system, fructose-specific transport protein
fruK		0.57	0.58	0.4	0.32	0.23	0.41	0.79	0.83	0.68	0.87	0.61	0.64	0.62	0.73	1.31	1.07	Fructose-1-phosphate kinase
fruB		0.4	0.73	0.27	0.2	0.11	0.37	0.9	1	0.86	0.84	0.53	1.57	0.71	0.7	1.13	1.26	PTS system, fructose-specific IIA/fpr component
yfbE	2363+	0.49	0.53	1.66	1.17	0.85	0.88	0.97	2.91	1.61	0.85	0.79	0.85	0.8	0.8	1.44	1.62	Putative enzyme
malP	3545−	0.5	0.67	0.97	0.71	0.75	0.61	1.04	1.73	1.55	0.76	0.72	0.92	0.78	0.64	1.28	1.01	Maltodextrin phosphorylase
malQ		0.56	0.84	0.69	0.32	0.56	0.39	0.47	0.87	0.87	0.98	0.93	1.02	0.95	0.79	1.19	1.04	4-α-glucanotransferase (amylomaltase)
glpD	3560+	0.56	ND	0.22	0.37	0.4	0.3	0.61	0.66	0.69								sn-glycerol-3-phosphate dehydrogenase (aerobic)
glgP	3561−	0.62	0.55	0.49	0.3	0.5	0.51	0.66	0.68	0.9	0.44	0.5	0.74	0.68	0.64	1.15	1.04	Glycogen phosphorylase
glgA		0.39	0.59	0.3	0.26	0.43	0.37	0.49	0.74	1.16	0.46	0.5	0.75	0.79	0.66	1.1	1.06	Glycogen synthase
glgC		0.63	0.63	0.57	0.53	0.5	0.64	0.87	1.01	0.96	0.5	0.66	0.8	0.79	0.62	1.05	1.1	Glycose-1-phosphate adenylyltransferase
glgX		0.64	0.54	0.33	0.48	0.5	0.61	0.86	0.94	0.81	0.36	0.49	0.68	0.57	0.47	0.99	0.91	A glycosyll hydrolase, debranching enzyme
glgB		0.71	0.62	0.72	0.67	0.45	0.8	0.99	0.94	1.11	0.56	0.72	0.84	0.7	0.64	1.17	1.05	1,4-α-glucan branching enzyme
nlpA	3836−	0.61	0.43	1.47	1.11	1.05	1.9	2.4	2.6	2.62	0.49	0.62	0.62	0.52	0.46	1.25	1.3	Lipoprotein-28
aceB	4213+	0.67	0.55	1.48	1.23	0.78	1.24	1.95	2.37	1.61	0.77	0.79	0.71	0.55	0.43	1.35	1.02	Malate synthase A
aceA		1	0.64	1.82	0.56	0.57	0.49	0.97	0.81	0.96	0.74	0.67	0.58	0.53	0.35	1.01	0.77	Isocitrate lyase
aceK		0.46	0.44	0.59	0.46	0.37	0.38	0.79	1.14	1.11	0.42	0.44	0.36	0.3	0.24	0.9	0.71	Isocitrate dehydrogenase kinase/phosphatase

[a] Ave represents the average transcript level in irradiated samples compared to that in unirradiated samples as described in MATERIALS AND METHODS.

[b] Values represent the fold change in transcript level at the time indicated compared to the beginning of the experiment as described in MATERIALS AND METHODS.

A second form of repression profile is exemplified by the *rfa* operon, which encodes gene products involved in lipid synthesis in the membrane. The *rfa* operon displayed a loss of transcript following UV irradiation in wild-type cells, but not in the *lexA1* mutant. The loss of transcript was more severe at the 5′ end of the operon, which may at least partially reflect the fact that most RNA degradation in *E. coli* is believed to occur 3′–5′ (Figure 5B).

A third pattern of transcript reduction can be seen in *nrdHIEFproVWX* and *fruAKB* operons shown in Figure 5C. In these cases, loss of the wild-type transcripts occurred uniformly throughout the operons.

The most severely reduced transcripts from operons for which we obtained signal are listed in Table 3. Of interest to note is the repression of operons such as *minCDE* following UV irradiation. These genes are required to induce and regulate septum formation prior to cell division and it is interesting to consider the loss of these transcripts with respect to the induction of other genes that regulate cell division such as *sulA* and *ftsK*. Also worth noting is that *minC*, which is severely repressed, actually contains a LexA-like box, which is predicted to be a very good LexA-binding sequence (Fernandez De Henestrosa *et al.* 2000). Dramatic reductions in transcript levels at predicted LexA boxes were also observed for *rfaJ* and *metE* (Figure 5B and Table 3). While it is impossible from these experiments to determine if the repression is directly due to a LexA regulation, we did observe cases where functional LexA boxes appeared to result in inhibition of gene expression following UV irradiation. The loss of transcription from *araDAB* following UV irradiation seems likely to be due to the upregulation of *polB*, which is located proximal to this operon (Figure 1 and Table 3). Thus, although LexA serves as a negative regulator for *polB*, it essentially behaves as a positive regulator of *araDAB* expression.

The study we have presented should serve as a starting point for follow-up projects. It provides some generalization with respect to the role of LexA in the regulation (up and possibly down) of genes in response to one type of environmental stress. Surprisingly, it shows us that, in the absence of LexA regulation, there are no other major responses to UV irradiation at the level of transcriptional regulation. It remains to be determined whether the minor changes are significant in terms of the overall stress response. The values and raw data for any or all of the genes can be retrieved either via the web locations listed in materials and methods or upon request.

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